Low Back Pain

Mechanism, Diagnosis and Treatment

Sixth Edition

James M. Cox

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Sixth Edition

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FOREWORD

In the fall of 1970, I attended a workshop of the American Chiropractic Board of Radiology. I was a new diplomate, having completed my residency program and receiving diplomate status in that same year. Although not a precise contemporary in our professional, educational studies, Dr. Cox and I nevertheless both became diplomates in radiology in 1970, and it was at that first workshop that I listened to Dr. Cox present some of his ideas on the acute low back syndrome. Twenty-eight years later, it is my pleasure to write this Foreword and to realize what a great distance has been traveled in that time period.

Today, Dr. Cox, along with others here at National College of Chiropractic, have had the privilege of working in collaboration with members of the Stritch School of Medicine at Loyola University, in a federally funded research project to study the biomechanics of the lumbar spine in particular as they relate to the clinical procedure known as "flexion distraction." During the past 28 years, Dr. James Cox has dedicated uncounted hours, months, and years to the research, development, refinement, and application of flexion distraction technique to those patients who suffer the ubiquitous, but elusive malady known as low back pain. His success in these efforts is unparalleled.

It is a clear tribute to the vision, purpose, and tenacity of Dr. James Cox that the 6th edition of his book titled *Low Back Pain* is now in print.

The condition known as low back pain has been studied by thousands of experts, covering uncounted articles, journals, and books. This vast literature has chronicled the development of diagnosis and treatment of low back syndromes throughout the last century. Despite all this, the causes of low back pain have sometimes eluded the grasp of even the best scientists.

In light of this history, I think it is particularly important that Dr. Cox has brought to us, once again, and in a clearly enhanced form, not only the thoughts, experiences, and experiments of many scientists who have studied the phenomenon known as low back pain, but also the more pragmatic art-based approach to the treatment of people who suffer from this condition, which we refer to in a general way as low back pain, despite its many causes. One cannot help but be impressed by the breadth of coverage of the topic, from the biomechanics of the low back through anatomic to neurologic elements. The importance of clinical laboratory diagnosis is carefully defined and the developments of the latest research are presented in a cogent and coherent process, which makes this book not only interesting to read, but particularly useful for the clinician.

Finally, the approach to the treatment of these patients, especially by those who choose to practice the conservative treatment of low back pain through chiropractic healing, is provided in a clear and concise manner, leaving a clearly open-ended opportunity for the development of new knowledge.

On a personal note, it has been my privilege to know Dr. James Cox since the mid 1960s and it is with great admiration and affection that I extend my sincere thanks and deepest appreciation for this lifelong dedication to the art and science of chiropractic healing.

James F. Winterstein, D.C.
President
National College of Chiropractic
Lombard, Illinois

A few years back, a lovely, young woman came to my office via a referral from Dr. Cox. At the time, I was gathering material for upcoming presentations I was to make, one of which was with Dr. Cox. This patient displayed some very interesting clinical findings, and I thought her case would make for interesting discussion during my lecture. She seemed approachable, so I asked her if I could take some slide pictures of her radiographs and other imaging and videotape some of her clinical examination findings. She started laughing at me, saying I was too late: Dr. Cox had already done all that. I couldn't help it; I laughed with her. At the next meeting I presented with Dr. Cox, he was using her as the model for his examination demonstration! My handiwork was demonstrated, too. At the end of the meeting, I found her showing off my "bikini" incision in the back of the room, so we both benefitted from the good work we did for this patient!

And so goes my longstanding relationship with Dr. Cox. We have educated each other about our respective fields and have worked side-by-side on many cases to the benefit of our patients. One of the first patients referred to me on my arrival in Fort Wayne was from Dr. Cox. The patient came to my office with a most concise letter of introduction: accurate history, specific time of pain onset, thorough medical history, detailed clinical examination findings, astute results of imaging, and an educated, well-founded diagnosis. I was impressed that this chiropractic physician knew when to refer the patient to a medical specialist, was confident in his diagnosis, and had the desire to do what was best for his patient. Patient satisfaction is high with these types of referrals as they raise confidence in both practitioners. This case and its letter of introduction helped to establish a good rapport between Dr. Cox and me that has lasted close to 25 years.

My undergraduate as well as medical and surgical training at Indiana University and residency programs at Georgetown Medical School and in the U.S. Navy during the Vietnam War prepared me well for medical practice and neurosurgery. I started practice knowing the scientific basis of medicine, down to the molecules and atoms, but soon found

out that not all beneficial care can be explained away by scientific methods. In developing my practice, which now includes six neurosurgeons (one of whom is my eldest son, Jeff) and eight neurologists, all top-notch physicians, I have tried to steer them beyond the strictly scientific to acknowledge the good that comes from the care beyond traditional scientific explanation. During my training in medicine and neurosurgery, there was very little talk of alternative care for back pain, or chiropractic care for that matter. Since then, I have watched alternative care, particularly chiropractic, slowly come into the mainstream of medicine. Most of medicine is more realistic and accepting of alternative therapies, especially in the realm of back pain management. Most back pain can and should be treated conservatively. I have seen many cases of good chiropractic care result.

Although medicine has slowly come to accept chiropractic, it has been a bit too slow in sharing its resources. I am most proud of the fact that I have been able to open doors to Dr. Cox in our local medical community. Dr. Cox has responsibly demonstrated that he knows when to refer patients for further medical and imaging testing, and I found no reason why he should have to be second guessed when sending a patient for tests. I ensure that he had cooperative, easy access to radiographic and imaging facilities as needed. Further, as is his reputation, Dr. Cox reads medical literature voraciously, but occasionally has trouble gaining access to it locally. After hearing about his, I made sure that the doors of local hospital libraries were open to him. I always get a thrill when I drive into the parking lot at the hospital on Wednesday afternoons and see Dr. Cox's car with the "L5S1" license plate framed with the slogan "discover chiropractic." No one knows back literature and research better than Dr. Cox, and I am proud to be able to ensure access, access that allows him to stay on top of the research literature and to share it via his writings and lectures around the world.

I have watched parts of Dr. Cox's lectures before and after my presentations at his courses and read his books. His presentation of material is the best in back pain management training. Dr. Cox disseminates more knowledge about back pain mechanics and diagnosis in his seminars than in other medical and neurosurgical CME training courses I have attended. He takes the highly scientific material he reads weekly and converts it into practical application.

Dr. Cox uses that same practical presentation style demonstrated in lecturing in his writing of this textbook. He provides all the scientific research findings accurately, descriptively, and practically so that a practitioner—chiropractic, medical, or otherwise—can easily relate to the new material. In describing the diagnosis of disc and back problems, Dr. Cox is most vivid, using illustrative x-ray studies and detailed case presentations to exhibit the diagnosis protocol. The algorithms of decision-making are in the simplest yet most detailed of formats. The physician following the Cox protocol outlined in the algorithms can confidently handle the patient's case without the fear of over-treating or mistakenly handling a case alone that may need co-management with another physician. Distraction treatment protocols are precisely portrayed in writing and in pictures to help both the practitioner perform the distraction technique and the patient understand how the technique will help manage his or her back pain condition, for, as Dr. Cox states, back pain is rarely cured but it can be controlled when all parties involved in the case work together.

After years of collaboration and my seeing the positive results of chiropractic management, I sent my younger son, Kenny, to Dr. Cox's office when he began considering a profession, to observe the quality of care that Dr. Cox offers his patients. I now proudly support my son in his choice to become a chiropractor and look forward to working with him and encouraging him to practice chiropractic in the way that Dr. Cox does, using the gentle, nonforce, distraction protocols for the relief of his patients' pain.

In every profession, be it medical, legal, entrepreneurial, or chiropractic, I have found those who strive to move it forward and keep it on the cutting edge. Dr. Cox is one of those people, and he shares his knowledge, protocols, and cases within this text as an example of successful, conservative, chiropractic patient care.

Rudy Kachmann, M.D.
Neurosurgeon
Fort Wayne, Indiana

Low Back Pain, the most common reason for seeking help from a health care provider in the dusk of the twentieth century, is a topic worthy of the persistent penchant of a Dr. James Cox.

As a resident in radiology and a gross anatomy laboratory assistant at National College of Chiropractic in the early 1970s, I had the privilege to assist Dr. Cox in dissecting and photographing the structures of the low back in preparation for his early lectures. He never tired of the thirst for more knowledge, a clearer understanding, and a better picture. Tenacity led to quality, and quality has asserted itself into the work of Dr. Cox in the low back.

But what about this "universal joint" of the body, as Dr. Joseph Janse would often make reference? What happened to this joint when in the antediluvian periods of the Earth's history, man decided to stand up and be different, or was man this way from the beginning? An answer we must await, but in the meantime, Dr. Cox has taken to a meticulous study of this incredible feature of upright bipedism. In no other text will you find such complete and complex coverage of the most difficult and challenging clinical and biomechanical marvel of the human body.

The reader will relish the treasures confined within the binding of this text. The teacher will have need for no other text in helping students master this subject. The student will be enriched beyond measure for every moment spent digesting morsel after morsel of wisdom and intellect. The clinician, ever challenged by this clinical syndrome, will return numerous times to this feast of practical information from which competence and confidence for patient care can be garnered.

To neglect this text is to cover the candle with a basket. Dr. Cox has placed his candle on the hilltop so we may all see. To see we must open our eyes and read what he has prepared for us. The feast is before us but it is our duty and opportunity to eat. I encourage all to become partakers at the table of low back pain instruction and reap the benefits provided by a master teacher, an experienced clinician, an empathic sufferer, and a sympathetic listener. From each of these perspectives, the low back and its associated pain syndromes are laid bare to their most fundamental elements for each of us to learn from and apply our understanding to benefit our patients.

Thanks Dr. Cox.

Reed B. Phillips, D.C., Ph.D.
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PREFACE

The sixth edition of *Low Back Pain: Mechanism, Diagnosis, and Treatment* contains 8 years of updated research in the care of low back pain. Astounding changes have occurred in that period, such as evidence that ergonomic programs, after decades of research, show no clear evidence that they can prevent back pain; little evidence exists that physical therapy provides long-term benefits for chronic musculoskeletal pain sufferers; epidural steroid injections are of questionable value; and plate and screw spinal fusions are controversial. Magnetic resonance imaging is considered wasteful as a routine procedure. The cost of low back care in the United States continues to rise in both human suffering and dollars.

In this same period, chiropractic has had a positive response in the literature, and research studies regarding its benefits and clinical outcomes have been largely positive. Chapter 1 covers the history and future of chiropractic as I view it and includes a brief history of the evolution of my work with distraction adjusting of the spine, which is methodically explained.

Research has finally advanced in chiropractic with the awarding of two studies by the Health Resources and Services Administration of the Department of Health and Human Services to study the biomechanics and clinical benefits of distraction adjustments of the lumbar spine. The first grant was awarded in 1994, entitled "Biomechanics of the Low Back Flexion-Distraction Therapy" and the second was awarded in 1997, entitled "Flexion Distraction vs Medical Care of Low Back Pain." Both studies are joint grants to National College of Chiropractic and Loyola Stritch School of Medicine. Ram Gudavalli, Ph.D., of National College, is the principal investigator of both studies, and in Chapter 8 he describes the research that has been completed in these studies at the time of publication of this textbook. Dr. Gudavalli's chapter is a historic and valuable addition to this textbook and to chiropractic history.

In Chapters 2 and 3, I update research literature in the biomechanics and neurophysiology of low back pain and neural compressive and chemical irritation. Chapter 4 covers the most recent material on the diagnosis, clinical features, and treatment of spinal stenosis. Chapter 6 addresses the transitional segment, Chapter 13 covers facet syndrome, and Chapter 14 on spondylolisthesis represents the latest literature on these conditions that I have collected during the previous 8 years.

Chapter 11, written by David Wickes, D.C., of National College, furnishes the practitioner a very ready outline of diagnostic tests to be ordered for pathologies causing low back pain. This chapter is very thorough but clinician friendly and usable. It will be appreciated when laboratory testing is needed and clear steps laid out for the doctor to follow.

Chapter 12 specifically covers the clinical and home treatment of the patient with low back and sciatic pain. Chapter 9 is a new and very detailed protocol of the principles, biomechanics, anatomic changes, and application of distraction adjustments of the lumbar spine for all its diagnoses. It is an anchor of knowledge of this textbook because it represents the clinical application of distraction adjustments for the doctor of chiropractic. It will be a constant source of therapeutic advice on manipulation and adjustment of the low back pain patient. These two chapters represent my clinical approach to the diagnosis and treatment of low back and sciatic pain.

Chapter 10 covers diagnosis of the low back pain patient, and in this chapter I detail the history, examination, clinical decision-making and therapeutic algorithms, and literature support for the performance and interpretation of standard low back tests in chiropractic today. It focuses on excellence of diagnostic testing leading to a flow chart instruction to arrive at the diagnosis of the patient's condition.

Chapter 16, written by James M. Cox, II, D.C., clearly illustrates the importance of the mental state in treating low back pain as the psychological side of low back pain is discussed. The depression of chronic low back pain, patient coping strategies, detection, and treatment by the physician are shown for practitioner clinical use.

Chapter 7 is the subject of fibromyalgia, written by Lee J. Hazen, D.C. This excellent chapter leads the practitioner in an understanding of the neuroendocrine and psychological basis for this somewhat controversial diagnosis and even more controversial therapeutic condition.

Chapter 15 is a great addition to this textbook because of the rehabilitation interest for the low back pain patient. Scott Chapman, D.C., gives maximal effort to furnish the general practitioner the tools to use for the practical application of rehabilitation in the clinic. This chapter is a very strong addition to this sixth edition and is a vital part of today's managed care treatment of back pain.

Sil Mior, D.C., accepted the challenge of bringing the literature to the chiropractic practitioner on the sacroiliac joint. Along with the brilliant anatomy of Chae Song Ro, M.D., Ph.D., Dr. Mior furnishes this vital subject in the general practice of chiropractic to the practitioner—the sacroiliac joint anatomy, biomechanics, and adjusting procedures.

This book is intended to be a clinical instrument for use by the chiropractic physician in daily practice. It is practical, everyday knowledge that can be used to stimulate excellent patient care and the best of clinical outcomes. Lastly, it is my hope that it serves as a stimulus to other chiropractic doctors to excel and produce a better seventh edition.

James M. Cox, D.C., D.A.C.B.R.

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ACKNOWLEDGMENTS

Practicing chiropractic has required endurance of less than full public awareness and support of the education and contribution of the chiropractor in modern healing. It has been an intense drive and motivation for me to place my profession in the mainstream of healing so that it would be accepted and understood for its gift to humanity. History will respect that modern chiropractic was maligned by its detractors and abused by its proponents, but in the end it proved to be a significant segment of the healing arts world. I am privileged to be able to contribute to my profession with this textbook.

This textbook is a true gift and sacrifice of my incredible family. This book acknowledges the efforts of the most important person in my earthly life, my best friend and confidante, my wife Judi. My intense drive to place chiropractic in its deserving posture has cost my family my time and attention, but more than that the endurance of my frustration and neglect because of the awesome personal commitment I undertook. As I complete this book I apologize to my wonderful wife Judi, and to my four children—Julie, Jill, Jim, and Jason—for the shortcomings I brought you as a husband and father. The statement that a woman stands behind every successful man is proved in my life because all direction and effort has sprung from or involved Judi's brilliant understanding of our profession and my strengths and weaknesses as a man. This book is dedicated to her unacknowledged sacrifice in our marriage, profession, and lives together to make this effort possible. I pray for the time and strength to show you how much I love you for standing by me as I worked as an architect of chiropractic.

Julie Cox-Cid is a unique and gifted human being and it is awesome to think she is my daughter. In 1992, while she was an English Literature high-school teacher, I was able to convince her that her great talents would be equally challenged working with me. This proved to be very true and her contribution to this book is an example of her literary writing abilities. My profession and I are both very fortunate to have her support. Thank you, Julie.

I have woven my professional and private life after a man who taught me anatomy, chiropractic technique, humility, love, perseverance, accomplishment of the impossible, power with gentleness, and sacrifice for the good of the majority. Joseph Janse, D.C., past president of the National College of Chiropractic, is that man. His leadership and principled life molded such leaders in our profession as Reed Phillips, Terry Yochum, Jim Winterstein, and so many others. To him I owe the fact that this book is written.

James M. Cox, D.C., D.A.C.B.R.

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CONTENTS

Foreword, v
Preface, ix
Acknowledgments, xi
Contributors, xiii

1 **Chiropractic and Distraction Adjustments Today, 1**
James M. Cox

2 **Biomechanics of the Lumbar Spine, 17**
James M. Cox

3 **Neurophysiology and Pathology of the Nerve Root and Dorsal Root Ganglion, 131**
James M. Cox

4 **Spinal Stenosis, 169**
James M. Cox

5 **The Sacroiliac Joint, 209**
Silvano A. Mior
Chae Song Ro
Dana Lawrence

6 **Transitional Segment, 237**
James M. Cox

7 **Fibromyalgia, 251**
Lee J. Hazen

8 **Biomechanics Research on Flexion-Distraction Procedure, 261**
MR Gudavalli

9 **Biomechanics, Adjustment Procedures, Ancillary Therapies, and Clinical Outcomes of Cox Distraction Technique, 273**
James M. Cox

10 **Diagnosis of the Low Back and Leg Pain Patient, 377**
James M. Cox

11 **Laboratory Evaluation, 509**
David Wickes

12 Care of the Intervertebral Disc Patient, 527
James M. Cox

13 Facet Syndrome, 591
James M. Cox

14 Spondylolisthesis, 611
James M. Cox

15 Rehabilitation of the Low Back Pain Patient, 653
Scott A. Chapman
Carol L. DeFranca

16 Psychological Perspectives in Treating Low Back Pain, 679
James M. Cox, II

Addendum A: Literature Update, 689

Addendum B: Biomechanics Research, 707

Index, 713

Chiropractic and Distraction Adjustments Today

James M. Cox, DC, DACBR

chapter 1

Chiropractic practice is an expression of life commitment to society. No greater treatise could be written than to be remembered, in some small way, as an architect of chiropractic in your time.

—James M. Cox, DC

HISTORY OF THE DEVELOPMENT OF COX DISTRACTION MANIPULATION

Why the Creation of Cox Distraction?

Simply, one of my first patients: a young, 24-year-old woman came into my and my stepfather's office in severe pain, leaning to her right at the thoracolumbar spine, complaining of pain radiating down her right leg along the fifth lumbar dermatome. This was 1964: the disc did not have a nerve supply according to the literature of the day, and I was not aware of sciatic scoliosis defining a lateral, medial, or subrhizal disc lesion nor did I understand the ramifications of ischemic hypoxia and axoplasmic flow of a nerve. Stenotic factors of the vertebral and osseoligamentous canals were not well known to me nor to medicine at large.

I took an x-ray, and her fourth lumbar vertebra was in right lateral flexion subluxation, which called for a corrective adjustment: the traditional side posture positioning where I placed my pisiform contact on her fourth lumbar lamina and made the usual thrust with her thigh in the usual leveraged flexed posture—incidently, an adjustment I used on patients with good clinical result previously. This time, instead of cavitation of facet joints, my adjustment was met with muscle spasm, my contact hand bounced off her spine, she yelled in pain, I started to sweat, and her family carried her out of my office and to the hospital for surgery for a ruptured fourth lumbar disc the next day. I was devastated. The adjustment did not work as I was taught it should. Could I have missed something in school?

In school, I was an intense (some might even say my approach to learning was a bit crazy) student. I studied hard; everything about chiropractic and the human anatomy fascinated me. In 1963, I graduated valedictorian which, as evidenced by the above scenario, meant nothing. Einstein once said and was right: The knowledge acquiring period *begins* upon graduation.

My stepfather, John C. Rodman, DO, DC, took me into his practice in Fort Wayne, Indiana, and fostered my knowledge acquisition. After the above case, he said: "Son, you may well be in the way of learning." I absolutely agreed.

He introduced me to osteopathic textbooks written by Taylor, Stoddard, Naylor, and other authors. These authors discussed the techniques of an osteopathic physician, John McManis, DO, who developed techniques of treating spinal problems under traction. As I started to study these techniques, I recalled the teachings of my chief of staff at the National College of Chiropractic, Floyd Blackmore, DO. When a difficult, painful low back case came into the clinic, Dr. Blackmore would lead us interns to the basement and say: "Come with me, and we will treat this patient differently."

In the basement was a McManis osteopathic manipulation table on which he would treat the patient until the acute phase of pain was over when he would turn the patient back over to the intern for care. Seldom, if ever, did he have the intern use the McManis table. Strangely enough, it never really struck me as important until after my encounter with the above patient, nor did I realize the seriousness of back pain.

Finally, back pain got my attention, and the oddity of my training, the McManis treatment, came into my repertoire of patient care. I sought out a McManis table—which was an all-purpose table equipped for ear, nose, and throat examinations; for surgery; and for gynecologic examinations—for my own use. An osteopath's widow in Michigan who had once told me I could have the table if I did not charge her a hauling

fee to get it out! It was a monster of a table and very heavy. I brought it back to Fort Wayne and began using it. The trouble was convincing patients that it was not a torture device! It was covered in horsehair and my rigging it up with pillows and thoracic spine straps to hold the patient's torso while lumbar traction was administered did not look inviting. I persevered though and continued to study and perfect a technique using the table. Eventually, more and more patients requested this type of adjustment.

Something Still Missing

The chiropractic adjustment procedures I learned in school were as important as ever to me. I used them regularly, but they were difficult to do on the McManis table (the gynecologic stirrups, among other things, got in the way).

Further, despite positive patient results and satisfaction, I used this manipulation only on difficult, stubborn, or very painful back conditions as had been demonstrated by Dr. Blackmore. With time and increased experience, I asked: "If this technique helps these difficult cases, why could it not also help the average low back conditions seen in our practice?"

That question fostered the evolution of what I termed in the early 1970s, "Flexion Distraction Manipulation," which changed my life. I never dreamed that I would follow the course that my professional life has taken as a result of patient satisfaction and, later, colleague inquiry. Local colleagues began to hear what I was doing and requested that I treat them and teach them how to do the same for their patients.

The old McManis table was cumbersome to use and difficult to find. However, I met with Jim Barnes, a man who owned a machine shop in Fort Wayne. I presented my basic ideas to him, and together we produced a new instrument that blended osteopathic manipulation concepts with chiropractic adjusting concepts: the Chiro-Manis (a term representing *chiro*practic and Mc*Manis*) table. With this, too, I gained a new title—"entrepreneur." Together, Barnes and I made and marketed the Chiro-Manis table from 1973 until 1984 when Williams Manufacturing Company (now Williams Healthcare Systems), manufacturers of Zenith tables, took over the engineering and construction of what is now called the Zenith-Cox table.

As an extension of my new entrepreneurial role, I offered courses to local colleagues on how to perform this new technique. I have always stressed that distraction manipulation is not intended to replace any of the valid, successful techniques of historic chiropractic, but rather it is an additional therapy in the armamentarium of the chiropractic physician in his or her daily practice. I uphold distraction manipulation in the same light today.

Personal Experience with Disc Herniation and Sciatica

During the 1970s and early 1980s, I passionately studied back pain, its mechanism and its biomechanical causes. I shared my positive patient case results with all who would listen. Although I publicized the fact that chiropractic had so much to offer back pain sufferers, I lived many of the days for those 10 years in severe low back pain myself.

Some days I believed my lower back hurt worse than the backs of my patients. Some days it was agonizing and nearly impossible to bend over to treat my patients. I refused to let *pain* stop me. The only thing that kept me going was a colleague's treating me with flexion distraction.

In April 1981, my education in low back and sciatic pain was magnified beyond my desires and expectations. My passion in studying back pain and sciatica spilled over into my "recreational" activities. I love farm life; I had a gentleman's farm with my family, although I had little free time for either. I had hired some men to put up a fence for my cows. While "helping" them unload fencing and removing an end post from the ground, a sudden sharp pain shot through my low back. The following morning, while bending over to wash my foot, I felt a sudden tearing in my low back that sent pain down my right leg, through the calf, along the bottom of the foot to the little toe. I no longer felt any pain in my back, but had the most unbearable pain in the leg, which lingered. I could not believe it. I had spent so much of my life—weekends away from home, weekdays treating patients, and weeknights studying—teaching about the diagnosis and treatment of low back pain. Now I was afflicted with severe pain and totally unable to function normally—a living example of a victim of a prolapsed lumbar disc. What a frightening, enlightening, and confusing nightmare. I was about to learn more about low back and leg pain than I had ever read or taught.

For the next 3 weeks, I was treated with distraction manipulation, positive galvanism into the L5–S1 right posterolateral disc space, acupressure massage of the low back and right lower extremity, rest, alternating hot and cold packs to the low back and leg, and, in the third week, side posture adjustment. My wife spent many a day and night taking me to the clinic for therapy. Barely able to walk, I still went to my office to treat patients. Nothing improved, but I still refused to be stopped by pain. I continued seeing patients at the office, barely able to walk or stand myself. I even gave a lecture in Chicago where I had to be propped up on the podium in order to speak.

The leg pain worsened, although low back pain did not recur. In the fourth week, however, I experienced numbness of the perineum, anal sphincter weakness, and urinary bladder difficulty—cauda equina syndrome. I had not wakened from nightmare, but was pushed further into it. I thought and still believe that God was saying: "You think you know something? Take this and learn from it."

The cauda equina symptoms got my attention. I called Rudy Kachmann, MD, a friend and neurosurgical colleague. We consulted and decided surgery was required now: My straight leg raise was positive at 10 degrees. My calf muscle had atrophied, and I could not walk on my right toes. The right Achilles reflex was totally absent. I had not slept in a month.

In 1981, myelography was still the gold standard of diagnosis. One month after the onset of pain, I had a myelogram performed, and it revealed a huge L5–S1 fragment. Dr. Kachmann

performed a microdiscectomy procedure on me. That night, I walked without pain. Starting urination was a bit difficult, but it became normal.

That was a great learning experience: one that made me a better doctor. I empathize with my patients and feel their pain and frustration in dealing with such a problem. My situation fell into the 5% of cases that develop neuropraxia and which demand surgical relief.

Since 1981, I have lived by my own rules. In my "middle-age" (50s), I am in better physical shape than I was at half my age. I do my own exercise program, practice ergonomics, treat patients from alternating sides of the table, treat smarter and not harder, and get treated with distraction manipulation regularly. This regimen allows me the flexibility and strength to maintain my practice, research, and lecture schedule. My L4--L5 disc showed a slight protrusion in 1981, which makes it the next vulnerable disc to prolapse if I do not maintain conservative care and good health. Pain and suffering taught me to take care of myself. I no longer have a farm nor do I lift heavy fence posts. I let others do their jobs. I do, however, devote my professional energy to the study of low back pain and strive to help my colleagues and their patients care for this disabling condition.

Maturation of Distraction Manipulation for Chiropractic

In 1990, I turned over my work to the National College of Chiropractic, and a certification course for the chiropractic profession in the use of distraction manipulation was born. This is a 36-hour postgraduate course of study with a written and practical examination that elevates a Doctor of Chiropractic to the status of a Certified Distraction Practitioner with a listing in the referral directory of chiropractors who have achieved this status. The success of this certification course is beyond my expectations. The wave of field doctors and new graduates entering into the program is gratifying. A referral network of distraction doctors is growing annually, which benefits both doctors and patients. In addition, the distraction manipulation technique course is also core or elective curriculum or taught in technique classes at most chiropractic colleges. This offers the student an introduction to distraction manipulation so that he or she can decide whether to use it in clinical practice.

Since 1973, I have lectured on distraction manipulation principles and practice throughout the United States, Europe, and Japan. Other certified instructors are teaching my work throughout the United States as well. Certainly, the 15 other copies of my manipulation instrument being marketed are a testimonial to the success of the procedure.

It was a combination of my ill-treatment result of the young woman with an L4--L5 disc herniation and the teachings of Drs. Rodman and Blackmore that opened my mind to the possibility of a different approach to treating low back and sciatic pain—namely manipulation under traction, a technique that has become known as "Cox Distraction Manipulation." Further, my personal fight with a sequestered L5–S1 disc has directed my understanding and approach to caring for patients and teaching of this technique.

Evolution of Cervical Spine Distraction Manipulation

Personal experience and/or involvement in painful problems bring change and improvement. My low back pain perfected my doctoring; my wife's cervical spine pain brought about the latest chapter in distraction manipulation: the cervical spine distraction headpiece. This unit allows the same principles of distraction adjustments that have been so successfully used in the lumbar spine to be adapted to the cervical spine.

My wife, Judi, developed right arm C6 dermatome radiculopathy in 1984. She told me, in not debatable terms, to develop a technique to treat cervical spine disc problems like I had done in the lumbar spine. After much procrastination, and some disturbed home life, I set about creating the cervical spine distraction technique and headpiece with the engineering department of Williams Healthcare Systems. Williams collected 338 patient cases from five clinical trials for the U.S. Food and Drug Administration (FDA) registration. As a result of relieving Judi's arm pain and the success of the clinical trials, this instrument has been available for professional use in clinical practice by the chiropractic profession since 1992.

In the final analysis, this technique developed from need—a need for a technique that complements traditional chiropractic adjustment procedures for those patients who will respond best to adjustments under traction.

THE SIXTH EDITION OF THIS TEXTBOOK

Why a sixth edition of this textbook? Primarily, the volumes of literature emerging daily in the mechanism, diagnosis, and treatment of low back pain make a new edition mandatory. Chiropractic physicians must be informed of these developments. Also, they must see the bridge between knowledge and its application, a task that is humbling to me as an author, but one which I enjoy with an almost bizarre feeling of excitement.

What Does the Literature Say About Distraction Manipulation?

The first recorded case of low back pain attributed to an occupation dated at about 2780 bc, when Imhotep, an Egyptian physician treating construction workers at the pyramid in Saqqara, described spinal strain (1), and today medicine struggles to improve on the definition and care of this condition.

Interest and clinical benefit are seen in manipulating the human spine under distraction. Two thirds of Los Angeles College of Chiropractic graduates (2) and 53% of practicing chiropractic physicians in the United States use the Cox Distraction technique (3). The Cox Distraction technique is the only one of its kind that has been described in a reviewed text and a number of well-respected, peer-reviewed journals; also, "of those

professing to use distractive procedures, only Cox has performed any statistical analysis on clinical effects for various conditions" (4). A 576-case study of low back and sciatica patients treated with distraction procedures showed 76% had good to excellent relief and 10% fair to poor results. The remaining 14% stopped care or were surgically treated (4).

Logan College students reported on the academic and clinic use of the Cox Distraction manipulation procedures and 100% of them reported feeling the course was more interesting, professional, understandable, rational, and the instructors more capable than those for any other course they had taken. Eighty-five percent of the students said they would incorporate the technique into their practices, and 15% said they would use it as the only technique in their practice (5).

Palmer College of Chiropractic West reported a prospective study randomly assigning 67 patients with chronic low back pain of at least 6 months duration to one of four therapy groups: (*a*) distraction manipulation, (*b*) inverted gravity traction, (*c*) detuned transcutaneous electrical stimulation (TENS), or (*d*) a waiting list. Objective and subjective study showed that distraction manipulation and inversion traction were superior to placebo and a waiting list control group. Chiropractors trained in both these techniques effectively treat patients with low back pain (6).

The success of the distraction manipulation technique in treating an L5–S1 herniated disc in a 28-year-old Soviet dancer, after rotation adjustment proved impossible due to muscle splinting, is reported from the Los Angeles College of Chiropractic (7). Cleveland College of Chiropractic, Los Angeles, reported a case of a 24-year-old man with an unstable lumbar spine, hypoplastic lumbosacral facets, lumbar spina bifida occulta, a transitional vertebra, and a lumbosacral disc protrusion, which was asymptomatic 6 weeks after injury. The authors of this paper felt this may be the first published report of distraction manipulation in treating the unstable segment (8).

The fact that peers in my profession were positively influenced by the distraction manipulation as described in this and earlier editions of this textbook encouraged me to take on the project of writing another edition. Of course, the insistence and encouragement of Williams & Wilkins also was an influence.

FACTS ON PATIENTS SEEING CHIROPRACTORS

Ninety four percent of manipulative therapy performed in the United States is performed by chiropractic doctors. For the past 50 years spinal manipulation has been equated with the practice of chiropractic and, in part because of this, the use of spinal manipulation has been labeled an unorthodox treatment by the medical profession. Spinal manipulation has been cited to be of short-term benefit in some patients, particularly those with uncomplicated, acute low back pain, whereas data are insufficient to comment on its efficacy on chronic low back pain (9).

About 5% of the population see chiropractors annually at a rate of approximately $2.4 billion (10, 11). About 45,000 chiropractors practice in the United States. Thirty-two to forty-five percent of chiropractic care is for low back pain with the average number of visits being 5 to 18 per episode (10–15).

Chiropractic care is most frequently used by persons who are white, middle-aged, and employed (10–12). High school graduate level persons use chiropractic care more often than other academic levels; great differences are seen by geographic area in the utilization of chiropractic services (11).

One third of patients who seek care for back pain choose a chiropractor. Chiropractors were the primary care provider for 40% of back pain episodes, and they were retained as the primary provider by a greater percentage of their patients (92%) who had a second episode of back pain care than were medical doctors (16).

Rising Use and Acceptance of Chiropractic in the United States

Of persons seeking care for low back pain in North Carolina, 59% received care from a physician, 34% from a Doctor of Chiropractic (DC), and 7% from other professionals (nurses, physical therapists) as the first provider for an episode of acute pain. An additional 5% sought care from a DC after first seeking care from an MD. Adults who were employed, insured, younger than 60 years of age, and more wealthy favored chiropractors. Satisfaction with care was higher in patients who saw DCs; 96% of individuals who saw a DC described the treatment as "helpful," compared with 84% of those seeing MDs ($P = 0.03$) (17). Younger age, male gender, and non–job-related pain correlate with the decision to seek care from a chiropractor (18).

Unconventional Therapy in the United States

The frequency of use of unconventional therapy in the United States is far higher than previously reported (19). Unconventional therapies are defined as medical interventions not taught widely at U.S. medical schools or generally available at U.S. hospitals. Examples are acupuncture, chiropractic, and massage therapy.

Use of unconventional therapy is significantly more common among people 25 to 49 years of age; is significantly less common among blacks; is more common among people with some college education than among those with no college education; significantly more common among people with annual incomes greater than $35,000; and significantly more common among those living in the western part of the United States.

Frequency of use of unconventional therapy is highest for back problems, anxiety, headaches, chronic pain, and cancer or tumors. Almost 9 of 10 respondents who saw a provider of unconventional therapy in 1990 did so without the recommendation of their medical doctor; 72% of those who used unconventional therapy did not inform their medical doctor of it.

Most respondents (55%) paid the entire cost of their unconventional therapy visits out of pocket. Third-party payment was most common for the services of herbal therapists (83%),

providers of biofeedback (40%), chiropractors (39%), and providers of megavitamins (30%). In 1990, the total projected out-of-pocket expenditure for unconventional therapy plus supplements was $10.3 billion.

An estimated one of three persons in the U.S. adult population used unconventional therapy in 1990. The estimated number of visits made in 1990 to providers of unconventional therapy was greater than the number of visits to all primary care medical doctors nationwide, and the amount spent out of pocket on unconventional therapy was comparable to the amount spent out of pocket by Americans for all hospitalizations. Roughly one of four Americans who see their medical doctors for a serious health problem may be using unconventional therapy (19).

Eighty-nine Israeli family physicians reported that 54% thought complementary medicine (chiropractic, naturopathy, hypnosis, homeopathy, and eastern medicine) was helpful and 42% had referred patients for it, with most feeling it should be incorporated into medical practice (20).

Potential Users (81 Million) of Chiropractic Services in the United States

The American Chiropractic Association data show:

1. Of over 3.5 million (3,560,000) privately insured individuals aged less than 65 years, the chiropractic profession delivered 75% of all services that included therapeutic manipulation.
2. Of Americans aged 18 years and older 29% (55 million people) have used chiropractic services.
3. Of all adults aged more than 18 years 10% (18.5 million people) have used chiropractic services in the last year, and 19% (more than 35 million people), within the last 5 years.
4. Chiropractic services were sought by 65% for such self-reported low-back disorders as muscle spasms, sciatica, pinched nerves, and ruptured discs.
5. Nonusers were asked if they would see a Doctor of Chiropractic for a condition they treat, and 62% responded favorably. This percentage of potential users projects to more than 81 million adults nationally (21).

Eighty percent of patients are satisfied with chiropractic care; 90% felt their treatment to be effective; and 80% felt the cost was reasonable (21).

Patients Are Satisfied with Chiropractic Care

Patients were most satisfied with the accessibility of their doctors and least satisfied with the financial aspects of treatment, especially those who reported lower incomes and no insurance coverage. A slightly higher degree of dissatisfaction was reported by a small percentage (12%) of patients who also reported either no improvement or minimal improvement in their health problem following chiropractic care.

Patients expressed high levels of satisfaction with their doctors and the care they received. Although women were slightly more satisfied than men, other patient characteristics (e.g., level of education, income, employment status, or previous chiropractic care) did not influence response means (22).

Survey of Chiropractic Practitioners' Education, Practice Procedures, and Patient Perception of Care (3)

A Gallup poll reported that 90% of patients seeing chiropractors felt chiropractic treatment was effective, more than 80% were satisfied with their treatment, nearly 75% felt most of their expectations had been met during their visits, 68% would see a chiropractor again for treatment of a similar condition, and 50% would likely see a chiropractor again for other conditions. Sixty-two percent of nonusers stated that they would see a Doctor of Chiropractic for a problem applicable to chiropractic treatment, 25% reported that someone in their household had been treated by a chiropractor, and nearly 80% of those had been satisfied with the chiropractic treatment received.

Chiropractic Practitioner/Respondent Demographic Summary (3)

Results of the National Board of Chiropractic Examiners Survey indicated that only four techniques were used by most practitioners: Diversified, Gonstead, Cox, and Activator. All other techniques were used by 43% or fewer respondents. Results also indicated that the responding practitioners used an average of 5.7 specific techniques in their practices (Table 1.1).

PHYSICAL THERAPY'S VIEW OF CHIROPRACTIC AND SPINAL MANIPULATION

Manual Therapy: Manipulation Versus Mobilization (23)

Mennell stated: "Beyond all doubt the use of the human hand, as a method of reducing human suffering, is the oldest remedy known to man; historically no date can be given for its adoption."

The American Physical Therapy Association has the following position on manipulation: "Manipulative techniques by licensed physical therapists in evaluation and treatment of individuals with musculoskeletal dysfunction has [sic] always been an integral component within the scope of practice . . .

1. Manipulation in all forms is within the scope of practice of a licensed physical therapist.
2. The force, amplitude, direction, duration, and frequency of manipulation treatment movements is a discretionary decision made by the physical therapist on the basis of education and clinical experience and on the patient's profile.
3. Manipulation implies a variety of manual techniques which is not exclusive to any specific profession" (23).

Physical therapists define mobilization as the act of imparting movement, actively or passively, to a joint or soft tissue. Therapists may want to avoid the term "manipulation" because of its strong association with the chiropractic profession. Ma-

Table 1.1

Chiropractic Practitioner Demographic Summary (3)

Gender

Male 86.7% Female 13.3%

Ethnic Origin

White (not Hispanic)	95.5%	Native American	0.2%
Hispanic	1.6%	Filipino	0.2%
Other	1.2%	Alaskan Native	0.0%
Asian	0.8%	Pacific Islander	0.0%
Black (not Hispanic)	0.5%		

Highest Level of Nonchiropractic Education

Baccalaureate degree	46.5%	Other	6.0%
Associate degree	24.1%	Master's degree	5.1%
High school diploma	16.2%	Doctoral degree	2.1%

Specialty Board Certification

None/does not apply	74.6%
American Board of Orthopaedics	9.9%
Other	9.5%
ACB of Sports Physicians	4.2%
ACB of Radiology	1.9%
ACB of Neurology	1.3%
ICA College of Thermography	1.0%
Chiropractic Rehabilitation Association	0.7%
ACB of Nutrition	0.6%
ACB of Internists	0.5%
ICA College on Chiropractic Imaging	0.4%
ICA Council on Applied Chiropractic Sciences	0.3%

Institution Granting Degree

Palmer	27.7%	Western States	3.2%
National	11.6%	Sherman	2.9%
Life	9.0%	Other	2.8%
Logan	8.0%	Palmer West	2.2%
New York	7.4%	Life West	1.3%
Los Angeles	6.6%	Pennsylvania	0.8%
Northwestern	4.5%	Parker	0.7%
Cleveland-KC	3.9%	Southern California	0.3%
Cleveland-LA	3.5%	Canadian Member	0.1%
Texas	3.5%	Foreign/overseas	0.0%

Patient Demographics Reported in Survey

Gender

Male 40.7% Female 59.3%

Age

< 17 years	9.7%	51 to 64	21.2%
18 to 30	19.1%	> 65 yrs	13.3%
31 to 50	36.7%		

Ethnic Origin

White	65.0%	Native American	3.0%
Hispanic	10.3%	Filipino	2.4%
Other	0.9%	Alaskan Native	0.3%
Asian	5.6%	Pacific Islander	1.4%
Black	11.3%		

Occupation

Tradesman/skilled labor	19.1%
White collar/secretarial	16.5%
Homemaker	13.8%
Unskilled labor	12.0%
Executive/professional	11.9%
Retired or other	11.7%
Student	7.6%
Professional/amateur athlete	7.4%

Chiropractic Treatment Procedures

Primary Approach

Full spine	93.3%
Upper cervical	1.7%
Other	5.0%

Adjustive Techniques

Diversified	91.1%
Gonstead	54.8%
Cox flexion distraction	52.7%
Activator	51.2%
Thompson	43.0%
SOT	41.3%
NIMMO/tonus receptor	40.3%
Applied kinesiology	37.2%
Logan Basic	30.6%
Cranial	27.2%
Palmer upper cervical/HIO	26.0%
Meric	23.4%
Pierce-Stillwagon	19.7%
Other	15%
Pettibon	6.3%
Barge	4.1%
Grostic	3.4%
Toftness	3.3%
Life upper cervical	2%
NUCCA	1.5%

Nonadjustive Techniques

Corrective/therapeutic exercises	95.8%
Ice pack/cryotherapy	92.6%
Bracing	90.8%
Nutritional counseling, etc.	83.5%
Bedrest	82.0%
Orthotics/lifts	79.2%
Hot pack/moist heat	78.5%
Traction	73.2%
Electrical stimulation	73.2%
Massage therapy	73.0%
Ultrasound	68.8%
Acupressure/meridian therapy	65.5%
Casting/taping, strapping	48.2%
Vibratory therapy	42.0%

continued

Table 1.1

Chiropractic Practitioner Demographic Summary (3)

Homeopathic remedies	36.9%	Acupuncture	11.8%
Interferential current	36.7%	Other	9.6%
Direct current, etc.	26.9%	Biofeedback	7.1%
Diathermy	26.7%	Paraffin bath	6.9%
Infrared	19.0%	Ultraviolet therapy	3.3%
Whirlpool/hydrotherapy	12.7%		

Reprinted with permission from Haminishi C, Tanaka S. Dorsal root ganglia in the lumbosacral region observed from the axial view of MRI. Spine 1993;18(13):1753–1756.
ACB, American Chiropractic Board; ICA, International Chiropractor's Association; HIO, Hole In One; SOT, Sacro-occipital technique; NUCCA, National Upper Cervical Chiropractic Association.

nipulation, in a general sense, means any manual procedure in which the hands or fingers are used to move a vertebral motion segment (i.e., two adjacent vertebrae and their interconnecting tissues), soft tissue structure, or a peripheral joint (23).

Two types of spinal manipulation have been labeled in chiropractic: nonspecific long-lever manipulation and specific, high-velocity spinal adjustments (24).

Physical Therapy's Effects on Connective Tissue (25)

One of the aims of manual therapy is to permanently elongate soft tissues that are restraining joint mobility through the application of specific external forces. Dense, regular connective tissue is a histologic category of connective tissue that includes ligaments, tendons, fasciae, and aponeuroses. It is important to note that a low level of connective tissue damage must occur to produce permanent elongation. The collagen breakage will be followed by a classic cycle of tissue inflammation, repair, and remodeling that should be therapeutically managed to maintain the desired tissue elongation.

The end result of both inflammation and immobilization is remodeled connective tissue with lower tensile stiffness and a lower ultimate strength than normal tissue. This weakening is caused by the more randomized collagen bundles easily sliding past one another (cross-linking and loss of water), and possibly by the substitution of collagen types that are less strong than the original collagen.

Manual therapy is often used to produce a desirable amount of plastic deformation of connective tissue (microfailure of ligaments, fasciae, and so on) and to produce movement of one joint surface with respect to another (25).

Ideal Ratio of Chiropractors to Population

In Saskatchewan, 366,848 people could be treated by chiropractors if enough chiropractors were available. Saskatchewan needs 391 chiropractors to effectively serve the musculoskeletal problems of the general population. The ideal chiropractor:population ratio is 1:2588. Health care policymakers should design incentives to channel the appropriate patients into chiropractic offices (26).

MEDICAL PHYSICIANS' INTERACTION WITH CHIROPRACTIC PHYSICIANS

Medical Doctors Utilize Manipulation in General Practice

A medical doctor who performed manipulation for the 18 years he has been in practice reports that manipulation is a safe and effective treatment for spinal pain (27).

Medical Practitioners Reluctant to Refer Patients to Chiropractors

Back pain is the second leading reason patients give for visiting physicians, and it is the third most common reason for visiting a family physician. Family physicians care for 38.6% of the patients with acute and chronic back pain, compared with 36.9% seen by orthopedists, 16.9% by osteopaths, and 7.6% by internists (28).

Many physicians, probably a majority, are still reluctant to make specific referrals to osteopaths or chiropractors. A recent study reported that less than 1% of patients were referred to chiropractors by other providers (28).

Physical Therapy Instead of Spinal Manipulation Is Ordered

A national random sample of 2897 physicians showed that of nine listed treatments, only physical therapy, strict bed rest for more than 3 days, and trigger point injections were perceived by a majority of physicians to be effective for patients with acute low back pain. Less than 3% of physicians would have ordered spinal manipulation for any of the hypothetic patients (29).

Osteopaths Treat Somatic Dysfunction with Manipulative Therapy

An osteopathic task force furnished guidelines for the use and documentation of osteopathic manipulative therapy (OMT) as a therapeutic intervention for patients with diagnoses of primary or secondary somatic dysfunction (30).

Many injuries, illnesses, and disease systems are associated with specific areas of musculoskeletal dysfunction, according to the report. Pulmonary system diseases (e.g., pneumonia and bronchitis) often have associated somatic findings at spinal segments T1 through T5. The osteopaths have associated the disease with an ICD-9 code (Table 1.2) (30).

The total patient must be examined so that somatic dysfunction can be identified and treated in all regions of the body as the patient's condition requires and tolerates (31). Osteopaths believe that somatic dysfunction in a single segment or multiple segmental regions may be the chief somatic manifestation of the patient's visceral disease. For example, a patient may have lower gastrointestinal illness associated with viscerosomatic reflex responses at spinal segments T10 and T12. If the physician restricted treatment to those two thoracic spinal segments, improvement probably would be limited. If the physician found somatic dysfunction of the first rib in addition to that of the lower thoracic region, and correctly treated it, the results generally would be more effective (31).

Physicians Encouraged to Refer Patients to Chiropractors

Family physicians who choose to refer their back pain patients to a chiropractor for spinal manipulation do not need to embrace the chiropractic belief system, which differs markedly from that of the family physician. Rather, they need only accept that spinal manipulation is one of the few conservative treatments for low back pain that have been found to be effective in randomized trials. The risks of complications from lumbar manipulation are also very low (32).

Physicians Not Fully Informed of Best Methods to Treat Back Pain

When 2897 physicians from nine different specialties were asked about treatments they would offer hypothetic patients with acute low back pain, sciatica, or chronic low back pain, the most popular treatments were systemic drugs, bed rest, exercise, and physical therapy. Two thirds of the physicians believed TENS, corsets, trigger point injections, and steroid injections to be effective treatments for chronic back pain.

Most of the treatments recommended by these doctors are not scientifically validated. They did not indicate an increasing acceptance of manipulation, although roughly 40% of the physicians who responded to the survey believe manipulation is an effective treatment for acute or chronic back pain (33).

Medical Doctors Lack Extensive Nutrition Training

Medical schools do not teach nutrition. It is not a required course at most of the medical schools in the United States. It has been reported that less than 40% of the medical schools in the United States even offer minimal hours of nutrition training. More than 75% of medical schools do not even require students to take a single nutrition course (34).

COST OF CHIROPRACTIC SERVICES

Chiropractic is Rapidly Growing and Lowering Cost (35)

Chiropractic represents the most rapidly growing segment of the professional health services market. Chiropractic payments

Table 1.2

Guidelines for Diagnostic Related Groups (DRG)/Osteopathic Manipulative Treatment (30)

DRG No.	Disease	ICD-9	Probable Primary Location of Somatic Dysfunction	Reference Page No.
243	Appendicitis	739.2	Thoracic region	192
243	Bronchitis,	739.1	Cervical region	13–51
	acute and chronic	739.2	Thoracic region	192
243	Congestive heart	739.1	Cervical region	55, 56, 66, 71
	failure	739.2	Thoracic region	72, 85, 185
243	Coronary artery	739.1	Cervical region	53–76
	disease	739.2	Thoracic region	
243	Cystocele	739.4	Sacral region	123–127
		739.5	Pelvic region	
243	Hypertension	739.2	Thoracic region	61–64
247	Otitis media, all types	739.0	Head	10, 15
243		739.1	Cervical region	

DRG, diagnosis related group; ICD, International Classification of Diseases—clinical modification.

represent only 1.8% of total insurance payments with payments per chiropractic patient averaging $411 across all plan types (35).

Chiropractic treatment was compared with medical and osteopathic treatment for 395,641 patients with 1 or more of 493 neuromusculoskeletal ICD-9 codes with patients receiving chiropractic care experiencing significantly lower health care costs of approximately $1000 over the 2-year period. The results also suggest the need to re-examine insurance practices and programs that restrict chiropractic coverage relative to medical coverage (35)

Chiropractors' Costs Low

A survey of 11 health conditions, including arthritis, disc disorders, bursitis, low back pain and spinal-related sprains, strains or dislocations, conducted in Virginia showed patients make visits to at least one of six different types of medical care provider (36).

Chiropractic is a lower cost option for several prominent back-related ailments, according to a survey comparing costs of chiropractors versus alternative medical practitioners. This is despite its "last resort" status for many patients. One explanation for this is the lower insurance coverage of chiropractic care. If chiropractic care is insured to the extent other specialists are it may decrease overall treatment costs (36).

Twenty-two studies examined the efficacy or outcome measures including the duration of work loss, period of disability, pain relief, and patient satisfaction with chiropractic treatment for low back pain. Only in one dimension in one study does chiropractic not rank more favorably than medical treatment of low back pain.

The conclusion of this analysis is that chiropractic is mandated to be an available health care option because it is widely used by the American public, and it has been proven to be cost-effective (37).

Australian Study Shows Chiropractic Care Is Cost-Effective

Workers' compensation payments for chiropractic versus medical doctor care were compared in an Australian workers' compensation study. The total utilization rate for chiropractic intervention in spinal injuries was 12%. Payments for physiotherapy and chiropractic treatment totaled more than $25.2 million and represented 2.4% of total payments for all cases. Average chiropractic treatment cost for a sample of 20 randomly selected cases was $299.65; average medical treatment cost per case was $647.20.

Chiropractic treatment seems to be cost-effective in certain conditions but not necessarily because chiropractors encounter patients with relatively less severe conditions. However, a possible limitation to this conclusion is that the measurement of relative percentage treatment costs does not reflect when the intervention was performed or the crossover effects of other interventions (38).

Chiropractic Care Not Always the Least Expensive

Of 8825 visits covering 1020 low back pain episodes in 686 different patients, chiropractors and general practitioners were the primary providers for 40% and 26% of episodes, respectively. Chiropractors had a significantly greater mean number of visits per episode (10.4) than did other practitioners. Orthopedic physicians and "other" physicians were significantly more costly on a per visit basis. Orthopedists had the highest mean total cost per episode, and general practitioners the lowest. Chiropractors had the highest mean provider cost per episode ($264) and general practitioners had the lowest ($95).

The drug costs associated with some chiropractic courses of therapy are surprising because chiropractic is promoted by its professional organizations as a "surgery-free, drug-free" healing profession. Analysis of the claim forms for these drug costs show that they are of two kinds: mineral and vitamin supplements purchased from the chiropractor and prescription drugs purchased from pharmacies.

The advantage that chiropractic care enjoyed in this study in terms of total costs is exclusively because of the lack of hospitalizations among chiropractic-treated patients. For outpatient care, chiropractic was among the most expensive of providers.

The number of chiropractic visits per episode is substantially skewed, and some chiropractors may be inappropriately over treating some patients. If this over utilization were controlled, then chiropractic's cost advantage would increase (39).

CHIROPRACTIC TREATMENT: LITERATURE'S NEGATIVES

Chiropractic Versus McKenzie Treatments

Randomization to McKenzie therapy, chiropractic adjustment, or a control of an education pamphlet was given to 506 patients. McKenzie and chiropractic treatments both provided modest levels of pain relief when compared with the control group. The control group functioned just as well at the end of a month as did patients who had the more expensive McKenzie or chiropractic therapy. No differences were seen between any of the groups in terms of function or disability.

The McKenzie therapists saw their patients for an average of 4.6 visits over 1 month, whereas the chiropractors had, on average, two visits more per patient. In terms of total contact time, however, the McKenzie therapists spent more time with their patients than the chiropractors (40).

Manipulation Complications Identified

Various neurologic complications attributed to chiropractic manipulation in 89 cases reported in the English language literature are listed in Table 1.3. One case was of bilateral diaphragmatic palsy temporally related to chiropractic manipulation of the neck. Severe orthopnea of acute onset during cervical manipulation was the main symptom. We chiro-

Table 1.3

Complications of Manipulation

Complication	Reported (No.) Cases
Ischemia in vertebrobasilar territory	63
Vertebral artery dissections	9
Locked-in syndrome	4
Wallenberg's syndrome	7
Occipital infarct (hemianopsia)	2
Verterbral artery pseudoaneurysm	1
Other	43
Subdural hematoma with temporal bone fracture	1
Atlantoaxial dislocations	4
Myelopathy	9
Spinal cord infarction	1
Vertebral body fracture-dislocation	2
"Activation" of dormant foramen magnum meningioma	1
Brown-Séquard syndrome due to cervical epidural hematoma	1
Thoracic disc herniation	1
Other	3
Horner's syndrome	1
Lumbar radiculopathy	4
Cauda equina syndrome	6
Unilateral diaphragmatic paralysis	1

practors must be aware of the possible complications (Table 1.3) (41).

Cauda Equina Incidence with Spinal Adjustment Manipulations

Between 1967 and 1987 750,000,000 lumbar manipulations were performed with four cases of the cauda equina syndrome following chiropractic spinal manipulation reported, which yields a rough approximation of the risk as 1 case per 100,000,000 manipulations. It is conceivable that the true number of cases is under reported by a factor of 10 or even 100, making the risk of this complication 1 in 10,000,000 or 1 in 1,000,000 manipulations, respectively. Therefore, although the exact risk level risk is unknown, it is probably very low (42).

Treatment of Visceral Conditions with Spinal Manipulation

More than half of 1311 Australian chiropractors favored a role for spinal adjustment in the management of patients with visceral conditions such as migraine, asthma, hypertension, or dysmenorrhea. The perceived usefulness of spinal adjustment varied according to the condition being managed, as did the preferred level of adjustment.

Chiropractors continue to use spinal adjustment in the management of visceral conditions despite this intervention being regarded as an obstacle to the recommendation of public funding for chiropractic management of visceral conditions (43).

CHIROPRACTIC TREATMENT: LITERATURE'S (AND GOVERNMENTAL) POSITIVES

Positive Placebo Phenomenon with Chiropractic Care

The placebo response appears to be an integral component of practice within the holistic paradigm that profoundly affects clinical practice. The benefits derived from this element of the therapeutic encounter should not be denigrated; on the contrary, it is argued that practitioners should be trained to maximize positive placebo outcomes (44).

Manipulation Is Appropriate for Low Back Pain Patients

The RAND corporation studied and concluded that spinal manipulation is appropriate for low back pain without the indication of sciatica. The all-chiropractic panelists agreed unanimously: "An adequate trial of spinal manipulation is a course of 12 manipulations given over a period of up to 4 weeks, after which, in the absence of documented improvement, spinal manipulation is no longer indicated" (45).

Chiropractic seems to be an effective treatment of back pain; however, more studies with a better research methodology are clearly still needed (46). Referral for spinal manipulation therapy should not be made to practitioners applying rotatory cervical manipulation because of the risk of vertebrobasilar accidents (47).

Chiropractic Serves Needed Role

Cherkin and Deyo (48) state that nearly half the hospitalizations in the United States for patients with nonspecific back pain and herniated discs were for diagnostic tests (especially myelography) and the other half for pain control. Many hospitalizations for "medical back problems" are unnecessary, which also suggests a need for improved outpatient and home-based alternatives to hospitalization.

Chiropractic physicians are trained as outpatient clinicians, capable and accustomed to working within restricted parameters of diagnostic facilities while being forced to develop competent clinical impressions on which to build treatment protocol. The chiropractor has been highly trained in the clinical practice arena for detailed work-ups, devoid of the sophistication of radiology and laboratory facilities. The chiropractic doctor is highly skilled in using personal faculties of observation, palpation, plain x-ray, and clinical diagnosis to evaluate patients. Such training is what is being called for in medicine today—a time of cost conservation with a demand for contin-

ued quality care—for which we can thank its ancestors for their insight in preparing our profession for this time in health care delivery.

Distraction Is Therapeutic Choice for Discogenic Conditions

A detailed description of chiropractic care parameters used at a large occupational California medical center presented treatment algorithms that were derived from clinical needs of the facility, expert opinion, and reviews of several contemporary written protocols (49). Twelve of the most common industrially related low back conditions are included. The algorithms were grouped according to non-discogenic and discogenic conditions. The guidelines declared the appropriate care for **discogenic conditions** to be myofascial work, **distraction manipulation** to provide centripetal pressure within the disc, and home exercise to increase range of motion (ROM) and reduce spasm.

Chiropractic Specialization in Low Back Pain Is Becoming a Reality

When discussing training of chiropractic doctors in the specialized field of low back pain, I quickly think of Crockard (50), who wrote that spinal surgery is a high-risk specialty that is still being tried by surgeons who perform it less than 10 times a year. He states that both orthopaedic and neurosurgeons want spinal surgery as part of their respective fields, but want it as a part of their general practice. To paraphrase Saint Augustine on chastity: these groups want spinal surgery, but not pure spinal surgery yet.

Crockard (50) calls for the next generation of neurosurgeons and orthopaedic surgeons to generate spinal surgery as a specialty and to classify the surgeon who operates on only the spine as a specialist such as is the hand surgeon or maxillofacial surgeon. No surgeon can be expected to clip a cerebral aneurysm, remove a meniscus through an arthroscope, and perform pedicle screw fixation of the lumbar spine, all with equal facility. I ask the same of the chiropractic doctor: Can he or she be expected to be equally skilled at treating all extremities and all parts of the manipulative spine? I say not—it demands too much ability for one person. Thus, the creation of the specialist in the most common area seen by the chiropractor—the low back. The certification course fostered and nurtured between myself and the National College of Chiropractic since 1991 stands as the model of specialization in distraction manipulation procedures of the low back.

Chiropractic Care Is of More Benefit Than Hospital-Based Therapy

The Manga Three Year Follow-up report compares the effectiveness, over 3 years, of chiropractic and hospital management for low back pain. Patients with low back pain (741 men and women aged 18 to 64 years) were randomly allocated to chiropractic or hospital outpatient management over a 3-year period. Results indicated that when chiropractic or hospital therapists treat patients for low back pain as they would in day-to-day practice, those treated by chiropractic derived more benefit and long-term satisfaction than those treated by hospitals (51).

Chiropractors Fill Need for Primary Care Practitioners

A need exists for chiropractors to be primary care physicians because of the current shortfall of approximately 100,000 generalist physicians to meet the 50:50 specialist-to-generalist ratio needed (52).

Chiropractic Radiologists Outperform Medical Radiologists on Testing

Four hundred ninety-six medical and chiropractic radiologists, residents, students, and clinicians completed a test of radiographic interpretation consisting of 19 cases with clinically important radiographic findings. Chiropractic radiologists', chiropractic radiology residents', and chiropractic students' test results were significantly higher than those of their medical counterparts (53).

Spinal Manipulation Consistently Outperforms Other Treatments of Low Back Pain

Twenty-three randomized controlled clinical trials on the effectiveness of spinal manipulation compared with other methods of care for low back pain, including sham, proved it to be consistently more effective in the treatment of low back pain than were any of the array of comparison treatments (48).

Spinal Manipulation Is Safer Than Other Therapies

A patient is 1 to 75 times more likely to die from nonsteroidal anti-inflammatory drug (NSAID) use than to sustain a vertebrobasilar insult from cervical manipulation; 30 to 1000 times more likely to die from an intravenous pyelogram than to sustain a vertebrobasilar insult from cervical manipulation; and 500 to 15,000 times more likely to die from lumbar disc surgery than to sustain a vertebrobasilar insult from cervical manipulation (54).

Steve Martin, PhD, Thesis on Chiropractic: The Only Truly Scientific Health Care System

The following thoughts from Dr. Martin's thesis are presented for their interest to the chiropractor (55).

Although physicians assumed that they were the sole legitimate arbiters of what constituted the science of healing, chiropractors were able to assert that they too were scientific, and they found sufficient common ground with medicine and popular understanding about science to make this argument tenable. Medicine failed to achieve a monopoly over science with a capital "S."

Chiropractors could, and did, derive many of the benefits of proclaiming themselves scientific that physicians did. Certainly, the assertion that science led to improved clinical outcomes was promoted as aggressively within chiropractic rhetoric as it was within medical discourse. Just as physicians attributed their "miracle" cures—the infant brought back from death's door by antitoxin, the child saved by insulin—to medical science, chiropractors paraded out a host of testimonials from patients cured by chiropractic science. By providing a rationale for chiropractic intervention and supplying clinical evidence of its efficacy, chiropractic science enhanced the economic competitiveness of chiropractors.

However, chiropractic science provided far more than a market advantage. Science was a fundamentally important constituent of chiropractors' self-identity. They were unwilling to be relegated to the status of craftsmen who offered an empirically useful treatment. By elaborating a unique conception of science, chiropractors developed an intellectual framework and justification for spinal manipulation that expanded chiropractic beyond an empiric craft and enhanced its professional credibility and stature. Although it is unlikely that most practicing chiropractors—or practicing physicians, for that matter—consciously dwelled on esoteric points of scientific epistemology, their science provided an essential part of their identity. Chiropractors were not simply spine-twisters, nor physicians pill-peddlers, because their actions rested on a scientific foundation.

Not only was chiropractic scientific, but chiropractors believed that their science was superior to medicine, both clinically and morally. Rejecting reductionism and materialism, chiropractors believed that their vision of science retained a necessary emphasis on vitalism and spirituality. Chiropractic science accepted the individuality of each patient in the context of a universe governed by God's natural laws. It has been argued that, for physicians, laboratory science promoted a new professional ethos, one in which "accountability to science replaced relations with patients." If the new scientific medicine placed a subtle but distinct wedge of science between doctor and patient, chiropractic science firmly anchored the practitioner to the bedside. The only science chiropractors performed was clinical-—observing patients. This characteristic allowed chiropractors to argue that their's was "the only truly scientific method of healing." True science incorporated a patient-centered system of values that embraced the integration of mind, body, and soul. Confidence in the moral and therapeutic superiority of their science provided the core of chiropractic's professional identity.

The many uses of science by chiropractors challenges historical scholarship that implicitly assumes that after 1900 only orthodox medicine and its allies successfully appropriated "science." The diversity of meaning and values attributed to science allowed chiropractors to gain many of the advantages that physicians acquired by stressing chiropractic's "scientific" status. The success of chiropractic highlights the vitality, persistence, and importance of alternative scientific systems within American society. We have only begun to tease out the implications of the enormous variety of meanings associated with 20th-century science, especially in the relationship between science and healing. Studying alternative healers provides a useful tool for examining these complex relationships. (Reprinted with permission from Martin SC. The ony truly scientific method of healing: chiropractic and American science, 1895–1990. ISIS 1994; 85:207–227, by the University of Chicago.)

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES RECOMMENDS SPINAL MANIPULATION FOR ACUTE LOW BACK PAIN

The Agency for Health Care Policy and Research of the U.S. Department of Health and Human Services division of the Public Health Service published treatment guidelines entitled "Acute Low Back Problems in Adults: Assessment and Treatment in 1994." This document stated that spinal manipulation using short or long leverage methods is safe and effective for patients in the first month of acute low back symptoms without radiculopathy. For patients with symptoms lasting longer than 1 month, manipulation is probably safe, but its efficacy has not been proved. If manipulation has not resulted in symptomatic and functional improvement after 4 weeks, it should be stopped and the patient re-evaluated.

This document also states that physical modalities such as massage, diathermy, ultrasound, cutaneous laser treatment, biofeedback, and TENS also have no proven efficacy in the treatment of acute low back symptoms.

Invasive techniques such as needle acupuncture and injection procedures (injection of trigger points in the back; injection of facet joints; injection of steroids, lidocaine, or opioids in the epidural space) have no proven benefit in the treatment of acute low back symptoms.

Acetaminophen was cited as the safest effective medication for acute low back pain. NSAIDs, including aspirin and ibuprofen, are also effective although they can cause gastrointestinal irritation/ulceration or, less commonly, renal or allergic problems. Muscle relaxants were found no more effective than NSAIDs, and opioids appear no more effective than safer analgesics for managing low back symptoms.

Shoe lifts for leg length inequalities less than 2 cm were found to be ineffective in treating low back pain. Low back corsets and back belts do not appear beneficial for treating acute low back symptoms. Shoe insoles were found safe and inexpensive options for patients who must stand for prolonged periods, if they request them (56).

U.S. Public Health Service's Health Resources and Services Administration Awards Grants for Research on the "Biomechanics of Flexion Distraction Therapy"

In 1994, federal grants totaling $313,167 were awarded to the National College of Chiropractic and Loyola Univer-

sity Stritch School of Medicine for a joint study of Cox Distraction manipulation. The goal is to describe with quantitative data the biomechanical events that occur in the spine during distraction manipulation, namely changes with the intervertebral disc space, osseoligamentous canal, and facet joints. Information on defining the limits of safety for distraction manipulation to the ligamentous and cartilaginous structures of the lumbar spine will be obtained. This will assist clinicians in the appropriateness of flexion distraction for particular patients and to assist investigators in designing clinical trials.

The principal investigator is M. Ram Gudavalli, PhD, of the National College of Chiropractic Research Department. James M. Cox, DC, DACBR will be the clinician in the study. From Loyola Medical School will be A.G. Patwardhan, PhD, director of the Orthopedic Biomechanics Laboratory at Loyola University and Research Department of Hines Veterans Affairs Hospital, and Alexander Ghanayem, MD, Chief of Spine Surgery, Department of Orthopedic Surgery at Loyola University.

This award culminates 35 years of study, research, and many failed attempts to gain research funding from the federal and private sources. It proves that persistence for a worthy goal pays off. Dr. Gudavalli has authored a chapter in this textbook on this study.

A second grant was awarded in 1997 by the Health Resources and Services Administration of the U.S. Public Health Service entitled "Flexion Distraction Vs. Medical Care for Low Back Pain." This grant, which will last into the year 2000, will compare chiropractic flexion distraction adjusting at the National College of Chiropractic to medical care administered at Loyola Medical School.

DISTRACTION ADJUSTING IS A SPECIALIST PROCEDURE—REQUIRING KNOWLEDGE AND SKILL LEVELS FOR OPTIMAL OUTCOMES

Casey Lee, MD, President of the North American Spine Society, stated the following in his presidential address in 1994:

> *The rate of laminectomy for disc herniation in the United States is three times higher than in Canada and nine times higher than in Europe.*

The rate of hospital admissions for medical and surgical procedures is eight times different between two hospitals, one in Boston, Massachusetts, and the other in New Haven, Connecticut. The rate of spinal fusion in the western region of the United States is nine times higher than in the Northeast (57).

The American College of Cardiology (ACC) and American Heart Association (AHA) Task Force reported that hospitals having inadequate caseloads have suboptimal outcome results. A minimal threshold volume was recommended to be 100 bypasses per year per cardiac surgeon to maintain competency.

In 1991, intersurgeon variability was reported to have ranged from 40 to 76% for cure after resection of colorectal cancer, and the intersurgeon variability ranged from 8 to 30% for postoperative mortality. Difference in training and competency was suggested as the probable reason for such a wide intersurgeon variability.

Results of a prospective study with 16 centers and 40 surgeons on factors affecting the outcome of obtaining solid spinal fusion indicated that the "surgeon factor" was the most important factor even after adjusting for other positive factors affecting the outcome. The range of successful fusion rate was 50 to 100% among surgeons.

What are reasons for "surgeon specific" variability? An important factor is variability in skill level. Skill is attained by acquiring basic knowledge, by exposure and training (learning curve), and by maintenance and additional improvement (volume). The clinical outcome is significantly affected by the surgeon's skill level and his or her position on the learning curve.

What does it take to reach the plateau of the learning curve? What type of supervision or training? How many cases? If so, how long? When one has reached the plateau, what volume is needed to maintain competency? Should we allow all clinicians to perform all types of surgery? Is it best to credential practitioners for certain types of procedures? Do we need a certificate of added qualification? (57).

Few Diagnostic and Therapeutic Treatments Are Proved

The Quebec Task Force on Spinal Disorders reported that there was *only 1 of 256 diagnostic test-disease conditions to have scientifically proved value* as shown by a randomized controlled study. *Among 1314 possible therapeutic modalities-disease conditions, only 26 treatment modalities for the lumbar spine and only 1 for the cervical spine had scientific value.*

The new era has begun! Every individual practitioner, group, institution, and level of government is expected to be accountable and responsible. If we do not prepare ourselves in a proactive way, surely we will be nothing but a sitting duck.

What can we do? Some of the proposed remedies for these problems are randomized clinical trials, practice algorithms, practice guidelines, consensus statements, and scorecard systems.

Is it a physician's responsibility to disclose a personal scorecard to the public? Is it the public's right to have individual practitioners' scorecards available? (57).

One Chiropractic Technique's Accountability

Cox Distraction Adjusting has a certification course through the National College of Chiropractic to train and credential Cox practitioners in this specific adjustment technique. It seems that this program is right in line with other specialty fields in medicine.

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Biomechanics of the Lumbar Spine

James M. Cox, DC, DACBR

chapter 2

Anyone who stops learning is old, whether at twenty or eighty. Anyone who keeps learning stays young. The greatest thing in life is to keep your mind young.

—Henry Ford

NEUROANATOMY OF THE CAUDA EQUINA IN THE LOWER LUMBAR SPINE

Wall et al. (1) dissected the cauda equina in cross section for excellent study and visualization of the thecal sac containing the nerve roots and the location and size differential of the sensory and motor components (Figs. 2.1–2.7).

Nerve Root Compression in Foraminal Narrowing and Subluxation

Compression of a spinal nerve root within an intervertebral foramen has been demonstrated in patients who have sciatica, but lateral recess stenosis, or nerve root compression within the spinal canal, is a more common clinical finding than foraminal stenosis (2). Regardless, such compression does not always cause sciatica; therefore, clinical correlation is necessary with such stenotic findings.

Figure 2.8 is a cross-sectional view through a normal foramen showing the digitized areas that were studied. Significant positive correlations are demonstrated between nerve root compression and the posterior disc height, the foraminal height, and the foraminal cross-sectional area for the four intervertebral levels between the second lumbar and the first sacral vertebrae. Nerve root compression was identified by inspection when findings included (*a*) contact between the nerve root and the adjacent tissue, (*b*) deformation of the root apparently caused by pressure of the adjacent tissue, and, in addition, no perineural fat seen in the contact areas of the nerve root within the foramen.

Figure 2.9 shows a nerve root compressed by the ligamentum flavum (*arrow*) and subluxation of the articular processes. No perineural fat is seen in the region of contact between the root and the adjacent tissue.

NEUROANATOMY AND ITS ROLE IN DIAGNOSING DISC HERNIATION

Let us discuss the anatomy of the lumbosacral plexus and other plexi of the lumbar spine and pelvis. Dietemann et al. (3) state that the main nerves of the pelvis and lower limbs arise from the lumbar and sacral plexi. An understanding of the neurologic findings related to paravertebral and pelvic pathology requires complete and accurate knowledge of the anatomy of these regions. The lumbar plexus is formed by anastomosis of the ventral rami of the four first lumbar nerves. The lumbar plexus lies within the posterior portion of the psoas muscle.

Iliohypogastric and Ilioinguinal Nerves

The iliohypogastric and ilioinguinal nerves arise from the first lumbar nerve. The iliohypogastric nerve is distributed to the skin of the upper lateral part of the buttock (lateral branch) and the skin of the pubis, and it also has muscular branches to the abdominal wall. The ilioinguinal nerve extends to the upper and medial regions of the thigh, and, in males, to the skin of the penis and scrotum; in females, it extends to the skin of the pubis and the labium majus.

Genitofemoral Nerve

The genitofemoral nerve arises from the first and second lumbar nerves. It has a genital branch, which supplies the creamaster muscle, the skin of the scrotum in males, or the skin of the mons pubis and labium majus in females; it has a femoral branch, which supplies the skin of the upper part of the femoral triangle.

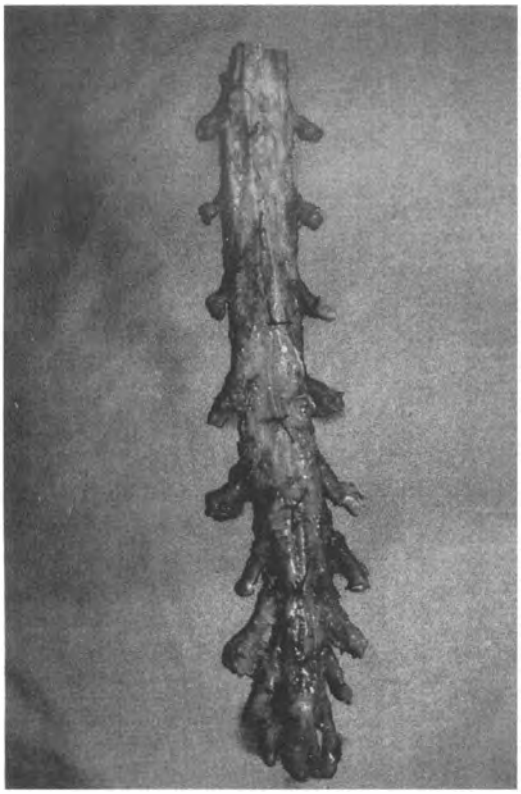

Figure 2.1. Posterior view of cauda equina and surrounding dural sac prior to sectioning. Sutures mark the individual disc levels. (Reprinted with permission from Wall EJ, Cohen MS, Masie JB, et al. Cauda equina anatomy I: intrathecal nerve root organization. Spine 1990;15(12): 1244–1247.)

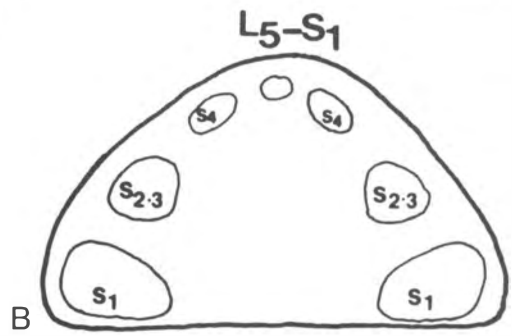

Figure 2.2. **A.** Cross-sectional view through L5–S1 disc level revealing S1 root anterolateral and crescent-shaped pattern of lower sacral roots (*top* = dorsal). **B.** Schematic diagram depicting pattern of sacral root orientation at the L5–S1 intervertebral level. (Reprinted with permission from Wall EJ, Cohen MS, Masie JB, et al. Cauda equina anatomy I: intrathecal nerve root organization. Spine 1990;15(12):1244–1247.)

Figure 2.3. Dura retracted exposing exit of first through third sacral roots from dural envelope. S4 and S5 roots have been reflected. (Reprinted with permission from Wall EJ, Cohen MS, Masie JB, Rydevik B, et al. Cauda equina anatomy I: intrathecal nerve root organization. Spine 1990;15(12):1244–1247.)

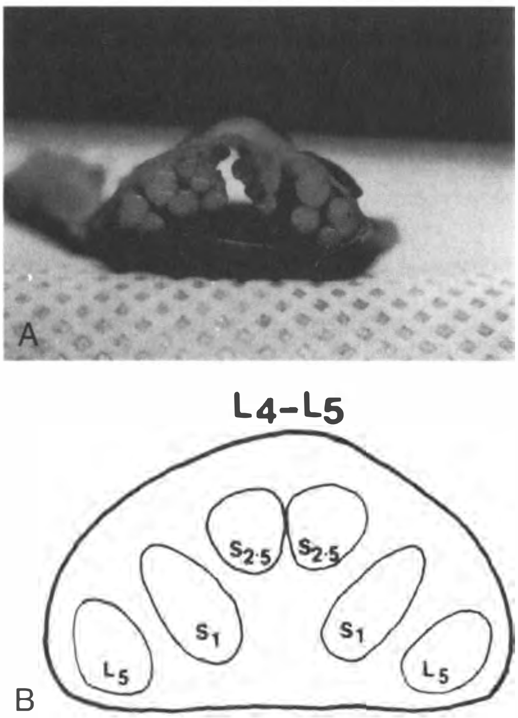

Figure 2.4. **A.** Cross-sectional view at L4–L5 disc level revealing L5 root in anterolateral position. S1 root is displaced medially forming diagonal layer (V configuration). S2–S5 roots remain dorsal midline (*top* = dorsal). **B.** Schematic representation of individual roots at L4–L5 cross-sectional disc level. (Reprinted with permission from Wall EJ, Cohen MS, Masie JB, et al. Cauda equina anatomy I: intrathecal nerve root organization. Spine 1990;15(12):1244–1247.)

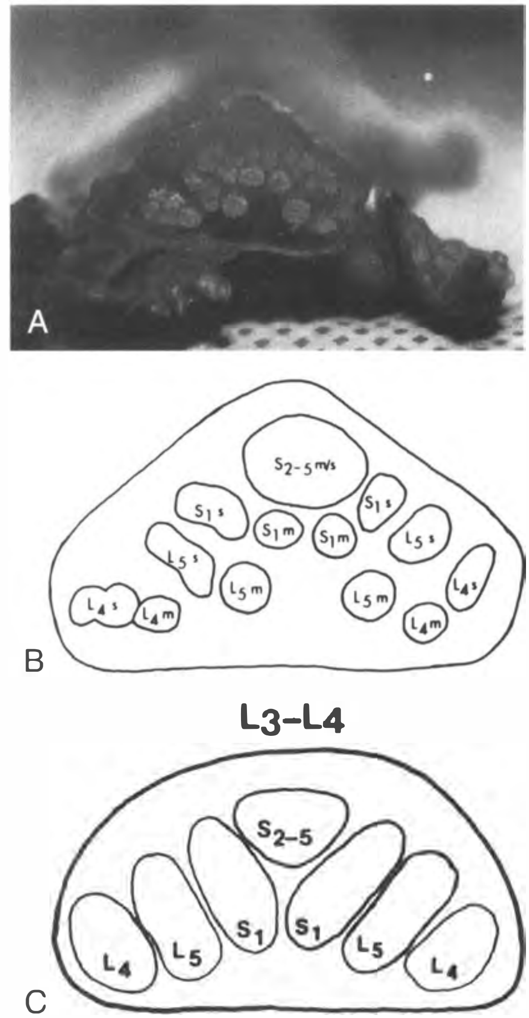

Figure 2.5. **A** and **B.** Cross-sectional view through L3–L4 disc level. Oblique layered configuration of roots evident bilaterally. Single motor bundle seen medial and ventral to multifascicular sensory bundle within each layer. S2–S5 roots remain dorsal (*top* = dorsal). **C.** Schematic representation of cross-sectional root organization at L3–L4 disc level. (Reprinted with permission from Wall EJ, Cohen MS, Masie JB, et al. Cauda equina anatomy I: intrathecal nerve root organization. Spine 1990;15(12):1244–1247.)

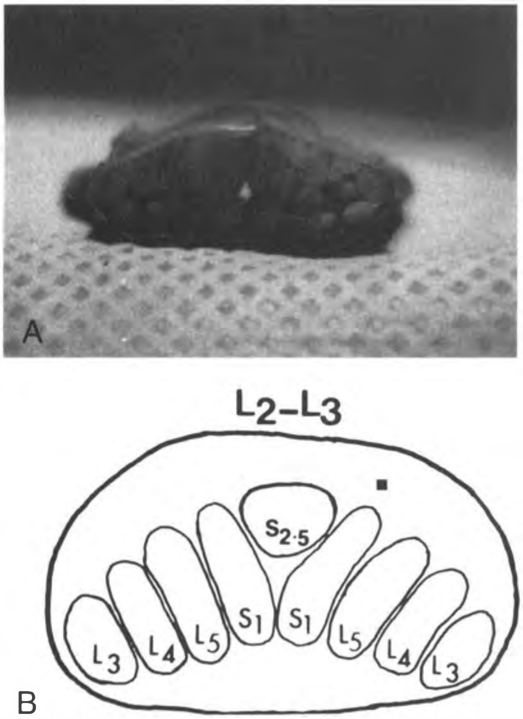

Figure 2.6. **A.** Cross-sectional view through L2–L3 disc level. L3-S1 roots continue oblique layered pattern with lower sacral (S2–S5) roots remaining dorsal (*top* = dorsal). **B.** Schematic depicting cross-sectional layered pattern of roots at the L2–L3 intervertebral level. (Reprinted with permission from Wall EJ, Cohen MS, Masie JB, et al. Cauda equina anatomy I: intrathecal nerve root organization. Spine 1990;15(12): 1244–1247.)

Figure 2.7. Cadaveric section of cauda equina between the L3–L4 and L4–L5 intervertebral levels. The dura is retracted, revealing the elegant laying of roots and the invaginations of arachnoid, which hold the roots in relation to one another. (Reprinted with permission from Wall EJ, Cohen MS, Masie JB, et al. Cauda equina anatomy I: intrathecal nerve root organization. Spine 1990;15(12):1244–1247.)

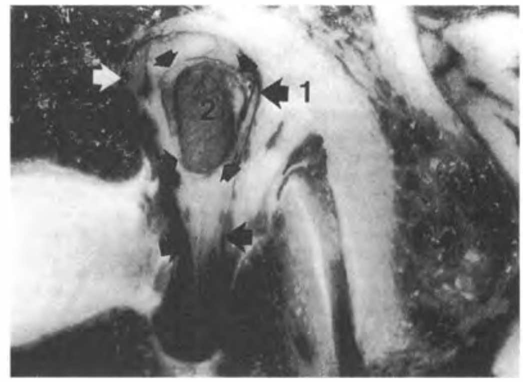

Figure 2.8. Section through a foramen, showing the digitized cross-sectional areas that were determined. *1*, foraminal cross-sectional area (*large black arrows*) and *2*, nerve root cross-sectional area (*small black arrows*). The *white arrow* shows the osseous margin. (Reprinted with permission from Hasegawa T, An HS, Haughton VM, et al. Lumbar foraminal stenosis: critical heights of the intervertebral discs and foramina. J Bone Joint Surg 1995;77A[1]:32–38.)

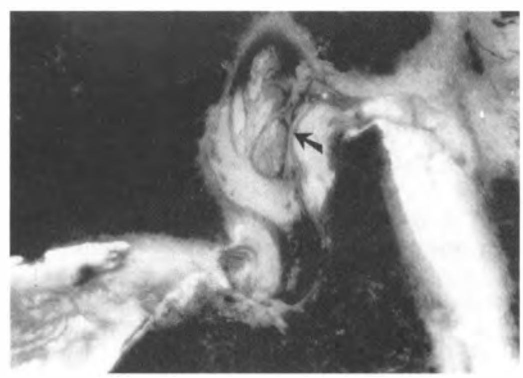

Figure 2.9. Section showing a nerve root compressed by the ligamentum flavum (*arrow*) and subluxation of the articular processes (*right*). No perineural fat is seen in the region of contact between the r adjacent tissue. (Reprinted with permission from Hasegawa T, An HS, Haughton VM, et al. Lumbar foraminal stenosis: critical heights of the intervertebral discs and foramina. J Bone Joint Surg 1995;77A(1):32–38.)

Lateral Cutaneous Nerve

The lateral cutaneous nerve of the thigh arises from the second and third lumbar nerves, supplying the skin on the lateral aspect of the thigh and the lateral aspect of the buttock.

Femoral Nerve

The femoral nerve is the largest terminal branch; it arises from the dorsal branches of the ventral rami of the second, third, and fourth lumbar nerves. The femoral nerve supplies the skin of the anterior aspect of the thigh and of the medial border of the leg, the quadriceps of the thigh and sartorius, and the iliac muscles.

Obturator Nerve

The obturator nerve arises from the ventral branches of the ventral rami of the second, third, and fourth lumbar nerves.

Sciatic Nerve

The sciatic nerve is the continuation of the sacral plexus. It is the largest nerve in the body, measuring 2 cm across at its origin. It leaves the pelvic cavity through the greater sciatic foramen, below the piriformis muscle, and passes behind the sacrospinal ligament at its insertion on the ischial spine, then running downward between the greater trochanter of the femur outside and the tuberosity of the ischium inside; at this level, the nerve is located in front of the greatest gluteal muscle and behind the obturator internal and gemellus muscles, and the quadratus femoris muscle.

The sciatic nerve supplies the skin of the posterior and lateral border of the leg and foot as well as the muscles of the leg and foot and the posterior muscles of the thigh.

Pudendal Nerve

The pudendal nerve derives from the second, third, and fourth sacral nerves; it is the most important branch of the plexus. It supplies the skin of the perineum, scrotum, and penis (or labium majus and clitoris); branches are also distributed to the external anal sphincter, the skin around the anus, the muscles of the perineum, and the pelvic viscera (3).

Scrotal Pain in Disc Compression of S2 and S3 Nerve Roots

Scrotal pain is described anatomically by White and Leslie (4), who present a 20-year-old man who had consulted his general practitioner 15 months earlier because of continuous right scrotal pain. A consultant urologist excluded testicular disease and referred him to a pain clinic, but an ilioinguinal and genitofemoral nerve block did not relieve the pain. The pain could be reproduced by straight leg raising, so an orthopaedic opinion was sought.

The pain was exacerbated by leaning forward, coughing, or moving suddenly. The patient's lumbar lordosis was slightly flattened, and no forward flexion of the lumbar spine was seen. Straight leg raising was limited to 20° on the right by severe scrotal pain. No objective neurologic signs were present.

At operation, an intervertebral disc protrusion was found to be impinging on the first sacral nerve root on the right. The disc was incised, and a good decompression was achieved. Pain relief was immediate and permanent.

The posterior two thirds of the scrotum is innervated by the second and third sacral nerves. Central disc lesions may compress the lower sacral roots, but no such compression was demonstrated in this case. An upper lumbar disc lesion is a rare cause of scrotal pain, and in such cases there may be no restriction of straight leg raising. The distribution of pain did not seem to be related to the level of the disc protrusion, yet decompression of the first sacral nerve root relieved the symptoms. Perhaps the anomaly of bone segmentation, besides predisposing to disc degeneration, was associated with an anomaly of nerve root segmentation. This case emphasizes the value of examination of the lumbar spine in cases of unexplained scrotal pain (4).

Summary of Low Back and Leg Pain Production

Nachemson (5), in a discussion of the role of the disc in low back and leg pain, concludes:

1. Disc hernia is usually preceded by one or more attacks of low back pain.
2. Following intradiscal injection of either hypertonic saline or contrast media, it is often possible, in patients with complaints of pain as well as in normal subjects, to artificially cause the same type of pain as that which occurs from disc degeneration.
3. Investigations have been performed in which thin nylon threads were surgically fastened to various structures in and around the nerve root. Three to four weeks after surgery, these structures were irritated by pulling on the threads, but pain resembling that which the patient had experienced previously could be registered only from the outer part of the anulus and the nerve root.
4. Pathoanatomically radiating ruptures are known to occur in the posterior part of the anulus, reaching out toward the areas in which naked nerve endings are located. Such single ruptures in the lumbar discs are first manifested in people about 25 years of age, the same age at which the low back pain syndrome becomes clinically important. Various theories on how these ruptures elicit pain exist.
5. Of all the structures that theoretically could be involved in the pain process, only the disc shows changes that could account for the anatomic changes at such an early age. Such changes in other structures in the region generally show up much later in life, and then only secondary to severe disc degeneration.

6. Although a late sign, disc degeneration is noted on radiographs of patients between 50 and 60 years of age, and it has been seen significantly more often in those who have had back pain than in those who have not.

The facet joints have been demonstrated to show histologic signs of arthritis very late in life and always secondary to degenerative changes in the discs.

Factors in Low Back Pain Onset

Genetics

Genetic factors play a much stronger role in disc degeneration than previously suspected. A study of 115 pairs of male identical twins showed that genetic inheritance accounted for as much as 50 to 60% of the disc changes (6). Disc degeneration in the lower lumbar spine had no significant association with occupational loading, history of back injuries, exposure to vibration, or smoking. Magnetic resonance images (MRI) of the lumbar spines of 40 male identical twins to assess degenerative disc changes showed similarities between the co-twins were significantly greater than would be expected by chance (7).

In a study group of 65 patients who had undergone surgery for degenerative disc disease, 44.6% were noted to have a positive family history, whereas 25.4% of the patients in the control group had a positive family history. In the study group, 18.5% of relatives had a history of having spinal surgery, compared with only 4.5% of the control group. A familial predisposition to degenerative disc disease can exist along with other risk factors (8).

Growth Period of Back Pain with a Familial Cohort Predicts Adult Low Back Pain

An 88% probability of low back pain later in life is seen if low back pain is present in the growth period and a familial occurrence of back pain exists. Growth period pain shows a trend toward aggravation as time passes. Thus, implementing preventive measures in schools may be important in reducing back pain later in life (9).

Low Back Pain Factors

Back injuries in the work place are rarely caused by direct trauma; typically, they are the result of overexertion. Of individual factors (10), age is the most important, whereas sex and smoking are probable risk factors. Occupational factors associated with an increased risk of low back pain are:

- Heavy physical work
- Static work postures
- Frequent bending and twisting
- Lifting, pushing, and pulling
- Repetitive work
- Vibrations
- Psychological and psychosocial

Individual factors often discussed as potential risk factors in low back pain are (10):

Factor	Importance
Age	Certain
Sex	Probable (age-dependent)
Posture	Low (severe only)
Anthropometry	Low (extremes only)
Muscle strength	Low (work-related)
Physical fitness	Low (work-related)
Spine mobility	Low
Smoking	Probable

Vibration with Heavy Lifting Is High Risk of Low Back Pain

Combined long-term vibration exposure followed by heavy lifting, driving as an occupation, and frequent lifting are the greatest risk factors for low back injury. Repetitive compressive loads put the spine in a poorer condition to sustain higher loads applied directly after a long-term vibration exposure, such as from several hours of driving. Another consideration is the vibration-induced accumulation of metabolites, which leads to a more accelerated development of degenerative changes in the disc. Drivers aged 35 to 45 years reported more "low back pain" than control subjects, whereas no difference was found between occupations in the younger and older groups (11).

Childbearing Increases Low Back Pain Incidence

Fifty percent of women have back pain some time during pregnancy and more than a third report it as a severe problem. Back pain occurs at night in more than one third of pregnant women, and it contributes significantly to insomnia. Pregnancy-related back pain is associated with a higher number of subsequent abortions, either spontaneous or induced. Weight gain, maternal obesity, and fetal weight at term were not found to be related to gestational back pain. Back pain occurring during pregnancy is also associated with a postpartum back pain prevalence of about 40% (12).

Postpartum backache probably results from both epidural anesthesia and posture, and because of the combination of stressed positions in labor, muscular relaxation, and lack of mobility. Clinical entities implicated as causes of back pain in pregnancy include pelvic insufficiency, sacroiliac joint subluxation, sciatica, lumbosacral disc pathology, spondylolisthesis, postural back pain and lumbar lordosis, thoracic back pain, and coccydynia. Sacroiliac joint subluxation incidence in pregnancy is about 28%, and therapeutic rotational manipulation of the sacroiliac joint reportedly results in relief of pain in 91% of cases (12).

Increased lifting and stress may be responsible for an increased risk of low back pain in both men and women with children (13).

Pelvic pain is associated with twin pregnancy, first pregnancy, older age at first pregnancy, larger weight of the baby, forceps or vacuum extraction, fundus expression, and a flexed position of the woman during childbirth. The pain is hypothesized to be caused by strain of the ligaments in the pelvis and lower spine, which result from a combination of damage to lig-

aments, hormonal effects, muscle weakness, and the weight of the fetus (14).

Thirty percent of women are on sick leave for an average of 7 weeks during pregnancy. Pain intensity is reduced and the expenses of extra physiotherapy was regained by a factor of 10 through reduced costs from sick leave (15).

Other Factors Associated with Higher Risk of Low Back Pain

Previous traumatic back injury increased the risk of having a low back syndrome 2.5 fold, and was responsible for 16.5% of sciatica and 13.7% of low back pain cases (16). Previous low back pain, or current pain in other sites doubles the risk of developing a new episode of low back pain (17).

Smoking was associated with increased risk of low back pain in all subgroups except women aged 30 to 49 years, but it was not associated with sciatica. The risk of sciatica increased significantly with increased body height in men aged 50 to 64 years (16).

No clear evidence points to a causal relationship between smoking and back pain. It is unlikely that smoking causes sciatica or disc herniation (18). Another study indicates smoking is likely to cause at least certain types of low back pain, such as longstanding low back pain or frequently reoccurring low back pain combined with problems in other musculoskeletal areas. Smokers with a low body mass index may be more likely to experience low back pain and/or other musculoskeletal problems than those of heavier build. A link between smoking, respiratory problems, and some types of low back pain is suggested, but respiratory problems alone are not obviously associated with low back pain (19).

Patients with chronic back pain consume more than twice as much caffeine as patients without chronic back pain (20).

EFFECTS OF DISC CIRCULATION AND LOW BACK PAIN INCIDENCE

Smoking

To open this discussion, I would like to cite an interesting study (21) on lung cancer incidence in smoking. Although this study does not deal with low back pain, it is an important issue and is comparable to the adverse effects on disc circulation that follow. In the study, the available epidemiologic studies of lung cancer and exposure to other people's tobacco smoke (exposure was assessed by whether or not a person classified as a nonsmoker lived with a smoker) were identified and the results combined. In 10 case-controlled and 3 prospective studies, overall, a highly significant 35% increase in the risk of lung cancer was found among nonsmokers living with smokers compared with nonsmokers living with nonsmokers (relative risk, 1.35; 95% confidence interval, 1.19 to 1.54). The increase in risk among nonsmokers living with smokers compared with a completely unexposed group was thus estimated as 53% (relative risk of 1.53).

This analysis and the fact that nonsmokers breathe environmental tobacco smoke, which contains carcinogens, into their lungs, and that the generally accepted view is that no safe threshold exists for the effect of carcinogens, lead to the conclusion that breathing other people's tobacco smoke is a cause of lung cancer. About one third of the cases of lung cancer in nonsmokers who live with smokers, and about one fourth of the cases in nonsmokers in general, can be attributed to such exposure (21).

It is often thought by physicians that veterans have a much higher prevalence of smoking than the general population. To test this perception, all patient charts on the medical and surgical wards of the Denver Veterans Administration Hospital were reviewed on August 24, 1986, for reported smoking habits.

Nearly twice as many inpatients (50.7%; 74 of 146) as outpatients (27.0%; 126 of 466) were current smokers ($P < 0.001$, X^2). The age-adjusted smoking rate among inpatients (63.5%) was almost double the national rate (33%), whereas no significant difference was found between the outpatient rate (35.6%) ($P > 0.10$, Poisson) and the national rate. Indeed, a high prevalence of smoking and smoking-related diseases is found among VA hospital inpatients. In contrast, outpatient veterans smoke at a rate similar to the national average (22).

Smoking Reduces Discal Circulation

Particularly in the case of large human discs in which the balance between nutrient use and supply is delicate, any loss in blood utilization and supply is precarious, and any loss in blood vessel contact or reduction in blood flow at the periphery of the disc could lead to nutritional deficiencies and buildup of waste products (23).

In an experimental study, the influence of cigarette smoke on nutrition of the intervertebral disc was investigated. Six dogs and eight pigs were anesthetized, intubated, and kept ventilated in a respirator. An additional pumping system was attached to the respirator so that the smoke could be administered. During the testing time, blood gases and intradiscal oxygen tension were measured continuously. After the smoking period, radioactive isotopes (sulfate and methyl glucose) were introduced intravenously. The animals were killed at various times after the infusion, and their spines were quickly excised and analyzed.

A smoking period of 3 hours reduced the transport efficiency of blood gases and oxygen to about 50%. The effect of smoke decreased when the exposure ceased. The concentration gradients were close to normal after 2 hours of "recovery."

These findings demonstrate that cigarette smoke significantly affects the circulatory system outside the intervertebral disc. The most pronounced effect was the reduction in solute exchange capacity. When the transport of substrate, which is necessary for the cells in order to fulfill the prevailing energy demands in the tissue, is reduced, the inevitable consequence over a longer period of time will be deficient nutrition (23).

Smoking and Exercise Incidence in Low Back Pain

We compared the exercise and smoking habits of 576 patients suffering low back and leg pain with those of 50 persons who stated that they were asymptomatic. Findings were that 33% of

low back and leg pain sufferers smoked and 14% of patients without low back or leg pain were found to smoke; and 47% of low back or leg pain sufferers exercised regularly, as compared with 86% of nonsufferers. Specifics on the amount of smoking (by packs of cigarettes, amount of pipe tobacco, or number of cigars smoked daily) were given in this paper, as well as the number of times weekly a person exercised and for how long. A higher percentage of persons not suffering from low back or leg pain exercise regularly, more frequently, and longer at each session than those who suffer from these pains. Likewise, a higher percentage of those without low back and leg pain did not smoke, as compared with those who did have low back or leg pain. These statistics would indicate that less low back and leg pain is experienced by those who exercise regularly and avoid smoking (24).

Further factors concerning low back pain and smoking are of interest. Cigarette smoking was associated with an increased risk of prolapsed disc (25). A person's risk of prolapsed disc was increased by about 20% for each 10 cigarettes smoked per day, on the average, during the past year. Patients with severe low back pain were more likely to be cigarette smokers and consumed greater amounts of tobacco, as measured by both the number of cigarettes smoked per day and the number of years of exposure (26). Fifty-three percent of 288 men with severe low back pain smoked, whereas only 39.6% of 368 men without pain smoked, and 43.8% of 565 men with moderate low back pain smoked.

In a retrospective study, smoking was identified as being significantly associated with medically reported episodes of low back pain. Svensson (27) and Svensson and Andersson (28) identified a similar association in Swedish industrial workers, and they speculated that coughing leading to increased intradiscal pressure was the mechanism responsible for this relationship. A Danish study (29) supported this idea by identifying coughing and chronic bronchitis, but not smoking, as important in the cause of low back complaints.

Frymoyer et al. (30) indicated that smoking and coughing were related to low back pain but that coughing alone was insufficient to account for the difference in back complaints in subjects who smoked. It might be that smokers have emotional, recreational, or occupational differences, although multivariate analysis of a retrospective and epidemiologic survey did not confirm that speculation. The nicotine equivalent of one cigarette, when injected into a dog, may cause a reduction in the blood flow in the vertebral body. It is believed that decreased diffusion of nutrients into the disc by such alteration of blood flow could adversely affect discal metabolism and render the disc more susceptible to mechanical deformities (31).

Other studies have suggested that smoking and/or coughing is a risk factor for prolapsed lumbar disc (29, 32) and for back pain in general. In fact, it now seems that spinal disorders can be added to the long list of diseases associated with cigarette smoking. The mechanisms for the association with smoking are not entirely clear. One plausible mechanism is that smoking brings about coughing, which in turn puts more pressure on discs. In one study (26), the association of coughing with prolapsed lumbar disc was negligible. Although this might suggest that some other mechanism causes the effect of smoking on intervertebral discs, the tendency of smokers to deny that they cough may also contribute to the lack of association with coughing. Smoking was identified as significantly associated with low back pain episodes in reports by Frymoyer et al. (26), Svensson (27), and Svensson and Andersson (28). Svensson and colleague (27, 28) studied low back pain in relation to other diseases in a random sample of 940 Swedish men aged 40 to 47 years. Included was the prevalence of smoking as one of nine variables correlated to low back pain. Smoking habits were evaluated in the following manner: those who had consumed 1 g of tobacco daily or who had stopped smoking within 3 months before the interview were considered to be smokers; persons who had never smoked or who had previously smoked continuously for less than 1 month were considered nonsmokers; and the remaining were regarded as ex-smokers. One cigarette was considered equivalent to 1 g of tobacco, and a cheroot, 2 g. Four categories were used: 1 to 4, 5 to 14, 15 to 24, and 25 or more grams per day, respectively.

Of all men investigated, 42.5% were smokers, 23.2% were ex-smokers, and 34.2% were nonsmokers. Twenty-seven percent of the men had a daily consumption of more than 15 g of tobacco. The median value of the smoking habit duration among the smokers was 25 years. Productive cough was found in 21.1% of the men and breathlessness on exertion in 16.6%.

Svensson and colleagues (27, 28) found that the proportion of smokers among the men with low back pain was greater than among the controls, and that the association between low back pain and smoking persisted in the analysis. This interesting finding was also reported by Frymoyer et al. (30). In recent years, a positive correlation between smoking and diminished bone mineral content has been identified (33, 34). Microfractures of the trabeculae in the lumbar vertebral bodies caused by osteoporosis are a possible cause of low back pain (35). Further investigations are needed to clarify the connection between smoking and low back pain.

Frymoyer et al. (30) analyzed the records of 3920 patients and found that 11% of men and 9.5% of women reported an episode of low back pain during a 3 year interval. The low back pain sufferers were more likely to be cigarette smokers, particularly when smoking was accompanied by a chronic cough. In 203 men aged 18 to 55 years with low back pain, 33% were smokers, whereas only 13.6% of 1649 men without low back pain were smokers ($P < 0.001$). Of 196 women aged 18 to 55 years with low back pain, 26% smoked, whereas of 1872 women without low back pain, on 12.1% smoked ($P < 0.001$).

Frymoyer et al. (30) believed this to be an unexpected association between low back pain and smoking. They speculated that smoking might influence low back pain by one of three possible mechanisms. First, smokers might possibly be constitutionally or emotionally selected in a biased fashion for the low back complaint. Although smoking was related to anxiety and depression, this was found in preliminary analysis to be uniform throughout the male and female populations with and without low back pain. Hence, no specific selective bias appears to exist for low back pain patients who smoke and also have other psychological risk factors to a greater extent than the population at large. Second, smoking might produce significant hormonal and other alterations that increase the low back pain. Third, smoking

might produce other problems that lead to a greater incidence of low back pain. Those patients with low back pain had a greater reported incidence of chronic cough, which suggests the possibility that mechanical stresses induced by coughing may be relevant to the low back complaint. The extent to which chronic coughing and smoking are related to this population is currently under study. Biering-Sorenson (36) identified coughing, but not smoking, as important in the cause of low back complaints.

Traumatic Onset Low Back Pain Is Not Common

In a study of more than 11,000 patients, low back pain was generally not precipitated by a clearly defined injury. Only about one third of patients who are not involved in workers' compensation, insurance claims, or pending litigation can identify an event that triggered their back problems. Spontaneous onset is the natural history of most back pain (37). Body mass, physical work load, and a history of sick leave increased the risk of back pain disability, but smoking and sex did not. Individuals who engaged in at least 3 hours of leisure-time physical exercise per week had a significantly reduced risk of work disability (37). Cardiovascular physicians wearing lead aprons may have an increased risk for the development of back pain and intervertebral disc disease (38).

Space Weightless State Causes Disc Expansion and Back Pain

The altered mechanics caused by disc expansion during space weightless flight and rapid compression after flight may be involved in low back pain. Back pain even during missions lasting only 1 week, with relief occurring by sleeping in the fetal position, is reported (39).

Loss of Diurnal Height

Loss of height of 11% in lumbar discs in subjects performing normal activities is measured. Creep under controlled loading conditions is 7.3% in the flexed posture and 9.0% in the extended posture. Creep may be greater in an extended near-seated posture than in a flexed posture (40).

Role of Abdominal Aorta Atherosclerosis: Role in Degenerative Disc Disease

Atherosclerosis in the abdominal aorta and especially stenosis of the ostia of segmental arteries may play a part in lumbar disc degeneration (41). Diminished oxygen and nutrient supply to the intervertebral disc may be harmful and lead to degenerative changes.

The blood supply of the lumbar spine is as follows: the upper three lumbar levels receive blood supply from the four lumbar arteries arising from the posterior wall of the abdominal aorta. The fourth segment is supplied by the fourth lumbar artery and middle sacral artery arising just above the bifurcation of the aorta, and the fifth lumbar segment receives its blood supply from the middle sacral artery and the iliolumbar arteries from the internal iliac arteries.

Atherosclerosis of the abdominal aorta obstructs the ostia of the blood vessels supplying the lumbar segments (Fig. 2.10), and it may affect disc degeneration through nutritional insufficiency. Stenosis of the ostia may be slow and collateral circulation may establish alternate blood routes, but rapid obstruction might cause abrupt symptoms.

At best, the disc has a minimal blood supply, and any disruption of it can lead to symptoms. The degree of decreased blood flow necessary to lead to degenerative disc disease is yet to be determined (41)

Back pain may be related to work in the same sense as angina pectoris is. Postmortem lumbar aortograms were done in 56 cadavers to study differences between subjects with and without low-back pain in the lumbar and middle sacral arteries. Insufficient arterial blood flow may be an underlying factor for low-back symptoms. Atheromatous lesions in the abdominal aorta or congenital hypoplasia of the arteries may explain the angiographic findings and incidence of low back pain (42). Women with arterial disease are likely to have back pain and vertebral fractures. Aortic calcification predicted disc degeneration at the corresponding intervertebral level (43).

Low Back Pain Results in Fourfold Incidence of Death from Heart Disease

Middle-aged men who suffer from back pain had more than a fourfold increased risk of dying of heart disease in a 13-year follow-up study than comparable men with no back symptoms (44). In another study, no relation was found between back pain and death from ischemic heart disease in older men (45).

Blood Supply and Nutrition of the Disc Regulated by End Plate Receptors

Blood flow in the sheep lumbar spine was measured and data showed the existence of muscarinic receptors in vessels of the vertebral end plate, which suggests that the vasculature may influence disc nutrition (46).

Characteristics of Surgical Patients

Both an increased body mass index and a tall stature seem to have a clear association with those severe lumbar intervertebral disc herniations that require operative treatment (47). Former female elite gymnasts did not have more back problems than an age-matched control group (48).

DISC AND FACET BIOMECHANICS IN LOW BACK PAIN AND SCIATICA PATIENTS

Pain Source in Low Back Pain

Figure 2.11 demonstrates that practically every anatomic structure of the lumbar motion segment is capable of producing pain.

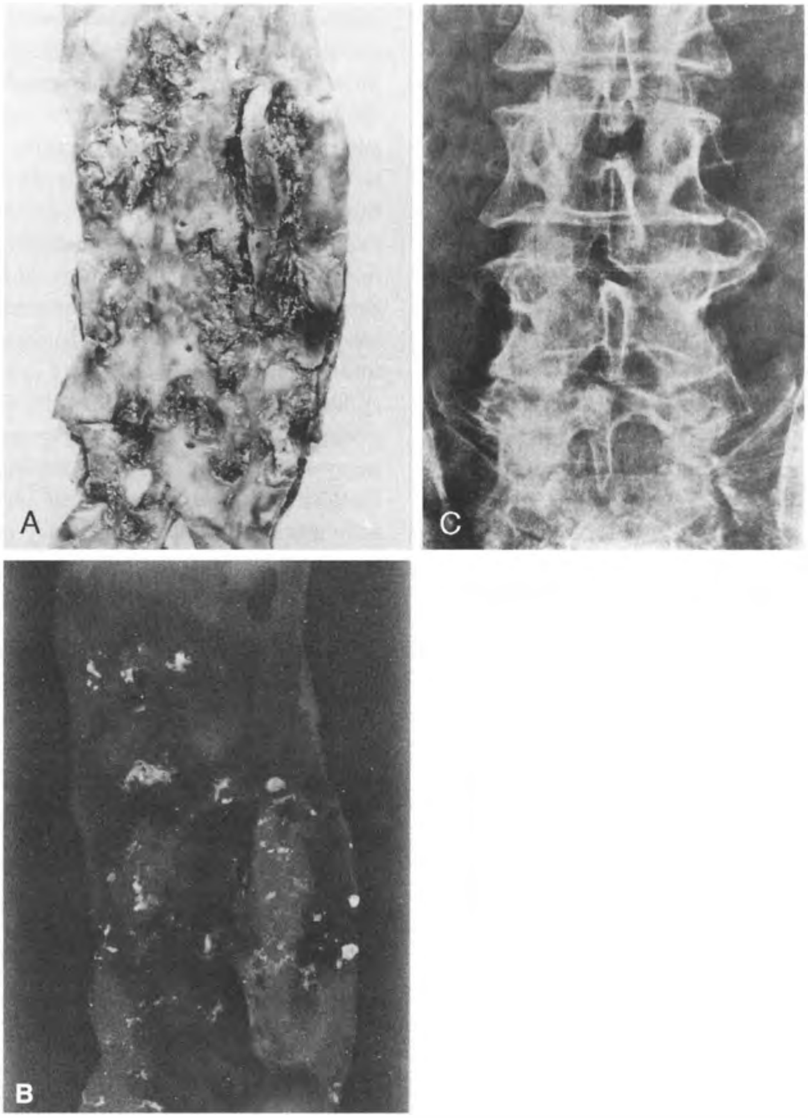

Figure 2.10. **A.** Abdominal aorta of a 59-year-old man. Advanced atherosclerotic changes with areas of ulcerations and intimal necrosis, and stenosis of ostia of several lumbar arteries. Ostia of the middle sacral artery is normal. **B.** Plain radiograph of aorta showing tiny calcium deposits scattered over large area. **C.** Anteroposterior radiograph of lumbosacral spine exhibiting large osteophytes and narrowing of intervertebral spaces at several levels. (Reprinted with permission from Kauppila LI, Penttila A, Karhunen PJ, et al. Lumbar disc degeneration and atherosclerosis of the abdominal aorta. Spine 1994;19(8):923–929.)

Pain source is an important place to start when discussing biomechanics and factors in the cause of low back pain. Information about the pain-sensitive structures of the lumbar spine must include the intervertebral disc, capsular structures, osseous structures, and the paraspinous muscles (49).

A synopsis of articles describing the sensory nerve supply of the intervertebral disc follow:

1. Bernini and Simeone (50) state that the sinuvertebral nerve (SVN) supplies the posterior longitudinal ligament, anulus fibrosus, and neurovascular contents of the epidural space.
2. Nachemson (5) found that the outer anulus and nerve root were the most pain-sensitive, and that they reproduced the patient's presurgical symptoms when stimulated 3 to 4 weeks postsurgically.
3. Farfan (51) points out that increasing evidence indicates that unmyelinated nerve endings are usually associated with pain reception in the posterior anulus, and they even penetrate the nucleus. The posterior longitudinal ligament is well innervated.
4. Helfet and Gruebel-Lee (52) have shown that when a radial tear penetrates the outer anulus, an attempt is made at healing by ingrowth of granulation tissue. Naked endings of the SVN have been identified in this granulation tissue. These may be pain receptors, which would explain discogenic pain in the absence of herniation.

5. Bogduk (53) believes that the SVN supplies the anulus fibrosus and the posterior longitudinal ligament. It runs up and down two segments, supplying the anulus and posterior longitudinal ligament above and below.
6. Tsukada (54) and Shinohara (55) claim that nerve fibers exist not only in the posterior longitudinal ligament but also in the nucleus and notochord. Malinsky (56) and Hirsch et al. (57) observed that nerve fibers penetrated into the outer layers of the disc. Tsukada (54) and Shinohara (55) found nerve endings in granulation tissue within the inner layers of the anulus and in the nucleus of some degenerated discs. In another article, Yoshizawa et al. (58) found profuse free nerve terminals in the outer half of the anulus but no such terminals in the nucleus.
7. Sunderland (59) stated that the recurrent meningeal nerve supplies the dura, intervertebral disc, and associated structures.
8. Edgar and Ghadially (60) say that the SVN divides into ascending, descending, and transverse branches adjacent to the posterior longitudinal ligament. Lazorthes et al. (61) state that this nerve supplies the neural laminae, the intervertebral disc at the adjacent levels, the posterior longitudinal ligament, the internal vertebral plexus, the epidural tissue, and the dura mater. Concerning the tissues supplied by the SVN, however, disagreement exists; some authorities do not believe that there is such a wide distribution. Tsukada (54) and Shinohara (55) found that the outer anulus is innervated in a normal disc but that fine nerve fibers accompany granulation tissue present in a degenerated disc. In one instance, fine fibers were observed in the nucleus. Most of these were naked nerve endings and probably mediated pain sensation. Edgar and Ghadially (60) found that sinuvertebral nerves supply the anterior dura. In spinal stenosis, therefore, irritation of the SVN may be the mechanism of claudication pain.

Well-Substantiated Neurologic Facts

In discussing the lumbar intervertebral disc syndrome, Bogduk (53) states that four elements of the nervous system may be involved in the production of this syndrome: the lumbosacral nerve roots, the spinal nerves, the dorsal rami, and the sinuvertebral nerves. The nerve root is usually irritated because of its being stretched over a protruding or prolapsed disc. Irritation of the spinal nerve may result from arthrosis of the zygapophysial joints, ligamentum flavum hypertrophy, osteophytes, intervertebral disc protrusion, subluxation, spondylolisthesis, infection, tumor, fracture, Paget's disease, or ankylosing spondylitis. The dorsal rami (which supply the zygapophysial joints, the erector spinae muscles and their related fascia and skin, the periosteum of the vertebral arches, the multifidus muscles, the interspinous ligament, and the interspinous muscles) are irritated by articular facet arthrosis, subluxation, sacroiliac joint arthrosis, spinous process impingement, strain of the sacral joints, hyperlordosis, scoliosis, myositis, muscle spasm, and reactions secondary to sclerosis or arthrosis of the articular facets. The SVN, also known as the recurrent meningeal nerve, supplies the posterior longitudinal ligament as well as the anulus fibrosus of the disc. A descending branch runs caudally for a maximum of two segments, sup-

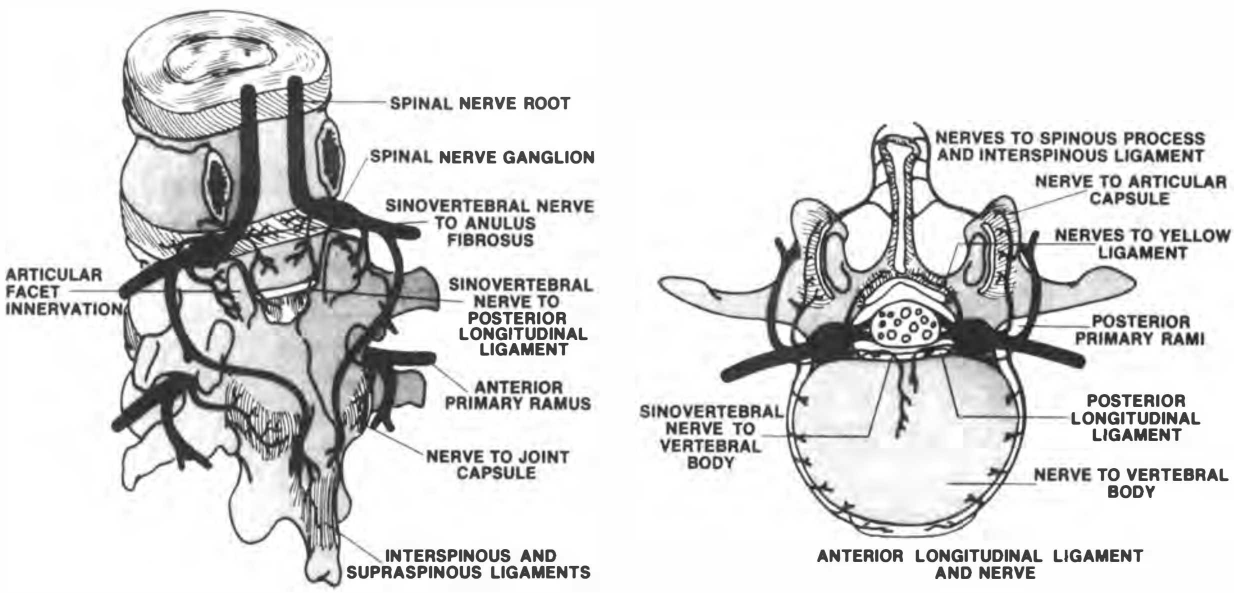

Figure 2.11. This figure demonstrates clearly the sensory innervation of practically every anatomic structure in the spine. The anulus fibrosus, the major ligaments, the intervertebral joints and their capsules, the vertebral body, and all the posterior osseous structures are provided with sensory innervation. Thus, virtually any structure can be a potential source of spine pain. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: Lippincott-Raven, 1978:279.)

plying the anulus fibrosus and the posterior longitudinal ligament. An ascending branch may also behave similarly. Any lesion of the anulus or posterior longitudinal ligament is capable of setting up pain impulses in the sinuvertebral nerve.

Two basic causes of low back pain are internal derangements of the intervertebral disc and irritation of the zygapophysial articulation. The ontogeny of low back pain concerns two structures: the disc and facet. Debate continues to which is the initial lesion and which is a secondary or compensatory change. After study, I believe that the initial change takes place in the intervertebral disc, which later affects the articular facet. Vernon-Roberts and Pirie (62) state that a direct relationship exists between the degree of disc degeneration, the marginal osteophyte formation on vertebral bodies, and the apophyseal joint change, which suggests that disc degeneration is the primary event leading to the clinical condition of degenerative spondylosis.

INTERVERTEBRAL DISC HAS DUAL NERVE SUPPLY: AUTONOMIC AND SPINAL NERVE

The anterior portion of lumbar intervertebral discs is innervated by sympathetic fibers alone, whereas the posterior portion is innervated by the sinuvertebral nerve. No boundary or septum is found between the anterior and posterior portion of the intervertebral discs histologically or developmentally. No other organs appear to be known to have such dual innervation.

Mechanical stimulation of the posterior portion of lumbar intervertebral discs causes low back pain, even after two roots on the same side have been anesthetized. These findings indicated that the nerve fibers in the SVN are not derived from the spinal nerves but suggest that they may originate from the sympathetic nerves, transmitting discogenic low back pain. In the modern description of the autonomic nervous system, efferent and afferent fibers are included.

Discogenic low back pain is poorly localized and often lacks tenderness to palpation over the pain site. This kind of referred pain resembles visceral pain. Visceral pain is transmitted by sympathetic afferent fibers. Discogenic low back pain could be transmitted by sympathetic nerves. From the point of view of innervation, discogenic low back pain may have similar features to visceral pain (63).

Afferent Pathways of Discogenic Low Back Pain

Discogenic low back pain is transmitted nonsegmentally by visceral sympathetic afferents mainly through the L2 spinal nerve root, presumably via sympathetic afferents from the sinuvertebral nerve, which may be perceived as referred pain in the L2 dermatome as shown in Figure 2.12 (64). This is based on evidence showing that lumbar sympathetic afferents play a role in transmitting low back pain and stating that low back pain is induced by stimulation of the lumbar sympathetic trunk and that it transmits pain (64). The sympathetic trunk originates from

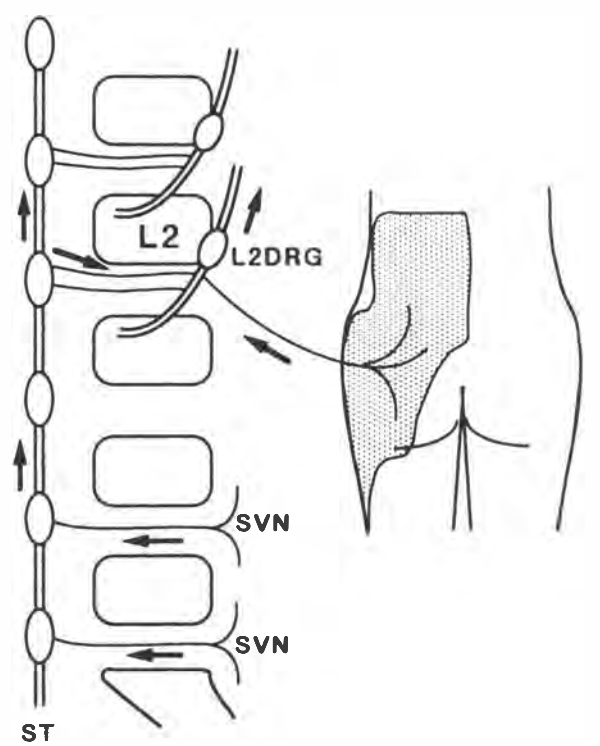

Figure 2.12. Diagram showing the proposed afferent pathways of discogenic low back pain. Pain from a lower lumbar disc is transmitted nonsegmentally by visceral sympathetic afferent fibers, mainly from the L2 spinal nerve root. This results in referred pain in the L2 dermatome (*DRG*, dorsal root ganglion; *SVN*, sinuvertebral nerves; *ST*, sympathetic trunk). (Reprinted with permission from Nakamura S, Yakahashi K, Takahashi Y, et al. The afferent pathways of discogenic low back pain: evaluation of L2 spinal nerve infiltration. J Bone Joint Surg Br 1996; 78B(4):606–612. Copyright 1996, The British Editorial Society of Bone and Joint Surgery, London.)

the myelomeres of T1 to L2 with L2 being the only dermatome corresponding to the low back.

Discogenic low back pain is relieved by L2 spinal nerve root injection and could serve as a diagnostic or treatment procedure.

Inguinal Pain

Inguinal pain in low back pain patients is suggested to be from L1 and L2 dorsal root ganglions via irritation of the anterior portion of the intervertebral disc (64A, 65).

Iliac Crest and T11–L1 Pain May Indicate L1–L2 Dorsal Ramus Entrapment

Unilateral low back pain with nonradiating pain localized to the lumbosacral triangle and buttock should include examination for tenderness at the posterior crestal point. When this tenderness is present along with ipsilateral articular tenderness at the T11–T12 or T12–L1 level and pinch-roll tenderness over the buttock, sensory nerve conduction studies of the L1–L2

dorsal rami may be useful in detecting conduction abnormalities that indicate an entrapment neuropathy at the iliac crest. If, in fact, this diagnostic entity is shown to exist, this group of patients may benefit from therapeutic interventions directed toward relief of the entrapment, including therapeutic injection or, possibly, surgical release (66).

Dura Mater Supplied with Sympathetic Nerves May Be a Source of Low Back Pain

Sensory fibers innervate the lumbar dura mater via L2–L3 sympathetic nerves in rats. Sympathectomy reduced the number of these nerve fibers in the lumbar dura mater. Sympathetic nerves may play an important role for low back pain involving the lumbar dura mater (67).

Lumbar Spine Dura, Ligaments, Discs, and Vertebral Bodies As a Source of Pain

Back Pain (70%) Should Be Diagnosable with Correct Testing

Bogduk (68) states that, collectively, lumbar zygapophysial joint pain, internal disc disruption, and sacroiliac joint pain account for nearly 70% of chronic low back pain. It has commonly been believed that in more than 70% of patients with chronic low back pain a diagnosis cannot be made. Painful discs occur only in patients with back pain. In the back, zygapophysial joint pain is found in only a minority of patients: 40% or as little as 15%, depending on country and clinical circumstances. However, internal disc disruption accounts for a further 39% of cases of back pain. Cervical zygapophysial joint pain accounts for more than 50% of chronic neck pain after whiplash. It is not 70% of low back patients who *defy* diagnosis, but 70% of patients who could be diagnosed. A diagnosis is impossible only for those who refuse to use available techniques (68).

MECHANISM OF LOW BACK PAIN IS UNCERTAIN—DISC, LIGAMENT, DURA?

The disc may be the primary source of pain, but the mechanisms of pain production are uncertain. Pain in and around the disc can originate in interdiscal nerve endings, in the posterior longitudinal ligament (PLL) near attachments to the disc, or in the ventral dura (69).

Pain arising from the intervertebral disc (IVD) has been demonstrated by several investigators (69). Severe pain with poorly localized deep aching across the back when 0.3 mL of 11% NaCl was injected into the IVDs of human volunteers has been shown. A surgical patient had backache reproduced when a nylon suture looped through the L5–S1 disc was pulled. A report of 144 back surgery patients studied under progressive regional anesthesia found the disc anulus was exquisitely tender in one third, moderately tender in one third, and insensitive in one third.

Profuse innervation extending as deep as half of the anular thickness has been reported as well as relatively dense innervation of the disc anulus, but only in the superficial layers (69).

What Is the Pain Production from Facet Joint Versus Disc Irritation?

Pain receptors (nociceptors) are found in disc, facet, nerve root, dorsal root ganglion, and muscle, some of which seem to be more pain sensitive than others. This raises the question of what structures of the lumbar spine do indeed cause the pain experienced by humans (70).

Disc and Facet As a Combined Source of Pain Is Rare

Ninety-two consecutive patients with chronic low back pain were studied using both discography and blocks of the zygapophysial joints. *Conclusion: In patients with chronic low back pain, the combination of discogenic pain and zygapophysial joint pain is uncommon* (71).

Pain arising from the disc is more common than pain arising from the zygapophysial joint. However, 49% of patients clearly had neither discogenic nor zygapophysial joint pain.

Zygapophysial joint pain is highly unlikely to occur in patients with symptomatic lumbar intervertebral discs. Discogenic pain appears to be a singular, independent disorder. Disc disease sufficient to cause discogenic pain does not, by and large, disturb the zygapophysial joints in a way to render them symptomatic (71).

Bogduk (72) reports no scientific data to sustain the belief that muscles may be a source of chronic pain.

Nucleus Pulposus Produces Inflammatory Chemicals

Based on the findings of clinical, histologic, biochemical, and neurophysiologic studies, the nucleus pulposus appears to contain a chemical or chemicals that are inflammatory, neurodegenerative, and, in the acute stage, neuroexcitatory. The causative agents may include hydrogen ions, phospholipase A_2 (PLA_2), immunoglobulin G, or stromelysis. These chemicals may play a role in both disc pain and increased sensitivity of inflamed nerve roots (70).

Human discs have been demonstrated to contain high levels of PLA_2, which theoretically has an inflammatory potential. Herniated lumbar discs have a higher level of PLA_2 than do normal discs (73).

ANULUS FIBROSUS IS THE MOST PAIN-SENSITIVE STRUCTURE IN THE LUMBAR SPINE

Kuslich et al. (74) studied the pain distribution and pain intensity patterns of lumbar structures (facet joint, disc, ligamentum flavum, muscle, scar tissue, nerve roots, and cartilage) of progressively anesthetized patients. The following important findings emerged from this study:

1. The outer anulus fibrosus of the intervertebral disc is the tissue of origin in most cases of low back pain. The pain produced was most similar to the preoperative pain of the patients. Application of local anesthetic to the disc obliterated the pain. Referral of pain depended on the exact site of the anulus being stimulated. The central anulus and posterior longitudinal ligament produced central back pain when stimulated. Stimulation to the left or right of center of the posterior longitudinal ligament directed pain to the side of the back being stimulated. This finding is felt to correlate with back pain on the side of disc "bulge."
2. The facet synovium was never sensitive.
3. The facet articular cartilage was never tender.
4. The facet joint capsule was sometimes tender; however, when it was, it referred pain to the back or, very rarely, to the buttock, and never to the leg.
5. Kuslich et al. also suggest that the facet contact with the posterior disc in cases with a trefoil-shaped vertebral canal could cause low back pain that has been called "facet syndrome."
6. The vertebral end plate caused deep, rather severe low back pain when compressed.
7. Buttock pain was found when the outer anulus and nerve root were irritated. Other tissues rarely produced buttock pain when irritated.
8. Normal nerve roots were completely insensitive to pain.
9. Muscles never produced pain under gentle pressure, whereas localized forceful stretching at the base of a muscle, especially at the site of blood vessels or nerves, or at its attachment to bone usually produced a localized low back pain. The pain was felt to arise from local vessels and nerves rather than from muscle bundles.
10. Lumbar fascia irritation at the supraspinous ligament produced low back pain.
11. Sciatica could be produced only by stimulation of a swollen, stretched, or compressed nerve root.
12. The surface of bone, even at the level of the periosteum, was insensitive. The spinous processes, laminae, and facet bone could be removed with a rongeur without anesthetic.
13. Scar tissue was never tender. It acts to fix the nerve root in one position, thus increasing the susceptibility of the nerve root to tension and compression.

The anulus fibrosus of the disc was the most pain sensitive tissue producing low back pain. Muscle, fascia, and bone were not found to be sensitive (74) (Fig. 2.13).

Porter (75) states the most common cause of low back pain of mechanical origin with or without referred pain is an acute disc protrusion. This may present as low back pain with or without lower extremity pain. The pain is caused by stretching of the peripheral discal anular fibers, which are pain sensitive. The shape and size of the vertebral canal will determine the severity and type of symptoms emanating from disc protrusion. Persons with patent and wider canals will be less susceptible to nerve root compression than patients with tighter stenotic canals (Fig. 2.14).

Chemical Sensitization May Cause Painful Disc

It may be that various neurochemical changes within the intervertebral discs are expressed by sensitized (injured) anular nociceptors, and in part modulated by the dorsal root ganglion. Therefore the concomitant pain sometimes associated with an abnormal discogram image may in part be related to the chemical environment within the intervertebral disc and the sensitized state of its anular nociceptors (76). It therefore appears that neuropeptides may mediate or influence certain stages of joint inflammation, although it is not known which neuropeptides are most important in this respect.

Nociceptors are the peripheral terminal endings of sensory neurons that are selectively responsive to potentially or overtly injurious stimuli that cause pain in humans. They play three important roles in the process of inflammation: (*a*) By evoking pain, they signal the presence of noxious physical or algesic chemicals; the latter, when endogenous, are inflammatory mediators originating from non-neural tissues (e.g., mast cells and blood vessels) and from peripheral endings of certain sensory afferent nerve fibers. (*b*) Some nociceptors become sensitized; that is, they develop a lowered response threshold and enhanced response to suprathreshold stimuli after exposure to noxious physical stimuli or inflammatory mediators.

It is probable that nociceptors responding directly to algesic stimuli serve as effectors and can release peptides and other neuromodulators that increase the excitability of neighboring

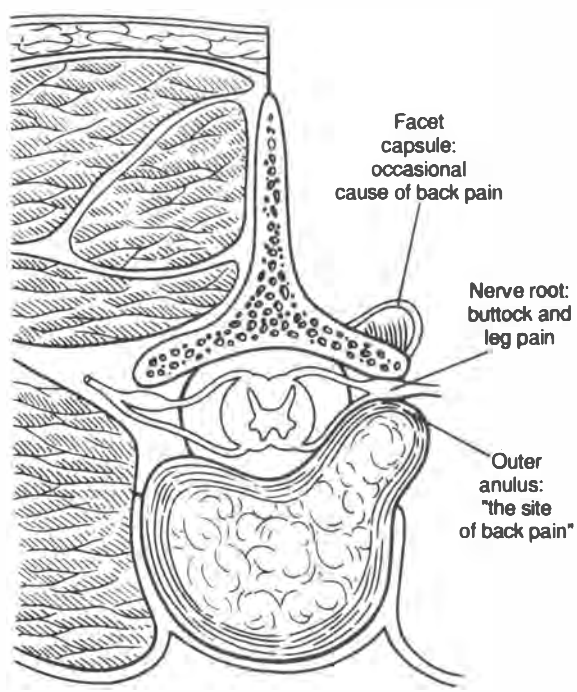

Figure 2.13. Different structures of the spine will produce pain when stimulated intraoperatively. (Reprinted with permission from Olemarker K, Hasue M. Classification and pathophysiology of syndromes. In: Weinstein JN, Rydevik BL, Sonntag VKH, eds. Essentials of the Spine. New York: Raven Press, 1995:11–25.)

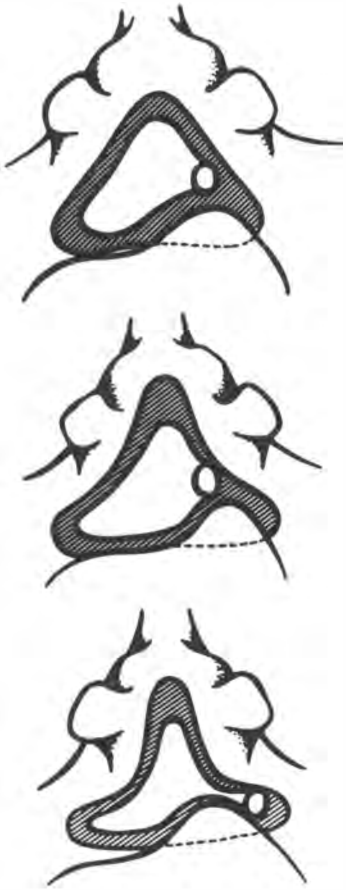

Figure 2.14. Diagram showing how a disc protrusion will have different effects depending on the size and shape of the vertebral canal. A protrusion will not significantly compromise a nerve root in a large triangular canal (*top*), nor in a lightly trefoil canal (*middle*). However, a similar disc protrusion into a small, markedly trefoil-shaped canal will significantly compress the nerve root in the lateral recess (*bottom*). (Reprinted with permission from Porter RW. Pathology of spinal disorders. In: Weinstein JN, Rydevik BL, Sonntag VKH, eds. Essentials of the Spine. New York: Raven Press, 1995:29–54.)

nociceptors, modulate the inflammatory process, and promote tissue repair (76).

Low Back Pain and Radiculopathy Can Arise from within the Disc

In some patients with low back pain and unilateral or bilateral radiation to the lower extremities, the pain arises from within the disc (77). The pain-sensitive structures responsible for the radiating pain to the lower extremity are located somewhere inside the disc, probably in the external part of the anulus fibrosus and in the longitudinal ligaments.

A patient's painful symptoms can be reproduced with a discographic injection of contrast medium into the disc demonstrating an anular tear, and then the symptoms can be relieved by injecting a local anesthetic. This anesthetic does not need to extend beyond the disc margins to relieve low back or leg pain, thus supporting the existence of discogenic pain. This leads to the conclusion that a simple disc rupture, without direct nerve root compression by disc material, can account for low back pain with radiating pain to the leg.

The fact that these discs are generally labeled "degenerated bulging discs" misleads the doctor and the patient to think that the cause of the symptoms has not been identified (77).

The intervertebral disc can be a source of pain without rupture or herniation. Structurally and mechanically, the anulus fibrosus resembles a ligament. As with other ligaments, it is innervated (at least in its outer third or outer half) and susceptible to mechanical injury; therefore, as with other ligaments, it s capable of being a source of pain if injured (78).

What is tantalizingly seductive about these deductions is that they describe what resembles so many presentations of back pain that are interpreted as back strain, ligament strain, or mechanical low back pain; and indeed, the "ligament" in question is the anulus fibrosus, which when strained, is indeed the causative lesion. What has escaped attention to date is the location of the lesion—not in the "disc" as such, but specifically in the anulus fibrosus (78).

Internal disc disruption can be symptomatic in its earlier stages before disc herniation occurs. If the degradation process of the nucleus reaches the outer third of the anulus fibrosus it can directly affect the nerve endings therein, which provides a mechanism for chemical pain in internal disc disruption. Alternatively, if the anulus fibrosus is affected by internal disc disruption, some of its collagen fibers may be disrupted, leaving fewer intact fibers to bear the stresses imposed on the anulus by normal movements. Reduced in number, these intact fibers must nonetheless bear a normal load; therefore, the strain they experience must be greater than normal, and it may exceed the threshold for nociception. This provides a basis for mechanical pain in internal disc disruption. Moreover, it is feasible that both chemical and mechanical mechanisms may operate concurrently, with the chemical mechanism producing a constant background of dull pain (like that of a sterile abscess) with bouts of mechanical pain superimposed whenever the anulus fibrosus is stressed by movements or compression (analogous to the increase in pain when an abscess is palpated) (78).

Muscle Strain Is Really Disc Disruption

Most chronic muscle strains are actually the result of degenerative or herniated discs (79). There is no such thing as chronic muscle strain; most are actually degenerative or herniated discs causing secondary muscle spasm (80).

Anulus Fibrosus Tears May Cause Discogenic Low Back Pain

Osti et al. (81) found peripheral anular tears more frequently in the anulus except at the L5–S1 level. Circumferential tears were equally distributed between the anterior and the posterior anulus. Almost all radiating tears were in the posterior anulus, and they were closely related to the presence of severe nuclear degeneration. Peripheral tears are caused by trauma rather than by biochemical degradation; they develop indepen-

dently of nuclear degeneration and are responsible for discogenic low back pain. McNally et al. (82) found discogenic pain is caused by anomalous loading of the posterolateral anulus or nucleus pulposus.

Maezawa and Muro (83) found herniated nuclear material to produce high pain provocation on discography. Yussen and Swartz (84) stated that herniated nucleus pulposus may produce vague low back pain without radiculopathy.

Sustained Loading Transfers Creep Load from Nucleus to Anulus

The central region of the disc acts as a hydrostatic "cushion" between adjacent vertebrae with creep reducing the hydrostatic pressure in the nucleus pulposus by 13 to 36%. The water loss from the nucleus transfers the load from the nucleus to the anulus. Such stress concentration may lead to pain, structural disruption, and alterations in chondrocyte metabolism (85).

Stress distributions within the disc show that the highest intradiscal stress is in the inner and middle anulus fibrosus, not the nucleus pulposus. As the disc is probably the most common source of chronic low back pain, stimulation of the anulus by posterior herniation of nuclear material or internal disruption of innervated tissues is the possible source of the pain (86).

Anulus Fibrosus Has Nociceptors and Proprioceptors

Afferent and efferent nerve fibers exist within the outer anulus fibrosus. Two types of terminal structures are associated with afferent nerves in the intervertebral disc: the complex, probably proprioceptive; and the free nerve endings, probably nociceptive. The form of nociception is probably not mechanical or thermal, but instead chemical, the stimulus originating in the environment of the inner IVD. It is suggested that the role for the free nerve endings is related to vascular changes and to the introduction of the immune system into the outer anulus fibrosus. Healthy motion of the intervertebral joint is seen as a method of maintaining the nutritive supply to the outer elements, preventing disruption of diffusion through the IVD, which would otherwise lead to local waste product buildup with its obvious consequences (87, 88).

Coppes et al. (89) *found nerve endings in abnormal discs that penetrated the anulus to reach the nucleus pulposus.*

Autonomic and Spinal Nerve Innervation to the Disc

Anterior disc extrusions (Fig. 2.15) are reported to cause dysfunction of the autonomic nervous system, resulting in pain referral into Head's zones and into the flank, groin, buttock, and thigh. Twenty-nine percent of peripheral disc protrusions were anterior and 56% were posterior (Fig. 2.16) (90). The *vertebrogenic* symptom complex of disc irritation includes:

1. Local and referred pain
2. Autonomic reflex dysfunction within the lumbosacral zones of Head

Figure 2.15. Radiograph showing anterior and posterior disc protrusion (*arrows*) as a cause of the vertebrogenic symptom complex of pain.

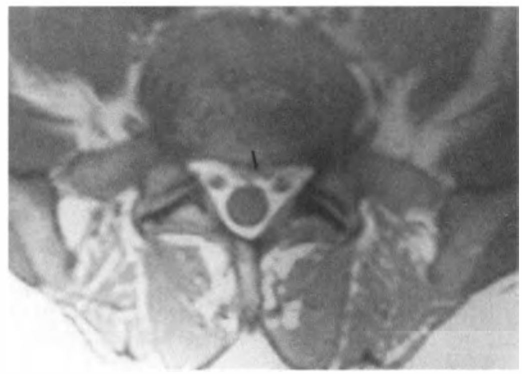

Figure 2.16. Radiograph showing the posterior central disc herniation (*arrow*) that causes the vertebrogenic symptom complex discussed in the text.

Disc-generated pain arises with afferent sensory fibers from two primary sources (90):

1. Posterolateral neural branches emanating from the central ramus of the somatic spinal root
2. Neural rami projecting directly to the paravertebral autonomic neural plexus

Thus, conscious perception and unconscious effects originating in the vertebral column, although complex, have definite pathways represented in this dual peripheral innervation associated with intimately related and/or parallel central ramifications. It is further proposed that the specific clinical manifestations of the autonomic syndrome are mediated predominantly, if not entirely, within the sympathetic nervous system (90).

Rat studies have shown autonomic nerves in the lumbar spine in the bone and periosteum of the vertebral body, disc, dura mater, and in the spinal ligaments. Human studies on "disc pain" have shown substance P and calcitonin gene-related peptide within the nerve fibers of the peripheral anulus fibrosus (91).

Sensory innervation of the rat disc has both myelinated and unmyelinated components, the latter being more extensive. Both types of innervation appear to be restricted to the outermost rings of the anulus fibrosus (92).

Pain Receptors in Anterior Disc and Ligament

Mechanoreceptors are found in the outer two to three lamellae of the human intervertebral discs and anterior longitudinal ligaments in 50% of discs and 15% of scoliosis patients with low back pain. The receptors resemble Pacinian corpuscles, Ruffini endings, and most frequently, Golgi's tendon organs. These provide the individual with sensation of posture and movement, and, in the case of Golgi tendon organs, of nociception. In addition to providing proprioception, mechanoreceptors are thought to have roles in maintaining muscle tone and reflexes. A greater incidence of mechanoreceptors was found in patients with low back pain compared with those in pain-free patients with scoliosis (93).

Posterior Longitudinal Ligament Causalgia

The lumbar posterior longitudinal ligament is dually innervated by two distinctive systems of nociceptive fibers. One of the systems is polysegmental sympathetic innervation, which under longstanding irritation becomes chronic and resistant to conventional treatment. This pain is called "causalgia of the spine" and it may be a clinical feature of chronic low back pain syndrome. The other is unisegmentally innervated and not associated with autonomic fibers (94). In causalgia, a severe painful condition of the locomotor system, relief can be obtained by regional block with guanethidine, which causes inhibition of postganglionic sympathetic efferents (91).

Supraspinous Ligament and Thoracolumbar Fascia Show Innervation

Bundles of nerve fibers are found in all ligaments except those from the ligamentum flavum. The supraspinous ligament and lumbodorsal fascia show individual axons and free nerve endings (95). The thoracolumbar fascia is found to contain free nerve endings and two types of encapsulated mechanoreceptors (Ruffini's and Vater-Pacini corpuscles). The presence of these nerve endings supports the hypothesis that the thoracolumbar fascia may play a neurosensory role in the lumbar spine pain mechanism (96).

Disc Nerve Supply Interruption Relieves Back Pain in Failed Back Surgical Cases

Fifty percent of patients whose back surgery failed received pain relief with radiofrequency cutting of the gray rami communicantes to interrupt the afferent conducting fibers from the anterolateral and anterior parts of the anulus fibrosus (97). Adams and Hutton (98) find that flexion improves the transport of metabolites into the intervertebral discs.

Comparison of Results of Disc and Facet Joint Injection on Pain Origin

Mooney and Robertson (99) reported on the relief obtained by facet joint injection in 100 patients with low back pain due to facet syndrome and stated that 20 patients remained asymptomatic at 6 months follow-up. Sixty-two patients had some type of relief, whereas 38 did not. Of the 62 receiving some relief, only 20 had complete relief at 6 months and 32 had partial relief. That means that one fifth of the 100 patients had complete relief and one third had partial relief, showing a total of half who had some relief at 6 months of treatment.

Difference of opinion exists over the benefit of facet injection relief. Jackson et al. (100) found that 390 patients with low back pain, normal neurologic examinations, and no root tension signs, underwent facet joint arthrograms and intra-articular injection of local anesthetic and cortisone. Initial mean pain relief was seen in only 29% of the cases. It was concluded that the facet joints were not commonly the single or primary source for low back pain in most (90%) of the 390 patients studied. The facet joints were not commonly the single or primary source of low back pain in most of the patients studied. Murtagh (101) stated facet injection (with lidocaine and betamethasone) was a diagnostic rather than a therapeutic maneuver. He found that 54% of 100 patients with posterior compartment lumbar spinal axis pain syndromes and focal tenderness received relief at the end of 3 months. Moran et al. (102) injected the facets of 54 patients for a total of 143 facets so treated. Only 9 (16.7%) diagnosed as facet joint causing the pain gained relief of the pain. It was shown that extravasation into the epidural space occurs following rupture of the joint capsule of the facet, which explains why good therapeutic results can be obtained if large amounts of the therapeutic agent are used.

Lewinnek and Warfield (103) injected the facets of 21 patients with low back pain and found 75% to have an initial response, but only 6 (33%) had relief at 3 months. Repeat injection only afforded temporary relief.

Disc or Facet As the Cause of Back Pain

Vernon-Roberts and Pirie (62) found a direct relationship between the degree of disc degeneration, marginal osteophyte formation on vertebral bodies, and apophyseal joint changes, which suggests that disc degeneration is the primary event leading to the clinical condition of degenerative spondylosis. They also found evidence that enables them to speculate on the role of prolapse in disc degeneration and in the genesis of osteoarthrosis of the apophyseal joints.

Nachemson (5) found that arthrosis of the articular facets was always secondary to disc degeneration. Thus, it is strongly implied that internal derangement of the intervertebral disc, namely, the nucleus pulposus, begins the aberrant mobility of the lumbar spine. The degenerative changes occurring thereafter in the disc spread posteriorly into the arch of the vertebra. We know that both the disc and the facet are pain-producing entities and that specific attention must be given to both of these structures in the treatment of low back pain. Furthermore, it also seems most likely that a combination of surgery and manipulation may be the answer for many people (i.e., surgery for the disc prolapse and manipulation for the altered motoricity of the articular facet.

The effects of rotation have been well summarized by Eagle (104), who states that the main cause of severe long-lasting back pain is the damaged intervertebral disc, and once a disc is damaged there is nothing a surgeon can do to repair it. Nor can discs repair themselves. Therefore, if we are to prevent back pain, it would be useful to know how much stress the disc fibers can withstand before they give way.

Eagle (104) quotes the work of Hickey and Hukins (105) of Manchester University, who find that the most hazardous maneuvers to the low back are bending and twisting. They won the 1979 Volvo Bioengineering Award for their work proving that anular failure and tearing are caused by torsion and forward bending, causing nuclear protrusion and low back pain. They found that the maximal rotation that will not damage the anular fibers at L5–S1 is 3°.

Miller (106) states that during the lifting of 200 pounds, the disc carries an average of 91% of the load and the facet joint carries no more than 12%. Low facet joints put more weight on the disc than do high facet joints. *The facets carry very little weight on compression but accept large loads on bending.* The amount of load on the facets is 50% on flexion and extension and 30% on torsion.

An in vitro experimental study was carried out to measure the induced loading on human lumbar facets with varying amounts of compressive axial load (107). Testing was done on the L2–L3 and L4–L5 spinal motion segments obtained from cadavers at autopsy. The compressive loading was applied with the spinal specimens first in a neutral position and then in an extended position. In particular, this study demonstrated that the absolute facet loads remain relatively constant with increasing segmental compressive loads such that the facet load expressed as a percentage of the load applied to the segment decreases with increasing axial loads. It also demonstrated that with increasing loads in extension the contact area moves cranially at L2–L3 and caudally at L4–L5. Furthermore, it indicated that after a facetectomy the load on the remaining facet is reduced substantially, although peak pressure increases. Finally, this study demonstrated that a substantial difference in facet loadings is found between the L2–L3 and the L4–L5 segments.

A comparison of segments at L2–L3 and L4–L5 at different axial loads in the neutral position shows that the facets at L2–L3 generally take more load than those at L4–L5. The same trend is also observed during extension. Furthermore, the normal load on the facets is always greater in extension than in the neutral position. This holds true for both the L2–L3 and L4–L5 levels.

Observations based on these data indicate:

1. The average peak pressure for all axial compressive loads is higher in extension than in the neutral position at both the L2–L3 and the L4–L5 levels.
2. The peak pressure is generally higher at the L2–L3 level than at the L4–L5 level in both the neutral and the extended positions.

Facet pressure rather than the facet load, therefore, may be playing a significant role in the degenerative changes of facets.

Contrary to expectations, a unilateral facetectomy causes a significant reduction in the load borne by the remaining facet in both the neutral and the extended positions. This may be explained by the fact that because the facet load on the left side is eliminated by performing a unilateral facetectomy, equilibrium is substantially altered. The superior vertebral body is now free to drift away from the inferior body, thus reducing positive contact at the remaining facets. This phenomenon again reinforces the above observation that pressure rather than load is the precipitating factor in facet degeneration. A second unexpected phenomenon observed in this study is that in many cases the contact decreases with increasing loads (107).

Vulnerability of Nerve Roots to Compression Defects

The dorsal nerve roots have a larger diameter than the ventral nerve roots, which some feel may explain the greater susceptibility of the sensory axons to compressive forces. The S1 nerve roots are approximately 170 mm long, whereas the L1 nerve roots are 60 mm long. The nerve roots as well as the spinal nerves are composed of axons that have arisen within the substance of the spinal cord and course to their final destination in the periphery. These axons may exceed 100 cm in length (108).

Spinal nerve roots lack the connective tissue protection that sheaths peripheral nerves. This sheathing has considerable mechanical strength and possesses properties to form a barrier to diffusion of certain molecules. The spinal nerve roots, therefore, are at a disadvantage mechanically and, possibly, biochemically. The nerve roots are surrounded by cerebrospinal fluid, however, and this, together with the dura, does give the spinal nerve roots an element of mechanical protection. The dura of a spinal nerve root appears to be continuous with the epineurium of the peripheral nerve.

It must be kept in mind that the nerve root complex must be extraordinarily mobile. Nerve roots must change length depending on the degree of flexion, extension, lateral bending, and rotation of the lumbar spine. Lumbar nerve roots limited in motion by fibrosis of either intraspinal or extraspinal origin will create traction on the nerve root complex, causing ischemia and secondary neural dysfunction. This fact must also be kept in mind during the rehabilitation process. Flexibility exercises must be designed to maintain nerve root mobility.

Intraneural blood flow is markedly affected when the nerve is stretched about 8% over the original length. Complete cessation of all intraneural blood flow is seen at 15% elongation.

The dorsal root ganglion, because of its fibrous capsule as well as its rich vascular supply, may, indeed, be more susceptible to changes in intraneural blood flow as well as to the development of secondary intraneural edema with consequent fibrotic change. This may explain sensory symptoms even in the absence of evidence of sensory loss on gross neurologic examination (108).

Weightbearing Stresses on the Disc and Facet

Changes in body height have been used as a measure of summarizing disc compression caused by creep. Under controlled circumstances, changes in body height can be used as a measure

of the load on the spine. This can be of great value in ergonomic evaluations of workplaces, equipment, and tasks. However, the many factors that influence the shrinkage as a response to a certain load have to be controlled. The duration of the load is one obvious example. Also, age and individual factors, time of day, hours of sleep, arising time, and previous loads are other influences. An interesting fact is that the spine recovers quickly when it is unloaded (109).

The in vitro static load displacement characteristics of the intact and injured human lumbar intervertebral joint have been investigated in a loading apparatus that allows entirely unconstrained relative motion between the joint members. The spatial relative displacement produced by a given load, with and without preloads, was measured. The significant observations are summarized as follows (110):

1. Joint flexibilities measured by raising the initial intradiscal pressure show that (*a*) for force loads, the joint is most flexible in anterior shear and least flexible in axial compression; the flexibility in anterior shear is an order of magnitude greater than in compression. The flexibilities in posterior shear and lateral bending are one half and one third of that in anterior shear, respectively. (*b*) For torque loads, the joint is most flexible in flexion. The flexibility in extension is about 60% of that in flexion, whereas in lateral bending it is approximately an average of those in flexion and extension. The joint is least flexible in axial torque; flexibility is less than 30% of that in flexion.
2. The load displacement results of the two sequential sectioning series of experiments show that: (*a*) In the load range considered in the experiment, the disc is by far the major load-bearing element in lateral and anterior shears, axial compression, and flexion. In lateral shear and axial compression, at higher displacements, the facets can transmit part of the load through the joint. Also, with increased displacement, the facet capsules (in anterior shear) and the facet capsules and the posterior ligaments (in flexion) are likely to be important. (*b*) The facets play a major load-bearing role in posterior shear and axial torque (110).

Facet Stiffness Under Loading

Three-dimensional load deformation data were obtained for intact posterior elements and isolated facet joint capsules of five lumbar motion segments. Considerable variability was observed among specimens.

Load deformation data showed that, in response to 30.2 N loads applied in anterior, posterior, or lateral shear, or in tension or compression, the mean displacements of the inferior facet joint centers of the superior vertebral body ranged from 0.5 to 1.8 mm (111).

Disc Versus Facet Weightbearing Proportion

Results of a study (112) of six lumbar segments revealed that the normal facets carried 3 to 25% of the weightbearing load. If the facet joint was arthritic, the load could be as high as 47%. The transmission of compressive facet load occurs through contact of the tip of the inferior facet with the pars of the vertebra below. The data also show that an overloaded facet joint will cause rearward rotation of the inferior facet, resulting in the stretching of the joint capsule. The finite element model predicted an increase in facet load caused by a decrease in disc height. The following hypothesis is proposed: Excessive facet loads stretch the joint capsule, and they can be a cause for low back pain (112).

The disc carries an average of 91% of the load in lifting 200 pounds, and the facet joint carries no more than 12%. Low facet joints put more weight on the disc, whereas high facet joints put less weight on the disc. *The facets carry little weight in compression but accept great amounts in bending* (106).

Articular Facets Carry More Weight Than Knee Joints

The pedicle-facet complex normally carries only 20% of the vertical pressure applied at the interspace (113). This constitutes ten times the weight per square inch applied to the knee joints (114). As the disc loses turgor and resilience, it also loses its ability to resist compressive forces and to maintain normal intervertebral separation and alignment. This throws an additional burden on the facet articulations and may accelerate the changes of degenerative arthrosis (115).

A comparison of segments L2–L3 and L4–L5 at different axial loads in neutral mode shows that the facets at L2–L3 generally take more load than those at L4–L5. The same trend is also observed for the extension mode at both the L2–L3 and L4–L5 levels. Furthermore, in extension the normal load at the facets is always higher than in the neutral mode. This holds for both the L2–L3 and L4–L5 levels. This indicates that facet pressure rather than the facet load may be playing a significant role in the degenerative changes of facets (107).

Effect of Degenerative Disc Disease on Weightbearing

The intervertebral disc is of major importance in painful conditions of the spine. An injury to the disc can affect overall spinal mechanics—both the behavior of the disc itself and that of other spinal structures. For example, injury can lead to altered sharing of the load between the disc and the apophyseal joints.

The two load-bearing components of the disc are (*a*) the nucleus, in the central region, which is surrounded by (*b*) the anulus fibrosus, consisting of fibrous tissue in concentric laminated bands. The nucleus is generally under compressive stress, whereas the anular layers, especially the outer layers, carry tensile stresses. The stresses in the two components balance each other as well as the load carried by the disc. A disturbance in any one component of the disc (e.g., a decrease in the water content of the nucleus or an injury to the anulus) may be thought to affect the mechanical behavior of the other component as well as that of the disc as a whole (116).

The stages of injury to the functional spinal unit (FSU) are:

1. Asymmetric disc injury at one FSU.
2. Disturbed kinematics of FSUs above and below injury.

3. Asymmetric movements at the facet joints.
4. Unequal sharing of facet loads.
5. High load on one facet joint.
6. Cartilage degeneration, and/or facet atrophy and narrowing of the intervertebral foramen (116).

Stages of Disc Prolapse

Fifty-two cadaveric lumbar motion segments were subjected to fatigue loading in compression and bending to determine if the intervertebral discs could prolapse in a gradual manner (Fig. 2.17). Prior to testing, the nucleus pulposus of each disc was stained with a small quantity of blue dye and radiopaque solution. This enabled the progress of any gradual prolapse to be monitored by direct observation and by discogram. Six discs developed a gradual prolapse during the testing period. The injury starts with the lamellae of the anulus being distorted to form radial fissures; then, nuclear pulp extrudes from the disc and leaks into the spinal canal. The discs most commonly affected were from the lower lumbar spine of young cadavers. Tests on 10 older discs with pre-existing ruptures showed that these discs were stable and did not leak nuclear pulp (117).

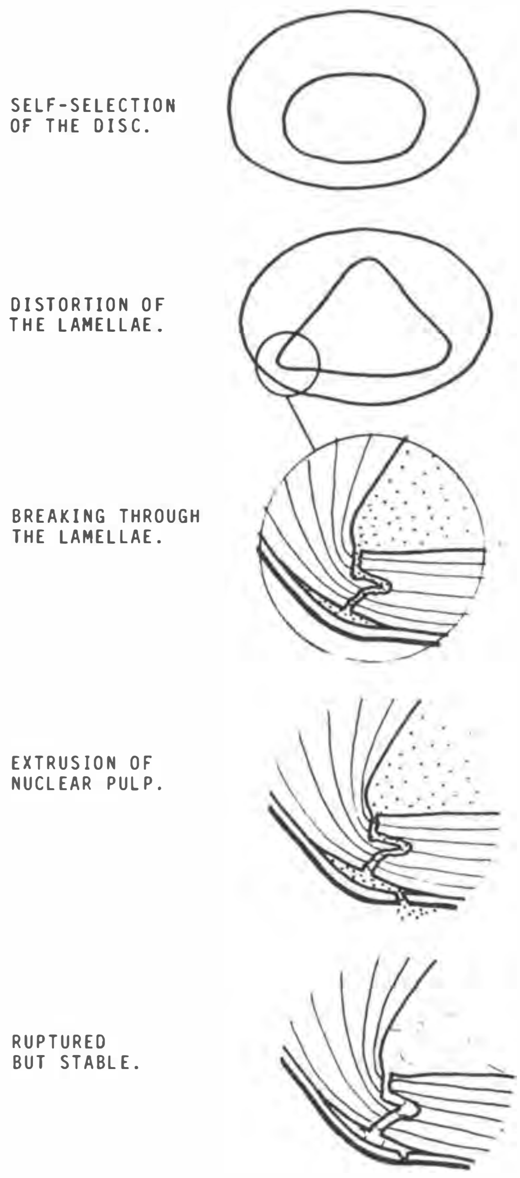

Figure 2.17. The five stages of gradual disc prolapse. (Reprinted with permission from Adams MA, Hutton WC. Gradual disc prolapse. Spine 1985;10(6):530.)

Table 2.1

Average Peak Pressures of 24 Joints

Posture	Peak Pressure (kg/cm²)	Disc Height	Peak Pressure (kg/cm²)
Flexion 4°	56.8	Unaltered	51.6
Neutral	63.9	Loss of 1 mm	70.1
Extension			
4°	72.8	Loss of 4 mm	83.3
6°	79.4		

Cadaveric lumbar spine specimens of "motion segments," each including two vertebrae and the linking disc and facet joints, were compressed. The pressure across the facet joints was measured using interposed pressure-recording paper. This was repeated for 12 pairs of facet joints at four angles of posture and with three different disc heights. The results showed that pressure between the facets increased significantly with narrowing of the disc space and with increasing angles of extension (Table 2.1). Extra-articular impingement was found to be caused, or worsened, by disc space narrowing. Increased pressure or impingement may be a source of pain in patients with reduced disc spaces (118).

Nerve Root Compression Changes in Disc Degeneration

An instrumented probe mounted on the anterior surface of the lumbar spine over an excised lumbar intervertebral disc was used to simulate a disc protrusion in 12 fresh cadavers. The contact force between probe and nerve root was measured as a function of two independent variables: probe protrusion depth and disc space height.

The force produced by the probe on the nerve root progressively increased as the probe was advanced against the nerve root because of the tension produced in the nerve root. The anatomic fixation of the nerve root within the neural canal, both proximal and distal to the intervertebral disc, appears to play an important role in this regard.

Narrowing the disc space significantly decreased the force on the nerve root for a given probe protrusion (119).

Pain Production in the Facet with Disc Degeneration

Disc space narrowing causes a marked increase in peak pressure between opposed facets in the zygapophysial joints. An associ-

ation is known to exist between osteoarthrosis of the zygapophysial joints and osteophytic lipping of vertebral bodies. However, osteoarthrosis of the zygapophysial joints need not always occur at the same level as intervertebral disc degeneration (120). Harrison et al. (121) found vascular profusion accompanying degenerative changes in the hip joint. According to Arnoldi (122), intraosseous hypertension may be a factor of importance in the pathomechanics of certain types of low back pain, and it is well known that pain of vascular origin is a recognized clinical phenomenon. Giles and Taylor's (123) study describes vascularization of a zygapophysial articular cartilage in minor osteoarthrosis (Figs. 2.18 and 2.19). This vascularization presupposes innervations of these blood vessels by vasomotor nerves.

A survey of the literature concerning zygapophysial joint osteoarthrosis of the lumbar spine has not revealed a description of an extensive vascular supply to cartilage showing minor osteoarthrosis, as has been demonstrated in Giles and Taylor's study. The vascular supply shown in their study may well be indicative of an attempt at cartilage repair, and this repair need not be limited to the periphery of the joint. Vascularization may indicate that low back pain of vascular origin could be experienced by patients with minor osteoarthrosis (123).

Degenerative Changes of Vertebral Plates

Degenerative change at the end plate of the discovertebral joint was studied in the elderly adult by correlating the histologic and radiographic findings. Undecalcified ground sections were made from 21 autopsied lumbar spines that demonstrated no evidence of disease except age-related osteoporosis. Histologic examination (Figs. 2.20–2.24) showed that the cartilaginous end plates were degenerated to various extent and were replaced by subchondral bone proliferation (endochondral bone formation) in the direction of the joint space. In advanced cases, this histologic finding was reflected in radiographs as a subchondral sclerotic zone protruding toward the disc space. The degree of end plate change was positively correlated with disc space narrowing and the vacuum phenomenon (degeneration of the nucleus pulposus), but not with osteoporosis and vertebral compression. Anatomically and functionally, this may be the most common form of degeneration at the discovertebral joint end plate. Further study is necessary to clarify the process (124).

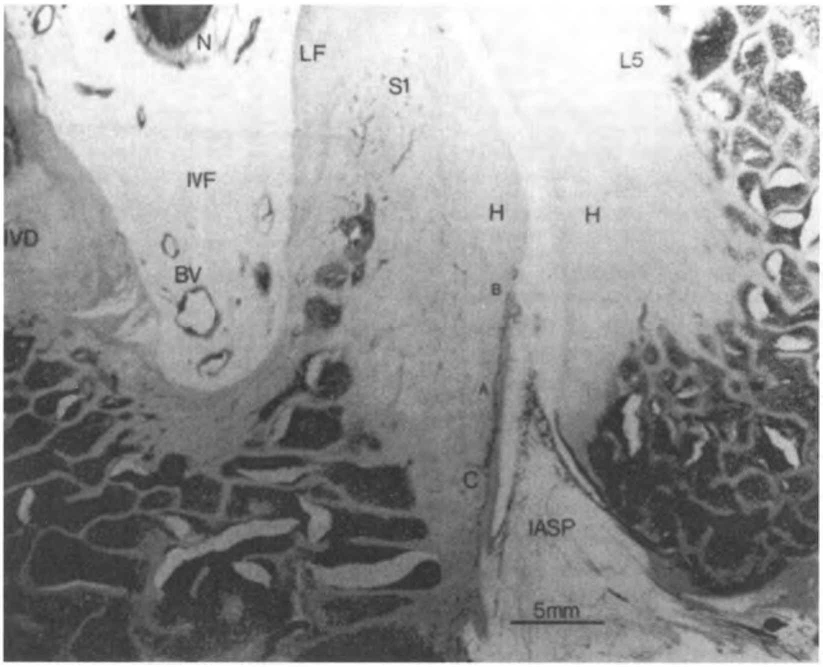

Figure 2.18. A sagittally cut histologic section from the medial one third of the right zygapophysial joint of a cadaver. Note the blood vessel extending from the subchondral bone of the superior articular process of the sacrum into the articular cartilage, which shows minor osteoarthrosis. Note that this sagittal section reveals the anatomy of the inferomedial intra-articular synovial inclusion that projects into the wide opening of the inferior joint recess. The intra-articular synovial inclusion is a highly vascular adipose structure with a synovial membrane lining. *BV*, blood vessels; *C*, capillary—parts A and B; *H*, hyaline articular cartilage; *IASP*, intra-articular synovial inclusion; *IVD*, intervertebral disc at the lumbosacral joint; *IVF*, intervertebral foramen; *LF*, ligamentum flavum; *L5*, inferior articular process of the fifth lumbar vertebra; *N*, nerve; *S1*, superior articular process of the first sacral segment. (Reprinted with permission from Giles LGF, Taylor JR. Osteoarthrosis in human cadaveric lumbosacral zygapophysial joints. J Manipulative Physiol Ther 1985;8(4):241–242. Copyright 1985, the National College of Chiropractic.)

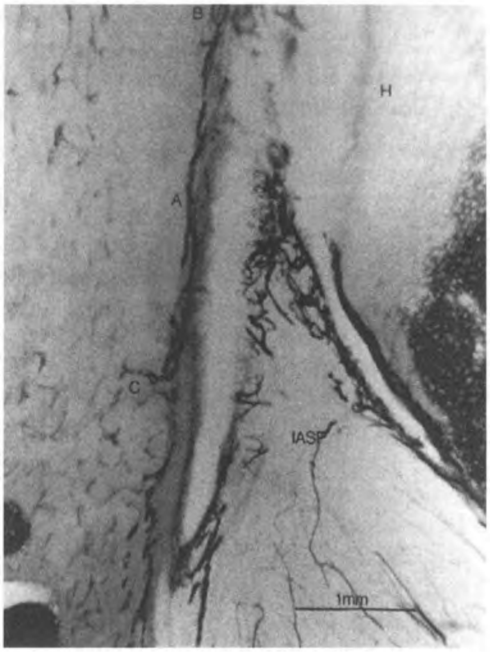

Figure 2.19. This figure represents magnification of the blood vessel shown in Figure 2.18. C, capillary—parts A and B. *H*, hyaline articular cartilage; *IASP*, intra-articular synovial protrusion. (Reprinted with permission from Giles LGF, Taylor JR. Osteoarthrosis in human cadaveric lumbo-sacral zygapophysial joints. J Manipulative Physiol Ther 1985;8(4):241–242. Copyright 1985, National College of Chiropractic.)

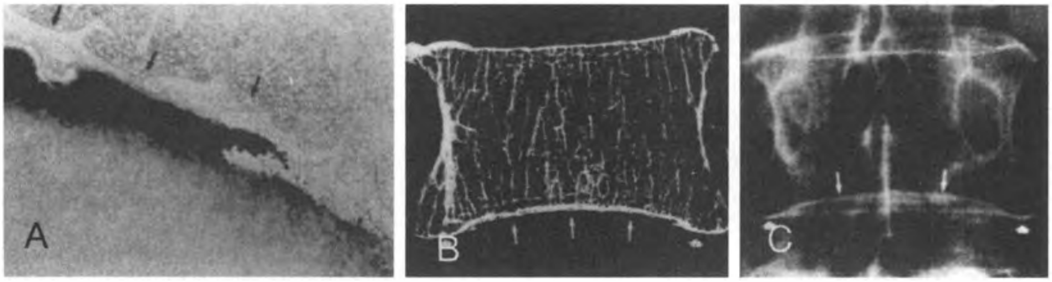

Figure 2.20. Inferior surface of L3 in a 75-year-old man. **A.** Histologic section (undecalcified, Villanueva bone staining, original magnification ×10). Cartilaginous end plate is sufficiently retained (*open arrows*). Note small protrusion of subchondral bone (*arrowhead*). **B.** Low-kilovoltage contact radiograph. **C.** Clinical radiograph. Appearance of the bone end plate (*thin arrows*) and epiphyseal ring (*thick arrows*) is almost normal. (Reprinted with permission from Aoki J, et al. End plate of the discovertebral joint: degenerative change in the elderly adult. Radiology 1987;164(2):412.)

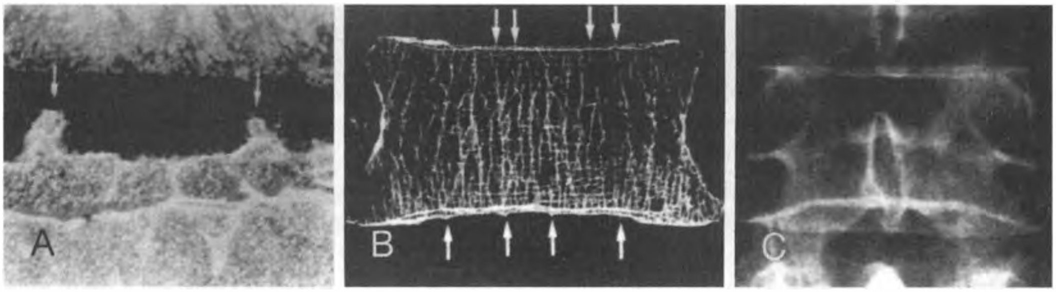

Figure 2.21. Superior surface of L4 in an 82-year-old man. **A** and **B.** Small projections of subchondral bone into the cartilaginous end plate can be observed (*arrows*). **C.** Radiograph shows completely normal appearance of the end plate. (Reprinted with permission from Aoki J, et al. End plate of the discovertebral joint: degenerative change in the elderly adult. Radiology 1987;164(2):412.)

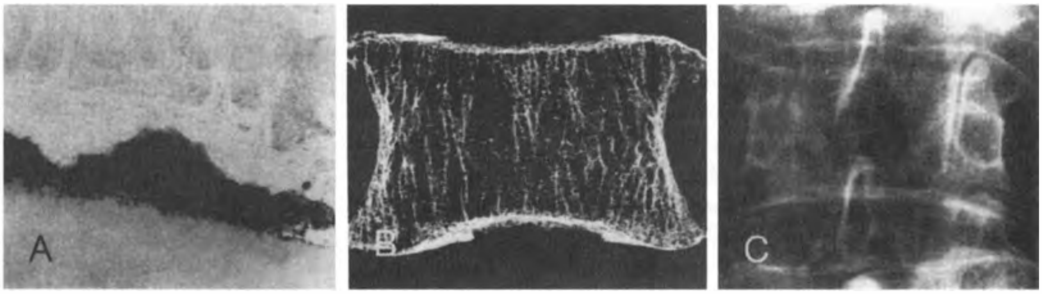

Figure 2.22. Inferior surface of L4 in a 72-year-old woman. **A.** Cartilaginous end plate is replaced by subchondral bone proliferation. Border between the cartilaginous end plate and fibrous cartilage is flat, whereas the cartilage-bone border is undulatory. **B.** Newly formed subchondral bone makes a thin sclerotic zone. **C.** Radiograph fails to reflect the histologic finding, because the direction of the x-ray beam is not appropriate. (Reprinted with permission from Aoki J, Yamamoto I, Kitamura N, et al. End plate of the discovertebral joint: degenerative change in the elderly adult. Radiology 1987;164(2):413.)

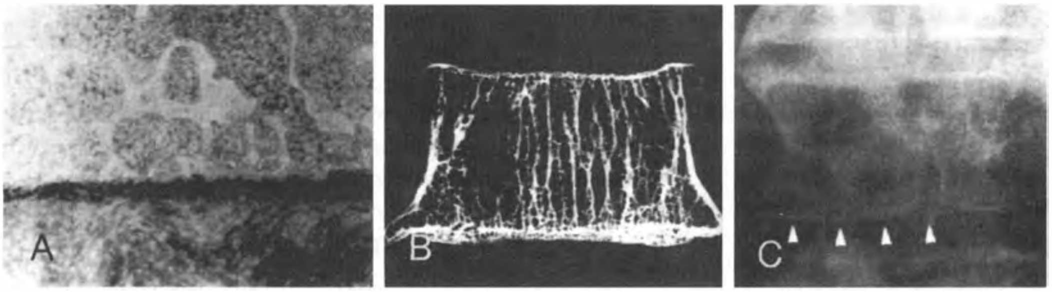

Figure 2.23. Inferior surface of L1 in an 82-year-old woman. **A.** Thickness of the cartilaginous end plate is markedly reduced. **B.** Newly formed subchondral trabeculae are fine and intimate, forming a protrusive thin sclerotic zone. **C.** Radiograph reflects the histologic changes well (*arrowheads*). (Reprinted with permission from Aoki J, Yamamoto I, Kitamura N, et al. End plate of the discovertebral joint: degenerative change in the elderly adult. Radiology 1987;164(2):413.)

Figure 2.24. Inferior surface of L1 in a 75-year-old woman. **A.** Cartilaginous end plate is completely lost, and the surface of subchondral bone is crushed. Small herniated cartilaginous nodes can be observed (*arrows*). **B.** Texture of end plate zone is different from that of the triangular area of thickened pre-existing trabeculae. **C.** Both triangular sclerotic area and protrusive subchondral sclerotic zone can be observed on the radiograph. (Reprinted with permission from Aoki J, et al. End plate of the discovertebral joint: degenerative change in the elderly adult. Radiology 1987;164(2):413.)

Treatment Effects on Disc and Facet Articulations

Epidural Steroid Injection

Epidural anesthetics and steroids have been widely used for more than 20 years in the treatment of low back pain and pseudoradicular or radicular pain.

Seven women and nine men, aged 27 to 59 years (mean, 45 years) with lumbar pain and sciatica had epidural blocks once with 80 mg of methylprednisolone acetate and lidocaine in individual doses. By means of a visual analogue scale, 10 of these patients (62%) reported relief of half the pain the following day. One month later, only seven patients (43%) reported relief of one third of the pain. Only one patient benefited ultimately (after 6 months). In the remaining patients, pain was unaffected by the epidural injection (125). I have found this procedure of limited benefit to low back pain patients.

Posture Effects on Lumbar Spine

A series of experiments showing how posture affects the lumbar spine is reviewed. Postures that flatten (i.e., flex) the lumbar spine are compared with those that preserve the lumbar lordosis. Flexed postures have several advantages: flexion improves the transport of metabolites in the intervertebral discs, reduces the stresses on the apophyseal joints and on the posterior half of the anulus fibrosus, and gives the spine a high compressive strength. Flexion also has disadvantages: it increases both the stress on the anterior anulus and the hydrostatic pressure in the nucleus pulposus at low load levels.

The disadvantages are not of much significance, and conclude that it is mechanically and nutritionally advantageous to flatten the lumbar spine when sitting and when lifting heavy weights (126).

On the basis of posture, humans can be divided into squatters and nonsquatters. A comparative study of the two groups is as follows:

1. On the basis of radiographic studies, the incidence of degenerative change in the intervertebral disc in primitive squatting populations is considerably less than that found in civilized peoples.
2. The suggestion is made that lordosis is implicated in the pathogenesis of degeneration, but further studies are required (127).

Intra-Abdominal Pressure Effects on Spinal Unloading

The ability of a partial or full Valsalva maneuver (voluntary pressurization of the intra-abdominal cavity) to unload the spine was investigated in four subjects. During the performance of five isometric tasks, intra-abdominal and intradiscal pressures and surface myoelectric activities in three lumbar trunk muscle groups were measured. The tasks were carried out first without voluntary pressurization of the intra-abdominal cavity and then when the subjects performed partial and full Valsalva maneuvers. A biomechanical model analysis of each task was made to help interpret the experimental measurements. Intra-abdominal pressure was found not to be an indicator of spine load in these experiments. The Valsalva maneuvers did raise intra-abdominal pressure, but in four of the five tasks it increased rather than decreased lumbar spine compression (128).

Apophyseal Joint Resistance to Compression

Cadaveric lumbar intervertebral joints were loaded to simulate the erect standing posture (lordosis) and the erect sitting posture (slightly flexed). The results show that, after the intervertebral disc has been reduced in height by a period of sustained loading, the apophyseal joints resist about 16% of the intervertebral compressive forces in the erect standing posture, whereas in the erect sitting posture they resist none. The implications of this in relationship to degenerative changes and to low backache are discussed below.

Compression forces of up to 11 times the superincumbent body weight can be imposed on the lumbar spine by daily activities. If the aim is to reduce the compressive forces on the disc, some degree of lordosis is needed. This posture, however, in addition to loading the apophyseal joints, places high compressive loads on the posterior anulus, which is the focus of degenerative changes. It has indeed been suggested that the Western lordotic posture promotes intervertebral disc degeneration. Slight flexion, on the other hand, has the advantage of relieving both the apophyseal joints and the posterior anulus of compressive force (129).

Routine daily activities seldom impose large loads on the spine in shear, bending, or torsion. In bending and torsion, in particular, the trunk muscles rather than the motion segments usually balance moments. This occurs because few physical activities require lumbar motion segments to flex, extend, bend laterally, or twist more than a few degrees. Few physical activities involve significant motions in shear. In response to only small motions, the motion segments can develop only small moment and shear resistances (130).

Trunk Length in Low Back Pain

Of 446 pupils aged 13 to 17 years, 115 were found to have a history of back pain. These pupils tended to have decreased lower limb joint mobility and increased trunk length compared with pupils without back pain. In 77 pupils whose site of back pain was identified, 38 had pain associated with the lumbar spine. These pupils had an increased trunk length, whereas those with thoracolumbar or thoracic pain did not. Back pain was more common in those who avoided sports (131).

Diurnal Stress Variations on Lumbar Spine

Forward bending movements subject the lumbar spine to higher bending stresses in the early morning compared with

later in the day. The increase is about 300% for the discs and 80% for the ligaments of the neural arch. It is concluded that lumbar discs and ligaments are at greater risk of injury in the early morning (132).

TROPISM

A variance of opinion is found in the literature on the subject of normal facings of the lumbar articular facets. Some investigators believe that sagittal facings are normal, whereas others believe that coronal facings are normal. In our clinical study (133) of patients with vertebral disc lesions, we recorded which facet findings were involved at all lumbar levels. We believe that this is the first controlled study documented in the chiropractic and, perhaps, the medical literature concerning which facet facings are involved in lumbar disc lesions. It must be stressed that these findings are based on radiographs of patients with disc protrusion or prolapse.

Tropism (from the Greek word *trope,* a turning) refers to an anomaly of articular formation in which the two articular facings are not the same (i.e., instead of both being sagittal or both coronal, each side assumes a different facing), as shown in Figure 2.25.

From Tables 2.2 and 2.3, it can be inferred that sagittal facet facings are typical in the upper lumbar spine, whereas coronal facet findings are typical in the lower lumbar spine. In 18 of 56 cases of disc lesion (32%), anomalies of articular tropism were present. The most difficult cases to treat were those involving the sagittal facet facings at the level of discal protrusion or prolapse, especially when a medial disc was involved.

The directional plane of articulation of the facets allows for specific movement. Sagittal facets flex and extend, whereas coronal facets bend laterally. The combining of these two directional opposing forces places excessive stress on the anular fibers of the intervertebral disc, which tear in nuclear protrusion. The axis of rotation of a lumbar vertebral unit is between the articular facets, with the body rotating forward of this axis (134). Therefore, the altered motoricity of a sagittal and coronal combination creates stress on both the disc and articular facets in all motions of the lumbar spine.

Facet tropism, therefore, creates stress on the lumbar spine during motion. In this situation, rotation takes on added importance, because it places maximal stress on the anular fibers, which must tear for the nucleus pulposus to protrude, creating the typical disc syndrome with sciatica.

According to Farfan et al. (135), the IVD is capable of great compressive loads. They also believe that Schmorl and Beadle were inaccurate when they stated that the compressive load was the mechanical basis of disc degeneration.

By application of torsional loading to 90 IVD joints (proved normal by discogram) from 66 necropsy specimens, 22.6° was the amount of rotation needed to cause failure of the normal disc; in cases of degenerated disc, the angle of failure was 14.3°. Degenerated discs show a consistently smaller torsional angle of failure. Farfan et al. (135) concluded that the IVD is injured by rotation within a small normal range of movement and that disc protrusion is a manifestation of anular tearing by torsional injury.

According to Cailliet (136), 75% of lumbar flexion occurs at the lumbosacral articulation. He further states that the shearing stress of the fifth lumbar vertebra on the sacrum increases proportionately to the anterior angulation of the sacrum. We have applied these ideas on stress to our knowledge of the facet articular plane and believe that the coronal facet facing at L5–S1 allows greater stability than does the sagittal facet facing at L5–S1. We believe, therefore, that the following conclusions are justified.

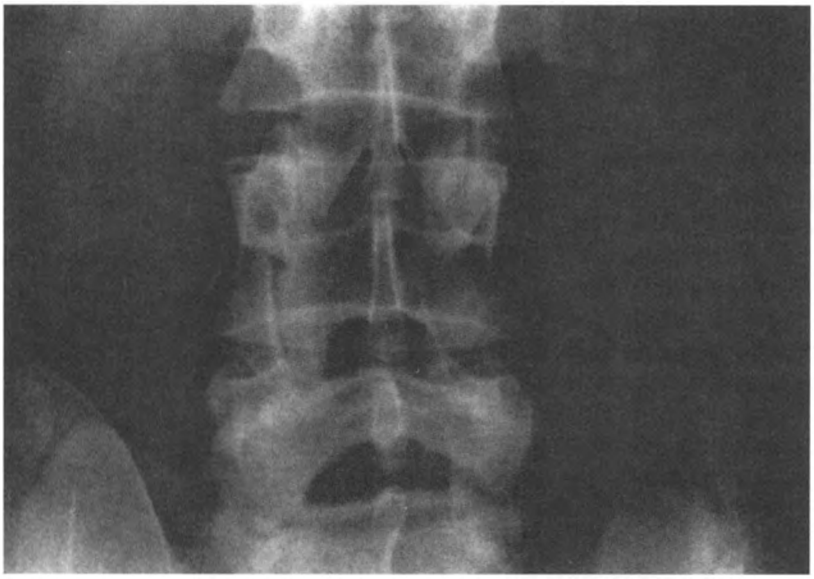

Figure 2.25. X-ray study reveals tropism of the articular facets, with the right L4–L5 facet facings being coronal and the left being sagittal. Note that the facet facings at L5–S1 are bilaterally coronal.

Table 2.2

Percentage of Facet Facings in 56 Cases of Lumbar Disc Lesion, by Location and Position

	L1–L2		L2–L3		L3–L4		L4–L5		L5–S1	
	Right	Left	Right	Left	Right	Left	Right	Left	Right	Left
Sagittal	74	72	55	64	47	43	29	29	7	5
Coronal	23	26	40	34	41	53	64	65	91	95
Semisagittal	3	2	5	2	12	4	7	6	2	

1. Sagittal facet articulation facings are normal for the upper lumbar spine, and coronal facet articulation facings are normal for the lower lumbar spine.
2. Even with the fewer numbers of sagittal facings in the lower lumbar spine, tropism occurred at the level of disc lesion in 32% of the cases; therefore, a prominence of disc lesions is found in cases of sagittal facings and of tropism.
3. Rotation is the most damaging motion of the low back, resulting in tearing of the lumbar disc anular fibers, which allows for nuclear protrusion.
4. Sagittal facets or anomalies of tropism create additional stress on the spine during rotation. Rotation in this situation may be much less than normal before anular disc fibers tear.
5. Patients with anomalous facet facings are at high risk for developing a disc lesion on rotation.
6. In one of every five patients, an asymmetric orientation is seen of the spinal articular facets at a single level and abnormal spinal motion; these patients, therefore, are predisposed to develop low back and sciatic pain syndromes (115).
7. In patients with articular tropism, the joints rotate toward the side of the more oblique facet (137). Figures 2.26 and 2.27 reveal how tropism changes the force distribution and applies additional torsion to the disc. Furthermore, tropism may predispose to degenerative arthrosis at these facets.
8. Finally, articular tropism or asymmetry of the articular facets can lead to the manifestation of lumbar instability as joint rotation. This rotation occurs toward the side of the more oblique facet, and it can place additional stress on the anulus fibrosus of the intervertebral disc and capsular ligaments of the apophyseal joints.

Because the posterior elements maintain stability of the spine, they play an important role in the triple joint complex of the facets and disc. Tropism occurs most commonly in the two lowest lumbar levels (138, 139). Keep in mind that these are synovial joints, and shearing forces place compression on facet surfaces. This compression is greater in less obliquely facing facets. Less oblique facets have greater interfacet forces, predisposing them to degenerative forces.

Arthrosis of the facets is rare in patients under age 30, and it is found progressively more frequently and is more severe as these patients age (140). Also, intervertebral arthritis is more common at L3–L4 and L4–L5 than at L5–S1, where the facets

Table 2.3

Average Percentage of Facet Facings at Each Level in 56 Cases of Lumbar Disc Lesion

	L1–L2	L2–L3	L3–L4	L4–L5	L5–S1
Sagittal	73	59.5	45	29	6
Coronal	24.5	37	47	64.5	93
Semisagittal	2.5	3.5	8	6.5	1

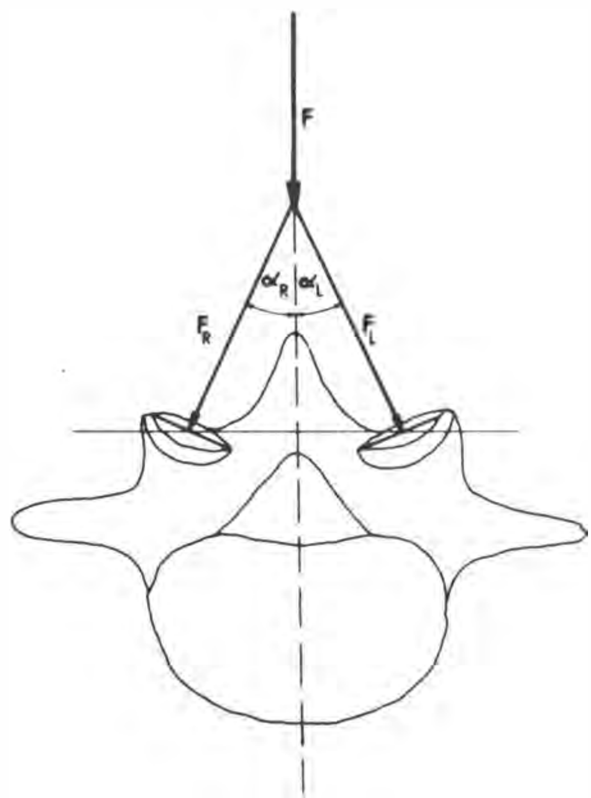

Figure 2.26. Forces (*F*) acting on symmetrically oriented superior articular facets. (Reprinted with permission from Cyron BM, Hutton WC. Articular tropism. Spine 1980;5(2):170.)

are less obliquely faced. Badgley (138) reports that arthritis of the facets is more common in cases of tropism and lesions of articular capsules, granular ossification, calcification, and adhesions of the meningeal covering of the nerve root adjacent to it.

The normal plane of articulation of the lower lumbar facets (Fig. 2.28) is 45° to the body sagittal or coronal planes (141). The inferior facets are convex, whereas the superior facets are concave.

Figure 2.29 shows the normal 45° angle of inclination (136). Figure 2.26 shows that the vector forces are equally balanced on the two facets in the case of a symmetrically oriented articular facet, whereas Figure 2.27 shows that the forces shift to the side of the more obliquely faced facet in the case of tropism. It is on the side of the more obliquely faced facet that the posterolateral anular fibers tear.

Thoracolumbar Facet Orientation

Disc degeneration in the thoracolumbar junctional region (T10–L1) of 37 male cadaveric spines was recorded by discography. From 24 of these spines, the facet joint orientation and degenerative findings of the facet, costovertebral joints, vertebral bodies (osteophytosis) and discs, and Schmorl's nodes were recorded directly from bones. At T11–T12, the most common site for the transitional zone between thoracic and lumbar facet type, a marked variation was seen in the orientation of facets (Fig. 2.30). The occurrence of degenerative findings and Schmorl's nodes at the three levels in the region differed (Figs. 2.31–2.33). At T10–T11, disc degeneration, vertebral body osteophytosis, and Schmorl's nodes were most common (anterior degeneration). At T12–L1, facet and costovertebral joint degeneration was dominant (posterior degeneration). At T11–T12, disc degeneration, vertebral body osteophytosis, Schmorl's nodes, and facet and costovertebral joint degeneration all occurred (anterior and posterior degeneration). The results point to a pathoanatomic association between degenerative changes and facet orientation (142).

Facet Facings Compared in Upper and Lower Lumbar Spine

The relationship between the angulation of the facet joints and that of the caudad parts of the corresponding laminae in the transverse plane was investigated with computed tomography (CT) at the vertebral levels L3–L4, L4–L5, and L5–S1 (Fig. 2.34). At the level of L3–L4, both the facet joints and the caudad portions of the laminae tend toward a sagittal orientation, whereas at L5–S1, the orientation is more toward the frontal plane; at the level of L4–L5, they occupy an intermediate position. A highly significant correlation between the orientation of these structures is demonstrated. The caudad parts of the laminae may be considered buttresses for the inferior articular processes of the same vertebra (143).

Sagittal Facets Promote Disc Prolapse

In the synergistic complex formed by the intervertebral disc and posterior articular processes, the latter play a significant

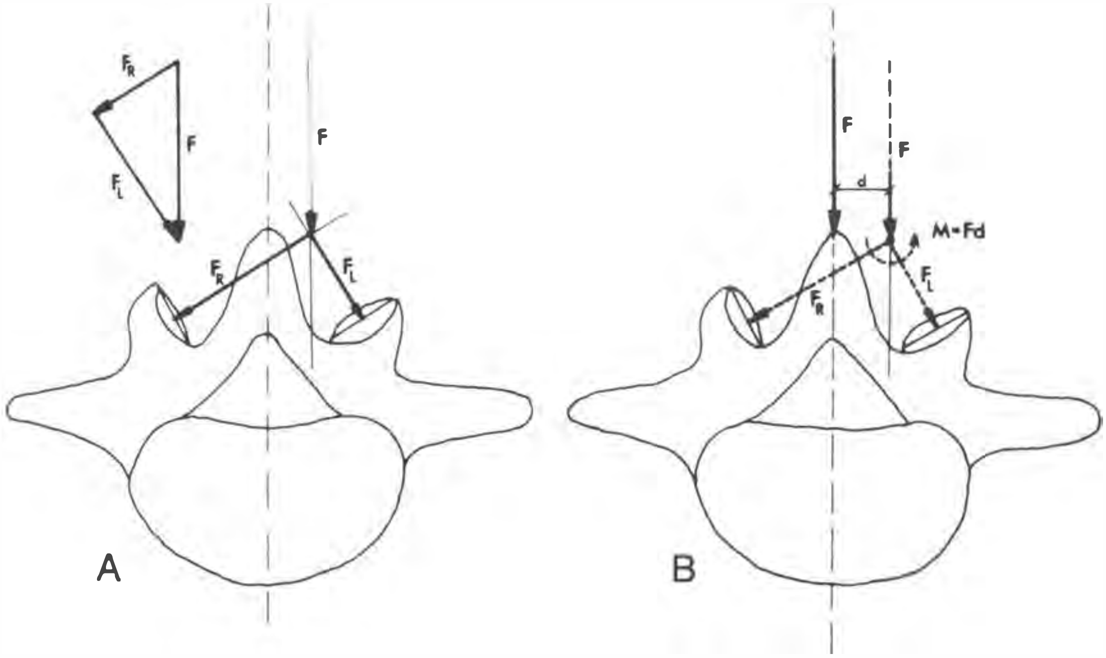

Figure 2.27. Forces (*F*) acting on asymmetrically oriented superior articular facets. **A.** The force F acts at the point of concurrence, and it is distributed unevenly to the articular facets. **B.** The force is offset from the point of concurrence, and additional torsion is applied to the joint. (Reprinted with permission from Cyron BM, Hutton WC. Articular tropism. Spine 1980;5(2):171.)

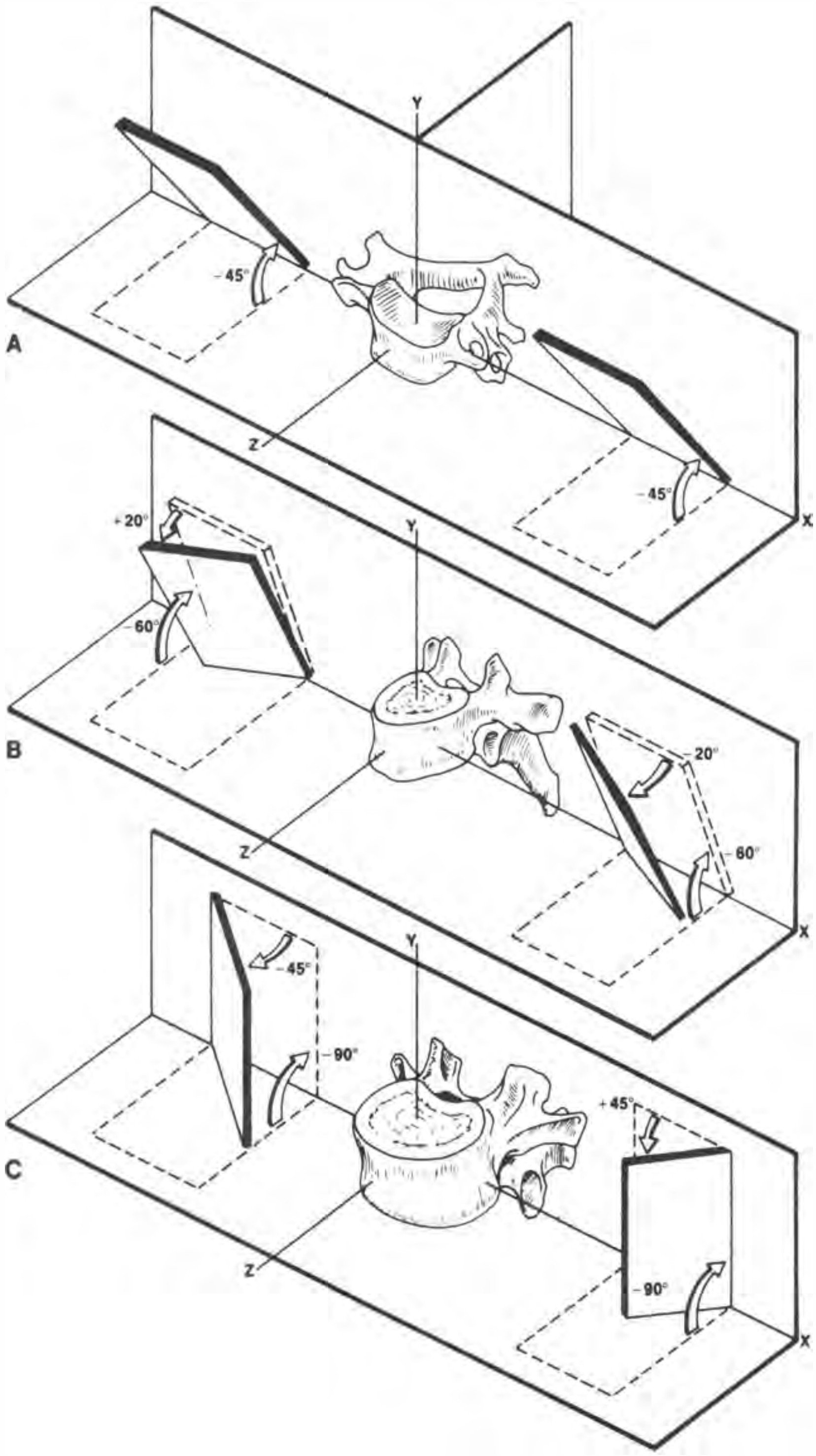

Figure 2.28. Orientation of the facet joints. A graphic representation of the facet joint inclinations in various regions of the spine is obtained by rotating two cards lying in the horizontal plane through two consecutive angles (i.e., x axis rotation followed by y axis rotation). Typical values for the two angles for the three regions of the spine follow. **A.** Cervical spine: $-45°$ followed by $0°$. **B.** Thoracic spine: $-60°$ followed by $+20°$ for right facet rotation, or $-20°$ for left facet rotation. **C.** Lumbar spine: $-90°$ and $-45°$ for right facet rotation or $+45°$ for the left facet rotation. (These are only rough estimates.) Variations are found within the regions of the spine and between different individuals. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:22.)

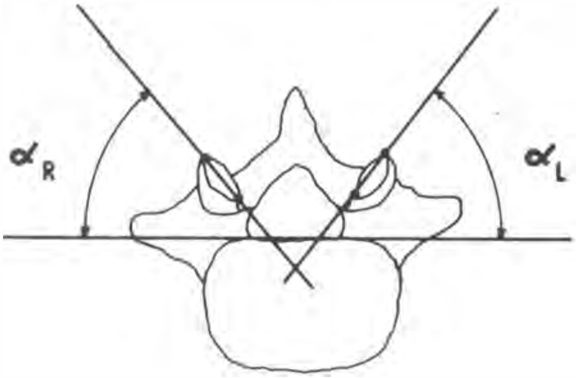

Figure 2.29. Measurement of facet orientation. (Reprinted with permission from Cyron BM, Hutton WC. Articular tropism. Spine 1980; 5(2):170.)

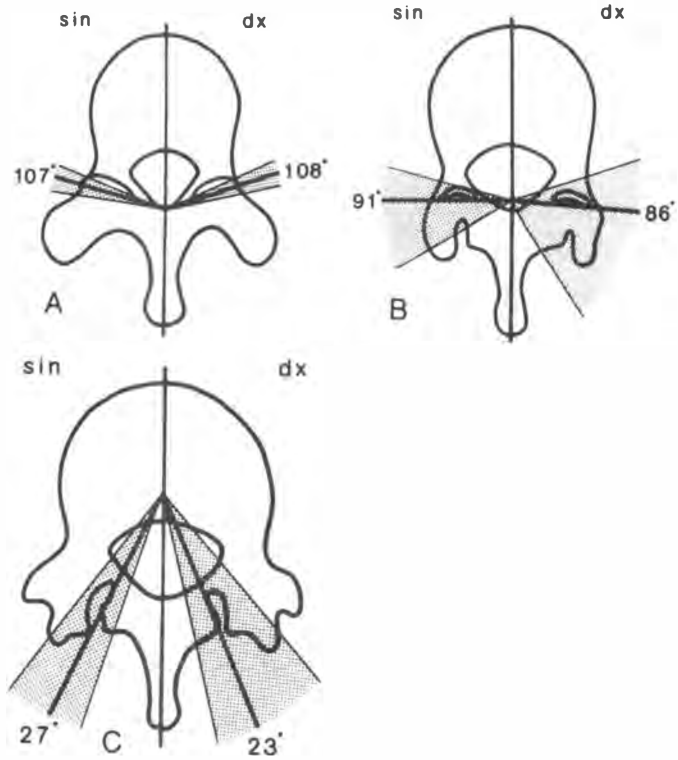

Figure 2.30. Median and 10th and 9th percentiles (*shaded area*) of facet joint angles of 24 cadaveric spines at **A,** T10–T11; **B,** T11–T12; and **C,** T12–L1. At T10–T11, facet orientation was always nearly frontal. At T11–T12, the facet angles showed widest variation. At T12-L1, facet orientation was usually of lumbar type (i.e., nearly sagittal). (Reprinted with permission from Malmivaara A, Videman T, Kuosma E, et al. Facet joint orientation, facet and costovertebral joint osteoarthrosis, disc degeneration, vertebral body osteophytosis, and Schmorl's nodes in the thoracolumbar junctional region of cadaveric spines. Spine 1987;12(5):460, 461.)

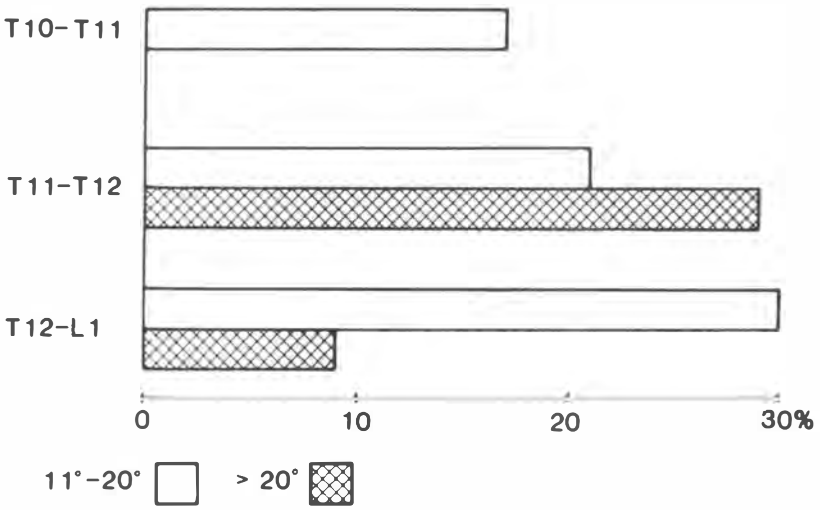

Figure 2.31. Asymmetry of the facet joints at different levels of the thoracolumbar junctional region (T10–L1) of 24 cadaveric spines. Asymmetry of greater than 20° was most common at T11–T12. (Reprinted with permission from Malmivaara A, Videman T, Kuosma E, et al. Facet joint orientation, facet and costovertebral joint osteoarthrosis, disc degeneration, vertebral body osteophytosis, and Schmorl's nodes in the thoracolumbar junctional region of cadaveric spines. Spine 1987;12(5):460, 461.)

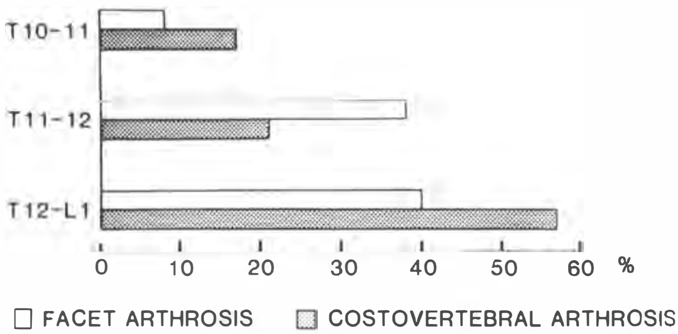

Figure 2.32. Percentages of slight to severe facet joint and costovertebral joint osteoarthrosis (posterior degeneration) at different levels of the T–L region (T10–L1) in 24 cadaveric spines. Assessments from bone specimens. Posterior degeneration was most common at T12–L1. (Reprinted with permission from Malmivaara A, Videman T, Kuosma E, et al. Facet joint orientation, facet and costovertebral joint osteoarthrosis, disc degeneration, vertebral body osteophytosis, and Schmorl's nodes in the thoracolumbar junctional region of cadaveric spines. Spine 1987;12(5):460, 461.)

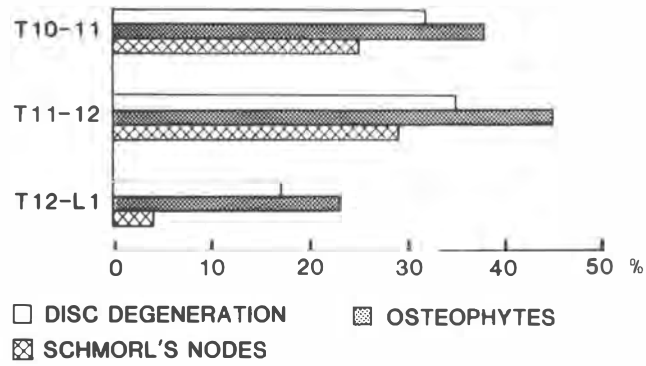

Figure 2.33. Percentages of moderate to severe general disc degeneration, vertebral body osteophytosis, and Schmorl's nodes (anterior degeneration) at different levels of the T–L region (T10–L1) in 24 cadaveric spines (disc degeneration in 37). Anterior degeneration was least common at T12–L1. (Reprinted with permission from Malmivaara A, Videman T, Kuosma E, t al. Facet joint orientation, facet and costovertebral joint osteoarthrosis, disc degeneration, vertebral body osteophytosis, and Schmorl's nodes in the thoracolumbar junctional region of cadaveric spines. Spine 1987;12(5):460, 461.)

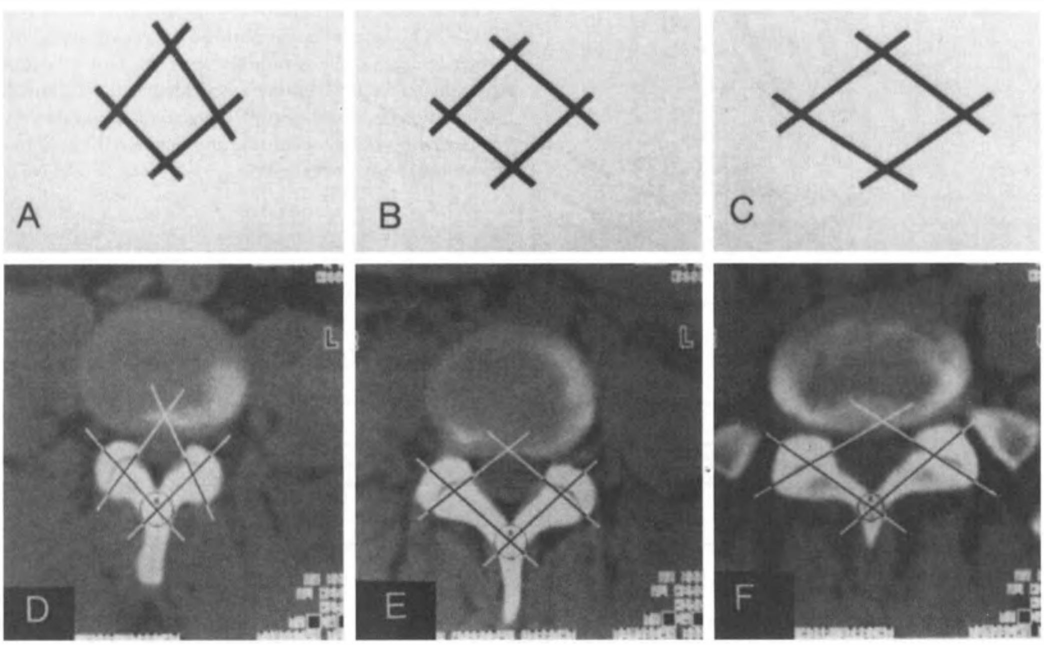

Figure 2.34. **A–C.** Quadrangles composed of the mean values for transverse interfacet-joint and interlaminar angles at L3–L4, L4–L5, and L5–S1, respectively. **D–F.** CT scans of each of the three levels show the formation of the quadrangles. They represent individual values, not the mean values shown in **A–C.** (Reprinted with permission from Van Schaik JPJ, Herbiest H, Van Schaik FDJ. The orientation of laminae and facet joints in the lower lumbar spine. Spine 1985;10(1):63.)

role in protecting the disc and blocking forward movement of the spine. This role is of special importance at the level of the lumbosacral interface, whose inclination contributes to increasing the shearing forces acting on the disc. The orientation of the lumbosacral articular processes modifies the distribution of the mechanical stress acting at their level. The relationship between the orientation of the articular processes and the stress transmitted to the disc was studied by CT (31 subjects without disc prolapse, 35 subjects with disc prolapse, 110 operative reports). Sagittal orientation of the facet joints, which is consistently more pronounced on the right side, seems to promote disc prolapse occurrence at the lumbosacral level (144).

Controversy Exists Over Facet Symmetry in Disc Degeneration

No useful correlations were found between facet and canal asymmetry, canal rotation, or degenerative change. Coronally oriented facets withstand shear but do not resist rotation. No greater incidence of degenerative change in vertebrae was seen with coronally oriented facets. The role of asymmetric apophyseal joints was discussed by Farfan et al. (135). In this study, however, no correlation was found between the degree of facet asymmetry and the size of the vertebral osteophytes.

If facet asymmetry predisposed an individual to rotational displacement, either it was not necessarily associated with degenerative change or it occurred so infrequently that it was not detected in this series of specimens (145).

Radiographs of the lumbar spine frequently demonstrate asymmetry of posterior articular facets, but this is asymptomatic in patients with good abdominal and lumbar muscles when the anomaly is only of a moderate degree. It can, however, cause rotatory instability of the subjacent vertebra, leading to lumbago. It is then frequently associated with osteoarticular complications affecting the posterior arch, a logical consequence of a sequence of changes that can be explained by simple mechanical factors (146).

Determining Tropism by Plain Film Versus CT Study

In one of our studies (147) we found the accuracy of defining tropism on plain x-ray study of 20 patients to have been 27% by one interpreter and 50% by another. CT was the accurate diagnostic modality against which plain x-ray study was compared.

Tropism is a common anomaly, with an occurrence of 17 to 31% in several large series (143, 148–150). Higher tropism incidence is reported in patients with clinically and surgically proved disc herniations as opposed to a lower incidence in persons without back complaints (151), which is a biomechanical factor of importance to the manipulative physician. Plain radiograph has limited accuracy in diagnosing facet articular plane, whereas CT is the best modality for viewing the entire contour of the zygapophysial joints.

Further Controversy

Magnetic resonance imaging and CT scans of 46 subjects under 50 years of age showed increased risk of disc degeneration in the presence of facet joint tropism (152). CT/discography at 324 lumbar levels showed no differences in the degree of disc degeneration or pain response with respect to facet tropism (153). Although reporting no association between either the presence or the severity of facet tropism and disc degeneration, patients who had severe facet tropism at L4 or L5 had a 6.6 times greater risk of disc herniation (154).

Dai and Jai (155) reported that 28% of normal subjects had facet asymmetry at the L4–L5 and L5–S1 levels, but 49% of lumbar disc surgery patients showed facet asymmetry. This study supported the causative significance of facet asymmetry in lumbar disorders. Also reported by Dai and Jai was a Farfan and Sullivan study showing asymmetry of the facet joints in 76 of 78 disc herniation patients with 95% of the herniations occurring on the side of the more obliquely oriented facet. Rotation was found to be greater to the more oblique faced facet side.

Facet Orientation Circle to Determine Tropism

Figures 2.35 and 2.36 depict the determination of facet orientation from a technique known as the "facet orientation circle." The transverse orientation of the lower lumbar facet joints is measured on CT scan as a reference for biomechanical and clinical determination of facet asymmetry. Facets at

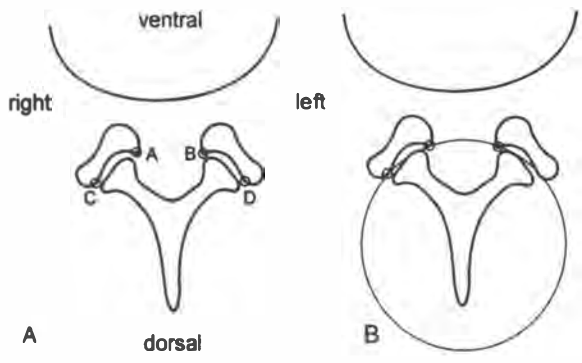

Figure 2.35. **A.** Reference points used for determination of facet orientation circles ([FOC]; all reference points located on the superior articular facets of the underlying vertebra): *A*, anteromedial edge of right facet. *B*, anteromedial edge of left facet. *C*, posterolateral edge of right facet. *D*, posterolateral edge of left facet. **B.** Right FOC through reference points A, B, and C. In the absence of facet joint asymmetry, this circle also passes through reference point D. Left FOC (through reference points A, B, and D) was drawn in a similar fashion (not shown). The diameter of the combined FOC was defined as the mean value of the diameters of right and left FOCs. (Reprinted with permission from van Schaik JPJ, van Pinxteren B, Verbiest H, et al. The facet orientation circle: a new parameter for facet joint angulation in the lower lumbar spine. Spine 1997;22(5):531–536.)

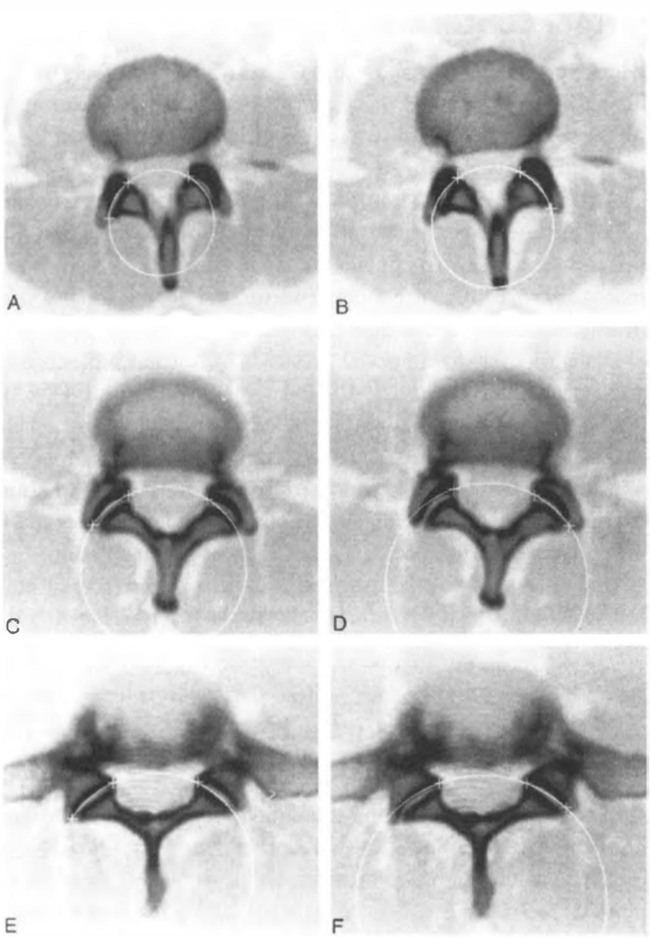

Figure 2.36. Examples of facet orientation circles (FOCs) at various vertebral levels (different patients). Reference points are indicated by crosslets through which circles are drawn. Right FOC (**A**) and left FOC (**B**) at L3–L4 (diameters, 42.4 mm and 43.9 mm, respectively; mean, 43.2 mm). Right FOC (**C**) and left FOC (**D**) at L4–L5 level (diameters, 64.3 mm and 75.2 mm, respectively; mean, 69.8 mm). Right FOC (**E**) and left FOC (**F**) at L5–S1 (diameters, 81.2 mm and 85.7 mm; mean, 83.5 m). Note the more frontal orientation of the facet joints at L5–S1, resulting in a larger facet orientation circle. (Reprinted with permission from van Schaik JPJ, van Pinxteren B, Verbiest H, et al. The facet orientation circle: a new parameter for facet joint angulation in the lower lumbar spine. Spine 1997;22(5):531–536.)

L3–L4 are oriented closer to the sagittal plane, whereas at L4–L5 and L5–S1 they are oriented progressively more toward the frontal plane. This technique should not be used primarily to direct clinical care, but to elicit morphometric data for consideration of biomechanical concepts in investigating the lumbar spine (156).

Concepts of Pain Production by Damaged Disc Tissue

The concept that the intervertebral disc is per se biochemically active after injury has not yet been widely accepted in clinical practice. Crock (157) finds:

1. The capillaries related to the vertebral end plate cartilage drain via a subarticular collecting vein system into the internal vertebral venous plexus or directly into veins of the marrow spaces in the spongiosa of the vertebral body.
2. Trauma to an intervertebral disc, inflicted by heavy lifting or by the high-speed application of force of short duration, may damage disc components, resulting in the production of irritant substances that can drain either into the spinal canal, irritating nerves, or into the vertebral body, thus setting up an autoimmune reaction.
3. The following clinical syndrome may then develop: (*a*) intractable back pain with aggravation of pain and loss of spinal motion with any physical exercise; (*b*) leg pain; (*c*) loss of energy; (*d*) marked weight loss; and (*e*) profound depression.
4. Patients with this syndrome will be found to have (*a*) normal plain radiographs of the spine; (*b*) normal myelograms; (*c*) normal CT scans of the spine; (*d*) usually normal blood examination; and (*e*) normal neurologic findings on clinical examination.
5. If this syndrome is present, (*a*) the patients will have abnormal discograms; (*b*) pain will be reproduced by as small a volume as 0.3 mL of dye because of the hypersensitivity of the pain fibers within the disc substance; (*c*) the final volume of dye accepted will be in excess of normal; and (*d*) the discographic patterns on x-ray films will be abnormal.

This hypothesis suggests that in certain individuals, especially after trauma, a syndrome develops because of the production of chemical substances by the damaged disc tissues (157).

Disc degeneration is characterized histologically by loss of tissue in the nucleus, increasing thickness of the collagen fibers, and the occurrence of fissures both in the center and in the periphery of the disc. Insufficient diffusion into the disc has been said to account for premature disc degeneration.

In a study of the pH of discs of patients operated on for lumbar rhizopathy, a marked decrease in pH was noted in some discs. These cases also showed an abundance of connective tissue scarring around the nerve roots. A number of mechanisms could have caused this increase in hydrogen ion concentration, but a separate study (158) demonstrated that the main factor was probably increased lactic acid concentration, which was found to be directly correlated with the hydrogen ion concentration of the nucleus.

Thus, this study suggests that two nutritional routes are open for the intervertebral disc: (*a*) diffusion through the central portion of the end plate from marrow space cartilage contacts and (*b*) diffusion through the anulus fibrosus from the surrounding vessels (158).

Does the Disc Have Circulation?

The imbibition of fluids into the nucleus pulposus has always interested me, as it relates to the possible nutritional advantages of supplying minerals and glucosaminoglycan orally to patients

with disc degeneration in an attempt to reverse the degenerative process. An exciting factor was shown by Eismont et al. (159) when they found penetration of antibiotics into the nucleus pulposus following an 8-hour course of intramuscular antibiotic injections.

Immunologic Implications of Lumbar Disc Disease

Naylor et al. (160) state that a hypothesis to explain the chemical process of disc prolapse would include the initial change as a disturbance of the normal protein-polysaccharide synthesis-depolymerization equilibrium in favor of increased or unbalanced depolymerization, with the changes in the proteoglycan metabolism being associated with an increased fluid content and, thus, increased intradisc tension. This could then produce an episode of disc nuclear herniation. Five acid glycerophosphatases have been isolated from disc material. These lysosomal enzymes can be shown to degrade the intervertebral disc. Of these five acid glycerophosphatases isolated in normal nuclei, two have the same activity during prolapse, one has a lower activity, and the others have some deficiencies. The Naylor et al. study suggests that lysosomal enzymes present in the nucleus pulposus of the prolapsed intervertebral disc are capable of degrading the protein-polysaccharide complexes.

Elves et al. (161) studied 12 patients with prolapsed intervertebral discs. All patients had discectomy performed. Eight of these patients had protrusion, and four had sequestration or prolapse with free fragmentation of the disc. Three of the four patients with prolapse showed an immune response to their own disc material. None of those with protrusion had a positive immune reaction.

Naylor et al. (160) found a significant enhancement of IgM and IgG in patients with lumbar disc prolapse. They suggest that either a nonspecific antigen process or stimulation of an antibody humoral system is the factor in the development of disc prolapse. Gertzbein (162) believes that evidence exists for an autoimmune mechanism in the degeneration of the lumbar disc.

It was Falconer (as discussed by Naylor et al. [160]) who originally stated that, on myelography, defects could still be observed in patients whose low back and leg pain had been completely relieved. Thus, evidence supports the claim that the pain from disc prolapse is caused by chemical as well as mechanical irritation of nerve roots. Once the degradation products of prolapse are dissipated, the relief of symptoms may be imminent.

Direct chemical analysis, x-ray crystallography, and electron microscopy have shown that disc degeneration shows a fall in total sulfate, both keratin and chondroitin, although no pH change occurs. In disc herniation a fall is seen in total proteoglycan level, chiefly chondroitin sulfate, and probably in keratosulfate fractions.

The chemical explanation of disc prolapse expressed here is that initially a disturbance of the normal protein-polysaccharide synthesis occurs, which is associated with an increased fluid content and intradiscal pressure that produces the damage to the anulus, with repeated episodes producing advanced degeneration of the disc. *What creates these changes? The lysosomal enzymes of arthritis and rheumatoid arthritis are similar and may produce the disc changes of herniation.* Ruptured discs have been shown to release *acid phosphatase,* which degrades the protein-polysaccharide complexes of the intervertebral disc (160).

It has been shown (160) that the intervertebral disc could act as an antigen, with the common antigenic determinant located in the region of the glycosaminoglycan to the protein core.

IgM, IgG, and IgA have been isolated in the serum of patients with prolapse and not in the serum of normal healthy people (162). It is primarily IgG and IgM that are elevated in patients with lumbar disc prolapse. A reaction between IgM and the protein polysaccharide complex has been shown to produce *amyloid* similar to that found in the amyloid-containing tissues of patients with rheumatoid arthritis (160).

Many believe that chronic degeneration of the disc is an autoimmune disease with antibodies directed at components of the nucleus pulposus that normally are shielded from the circulation and the reticuloendothelial system. A highly significant increase of serum IgM was reported in patients with proved Schmorl's nodes, narrowed disc spaces, or neurologic signs of disc damage, compared with age-matched controls (163)(Fig. 2.37).

Chemical Irritation of a Nerve Root As a Pain Producer

Disc Prolapse As Chemical Irritant of Nerve Root

For more than a decade, orthopaedic surgeons have considered the likelihood of chemical irritation of the nerve root in association with disc prolapse as the cause of the acute pain following injury. This view has arisen from the frequent finding at operation of a swollen, inflamed nerve root without bone pressure. Glycoprotein is a constituent of the chemical content of the nerve root. Previously, it was shown that the carbohydrate capsule of the pneumococcus liberates histamine and other H substances from perfused organs much in the same way as venom. Direct pharmacologic tests of the nucleus pulposus show the presence of 1 to 4 μg of histamine per gram, but no tryptamine and no slow-reacting substance or kinin. Extract of the glycoprotein from human nucleus pulposus releases considerable quantities of histamines, edema fluid, protein, and another amine with four times the mobility of histamine from the isolated perfused lung of the guinea pig. The acute pain in disc lesions is caused by local irritation of the nerve root producing edema and releasing protein and H substances at the site of disc injury. Relief of pain by cortisone accords with these findings, because cortisone inhibits the peripheral response to H substances (164).

Disc Anular Irritation As Source of Low Back Pain

Anatomic studies have demonstrated the presence of nociceptive nerve endings in the anulus fibrosus of the lumbar intervertebral disc. Anular tears can, therefore, cause pain referral of purely discogenic origin into the low back, buttock, sacroiliac region,

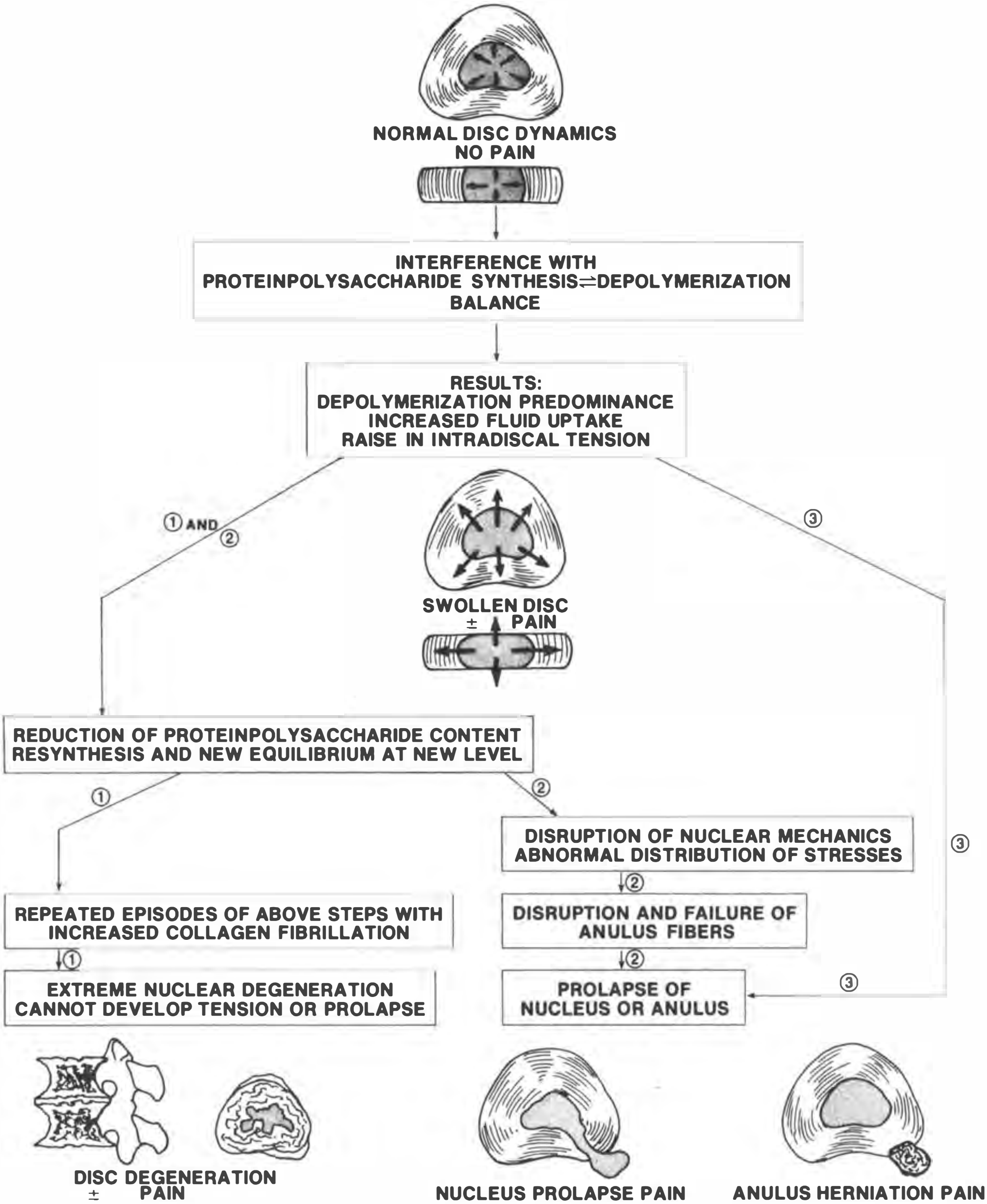

Figure 2.37. This flow diagram explains the biomechanical hypothesis of the basic mechanisms of spine pain, disc prolapse, and disc degeneration. A number of mechanical factors mentioned in this chapter probably play a large role in the clinical presentation and outcome of these various biochemical phenomena. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:291.)

and lower extremity even in the absence of neural compression. Neural compression caused by an anular tear that has progressed to become a protruded disc is an obvious source of pain. Disc protrusion without neural compression can precipitate an inflammatory response with secondary radiculitis, raising the possibility of chemically induced inflammatory neural pain (165).

Lumbar zygapophysial joints (i.e., facet joints) are well innervated and thus are potent potential pain generators. Facet arthropathy can cause low back pain as well as refer pain into the buttock and lower extremity.

Basic anatomy and pathophysiology of lumbar nerve injury reveal that the motor (i.e., ventral) nerve root and sensory (i.e., dorsal) nerve root pass dorsal and lateral to the intervertebral disc.

INTERVERTEBRAL DISC BIOMECHANICS—NORMAL AND ABERRANT

In people between the ages of 30 and 40 years, their nucleus has a water content of 80% (166), which Puschel (167) believes decreases with age. DePukey (168) found that the average person is 1% shorter in height at the end of the day than on first arising in the morning. He also found that a person in the first decade of life is 2% shorter at bedtime, and a person in the eighth decade of life is 0.5% shorter. This difference he attributes to decreasing water content in the disc, which occurs with advancing age.

Hendry (169) believes that the hydrodynamics of the disc result from the gel structure of the nucleus pulposus, enabling it to absorb nine times its volume of water. No chemical bond influences this water content, as it can be mechanically expressed under pressure; thus, weightbearing causes the decrease of 1% average height in a day.

The nucleus pulposus, which occupies about half the disc surface area, bears the vertical load, whereas the anulus bears the tangential load (134). Because of nuclear degeneration, shift occurs in stress and weightbearing forces. Bradford and Spurling state that the ratio of the anterior to posterior weightbearing forces of the body is 15 to 1; therefore, lifting 100 pounds with the arms extended places a total pressure of 1500 pounds on the nucleus pulposus. Even more revealing is the finding of Morris et al. (170) that a 170-pound man lifting 200 pounds exerts a force of 2071 pounds on the L5–S1 disc space.

Discography is performed by injecting contrast material into the nucleus, which normally accepts approximately 1 mL of solution. If the injection duplicates the patient's symptoms, disc protrusion, irritating the anulus or nerve root, is signified. Figure 2.38 reveals abnormal nuclear appearances on discography.

Gresham and Miller (171) carried out postmortem discography on 63 fresh autopsy specimens; the subjects ranged in age from 14 to 80 years, and they had relatively asymptomatic backs. The results of this study are presented in Table 2.4.

Abnormalities in the disc reduce its capacity to aid in supporting torsional loads of the spine by about 40% (134).

Degeneration of the intervertebral disc and subsequent changes in adjacent vertebrae and ligaments are termed "spondylosis." Fissuring of the anulus fibrosus occurs posteriorly, usually where the common ligament is least strong (172). Finneson (134) describes two disc changes following injury (Fig. 2.39): disc herniation (or protrusion) and spondylosis. He notes that in less than 20% of patients with anular tears or fissures, a large fragment of nucleus bulges forth to compress a nerve root, producing classic disc symptoms. Usually, however, the anulus never completely tears and contains the nucleus within its boundary with only slight protrusion.

Finneson goes on to say that fibrosis of the anulus fibrosus

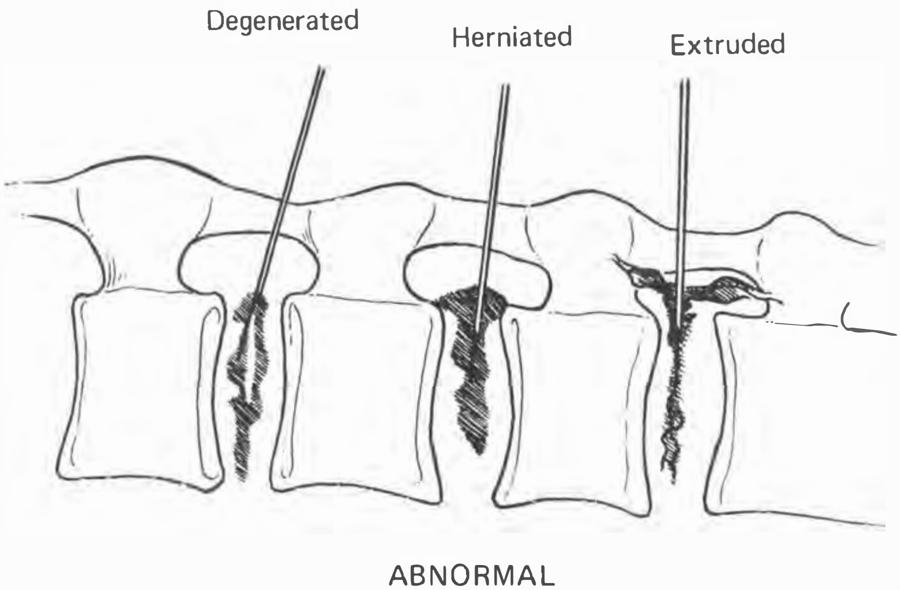

Figure 2.38. Some abnormal discogram configurations. (Reprinted with permission from Finneson BE. Low Back Pain. 2nd ed. Philadelphia: JB Lippincott, 1980:104.)

Table 2.4

Results of Postmortem Discography from a Study by Gresham and Miller (Total Autopsies, 60)

Group	Age Range (years)	Findings
I	14–34	90% normal discs
		10% degenerated discs
II	35–45	25% normal discs
III	46–59	25% normal discs at L3–L4
		0% normal discs at L5–S1
IV	60 and over	5% normal discs
		0% normal at L5–S1
		2% normal at L4–L5[a]
		3% normal at L3–L4[b]

Data from Gresham JL, Miller R. Evaluation of the lumbar spine by diskography. Orthop Clin 1969;67:29.
[a]One autopsy in 60.
[b]Two autopsies in 60.

occurs as the anulus loses its sponginess and elasticity. The disc space thins, with sclerosis of the cartilaginous end plates and new bone formation around the periphery of the contiguous vertebral surfaces occurring. The altered mechanics place stress on the posterior diarthrodial joints, causing them to lose their normal nuclear fulcrum for movement. With the loss of disc space, the articulation plane of the facet surfaces is no longer congruous. This stress results in degenerative arthritis of the articular surfaces. Complete fibrous ankylosis of the disc and articular surfaces is possible.

Definitions and Illustrations of Disc Protrusion and Prolapse

Two terms are used to describe disc degenerative change allowing nuclear herniation: "contained disc" and "noncontained disc." They refer to the state of the anulus fibrosus, that is, whether it is intact and restraining the nucleus pulposus (a contained disc); or whether it has completely radially torn to allow the nuclear material to sequester or free-fragment into the vertebral canal (a noncontained disc).

Disc protrusion (Fig. 2.40) is an extension of nuclear material through the anulus into the spinal canal with no loss of continuity of extruded material. The anulus is intact. Protrusion and herniation are synonymous.

Disc prolapse (Fig. 2.41) occurs when the extruded mater-

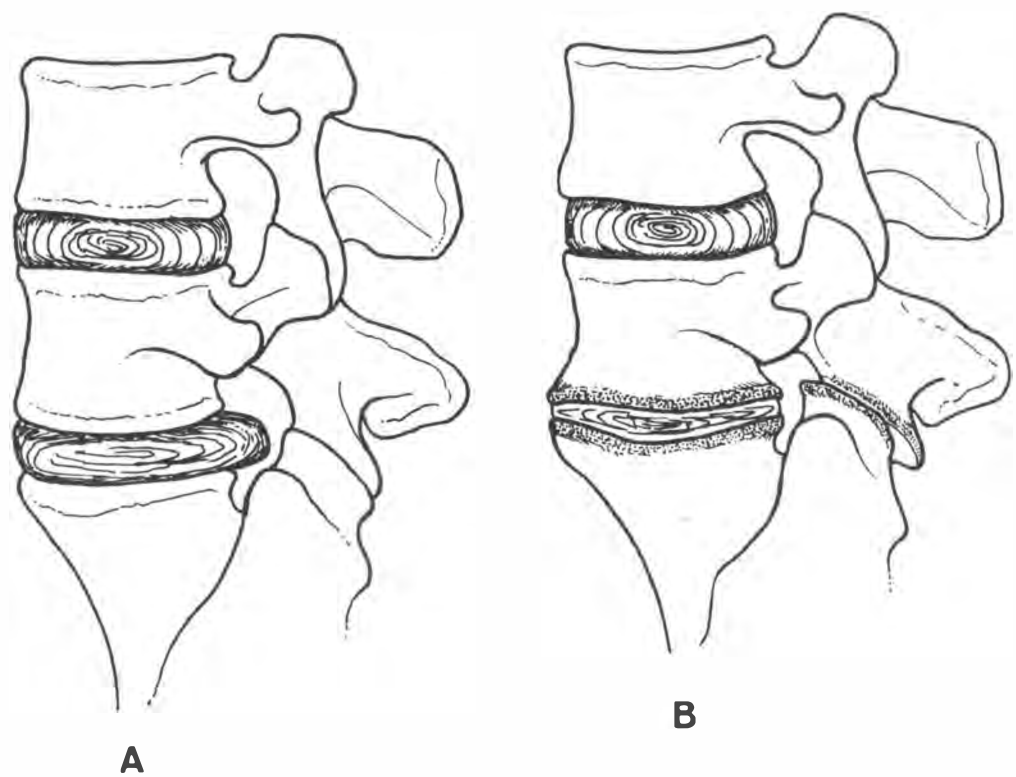

Figure 2.39. **A.** Herniation of the nucleus pulposus. **B.** Spondylosis. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:437.)

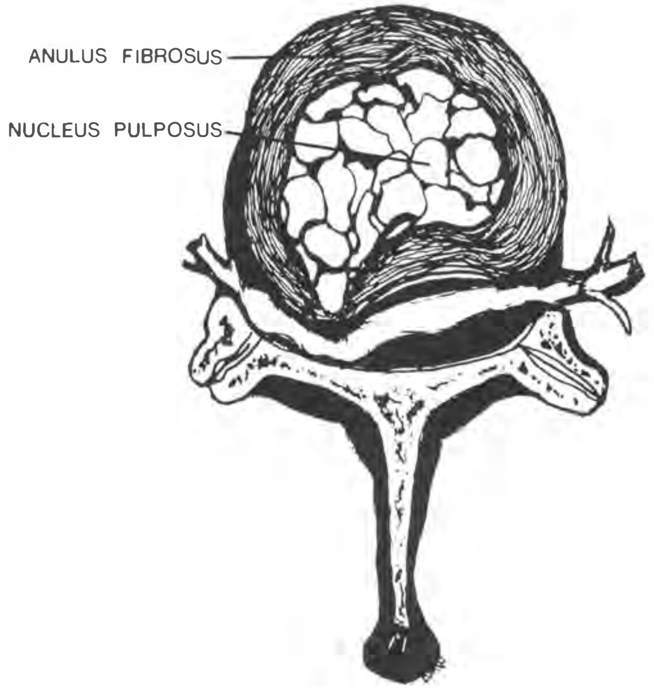

Figure 2.40. Nuclear protrusion. The anulus fibrosus is still intact, although weakened with nuclear bulge.

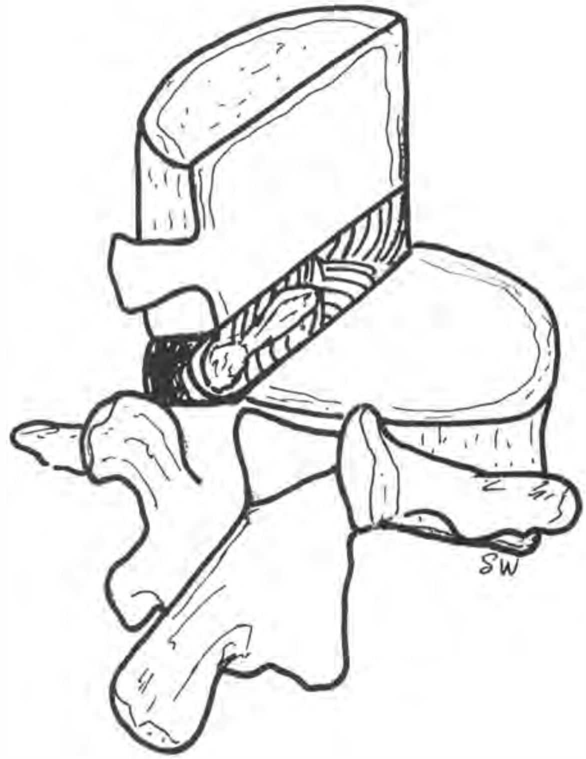

Figure 2.42. In nuclear protrusion, the anular fibers are containing the bulging nuclear material.

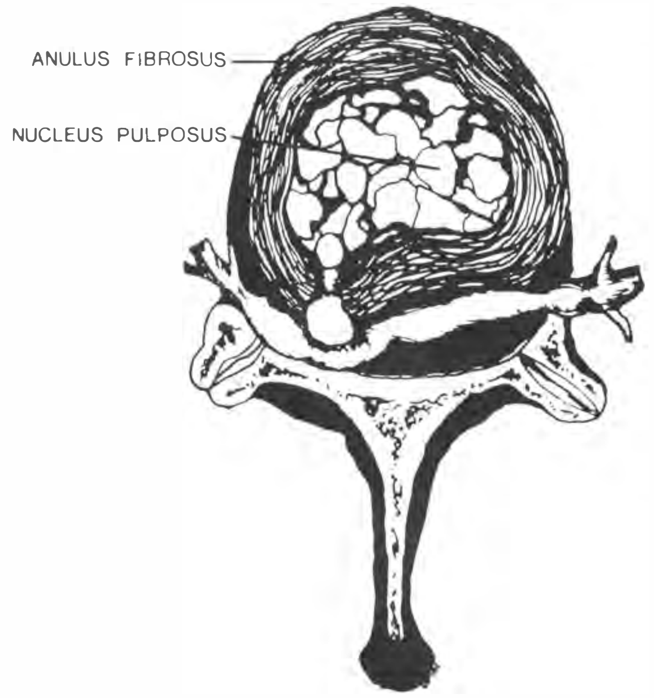

Figure 2.41. Nuclear prolapse. The anulus fibrosus is completely torn, allowing nuclear escape into the posterior vertebral canal as a free fragment.

ial loses continuity with the existing nuclear material and forms a free fragment in the spinal canal. The anulus is not intact.

Protrusion of disc material (Fig. 2.42) exists when the bulging nuclear material is contiguous with the remaining nucleus pulposus, and the anulus fibrosus is stretched, thinned, and under pressure. Epstein (173) notes that the pressure within the nucleus pulposus is 30 psi and mentions that this pressure was found to be 30% less in the standing position than in the sitting position and 50% less in the reclining position than in the sitting position. Also keep in mind that cerebrospinal fluid pressure is 100 to 200 mm of water in the recumbent posture and 400 mm in the sitting posture (174). It is important, therefore, that the patient with a protruding disc avoid sitting. Disc prolapse is shown in Figure 2.43.

Figure 2.44 illustrates that a disc can protrude either lateral to a nerve root, medial to a nerve root, under a nerve root, or in a central position. When the disc protrudes lateral to the nerve root, the patient assumes an antalgic lean away from the side of the disc lesion (Fig. 2.45). When the disc protrudes medial to the nerve root, the patient assumes an antalgic lean into the side of the disc lesion or pain (Fig. 2.46). With a central disc lesion, the patient assumes a flexed posture of the lumbar spine with or without lean to either side. With protrusion under the nerve root, the patient may assume no lean.

Figure 2.47 illustrates the great challenge in low back complaints—a patient with low back pain with radiating sciatic radiculopathy; inability to bear weight; pain on coughing,

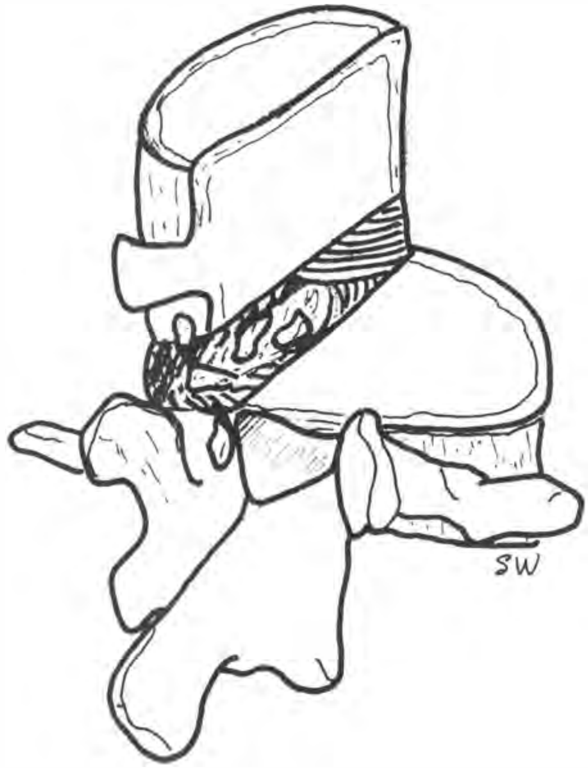

Figure 2.43. In nuclear prolapse, the anulus completely tears, allowing escape of free fragments of nuclear material into the vertebral canal or extremely laterally into the intervertebral foramen.

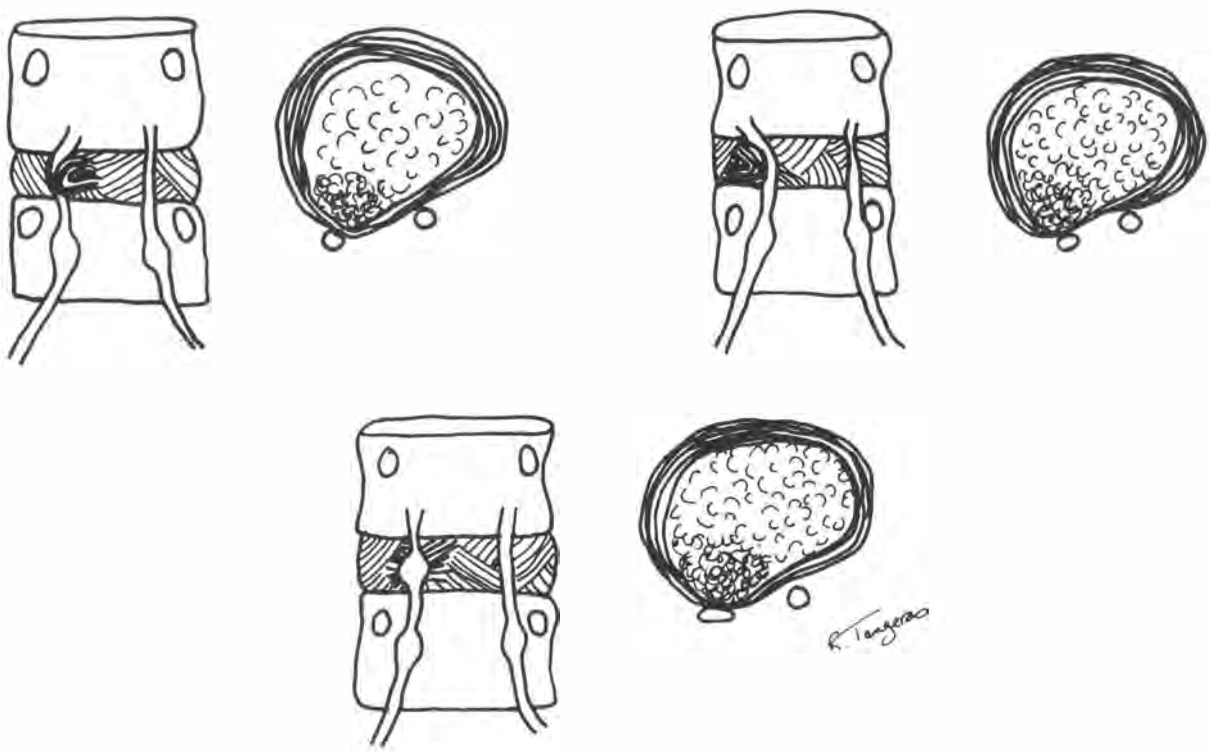

Figure 2.44. Nerve root displacement by disc protrusion. *Upper left*, Medial disc displaces nerve laterally. *Upper right*, Lateral disc displaces nerve root medially. *Lower center*, Disc lies directly under nerve root, stretching it.

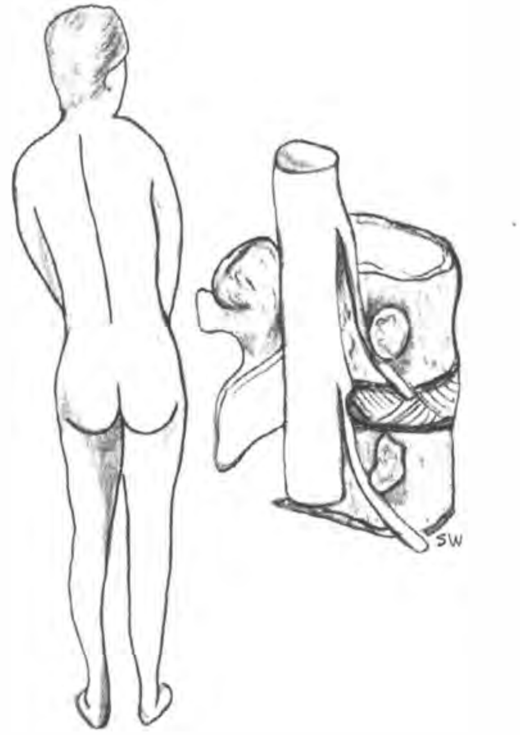

Figure 2.45. Sciatic scoliosis in a patient with a right lateral disc protrusion.

Figure 2.47. The great challenge in low back pain patients—this figure shows a patient with low back pain and radiating sciatic radiculopathy, leaning in a flexed position and unable to straighten the leg. This is a typical herniated nucleus pulposus case.

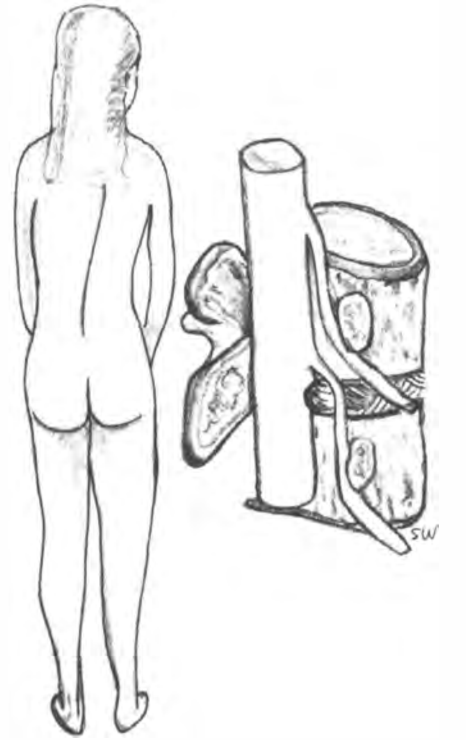

Figure 2.46. Sciatic scoliosis in a patient with right medial disc protrusion.

sneezing or bowel movement; reflex change; and great fear and anxiety. The patient's radiograph (Fig. 2.48), except for an obvious sciatic list to the right side, is devoid of degenerative changes, which may be a certain part of her future, whereas the CT scan seen in Figure 2.49 reveals good reason for her discomfort. This is an obvious diagnosis; unfortunately, most low back diagnoses are not so easily made.

The dermatome chart (Fig. 2.50) reveals innervation of the sensory nerves of the lower extremity. Ninety percent or more of lumbar disc lesions occur either at the L4–L5 or L5–S1 disc level. The L4–L5 disc usually compresses the fifth lumbar nerve root, resulting in pain sensations down the lower extremity in the fifth lumbar nerve root innervation. The L5–S1 disc usually compresses the first sacral nerve root, resulting in pain distribution down the first sacral dermatome of the lower extremity. Lecuire et al. (175) found that of 641 patients with disc lesion, 307 showed definite S1 dermatome patterns, 267 showed definite L5 dermatome patterns, and 67 showed mixed patterns; 60% of these patients had an antalgic lean. A single disc lesion was noted in 562 patients, with 47% occurring at L5–S1, 39% occurring at L4–L5, and 2% occurring at L3–L4. Myelograms were performed on 238 of the 641 patients prior to surgery.

Knowledge of specific innervation of the nerve root is important in deciding which disc is involved. By ascertaining the antalgic posture, the clinician can determine whether the prob-

Figure 2.48. Posteroanterior radiograph of the patient in Figure 2.47. This is a Lovett reverse sciatic scoliosis in that the spinous processes rotate to the left convexity instead of to the right concave side. This is a typical rotation scoliosis seen in serious disc lesions.

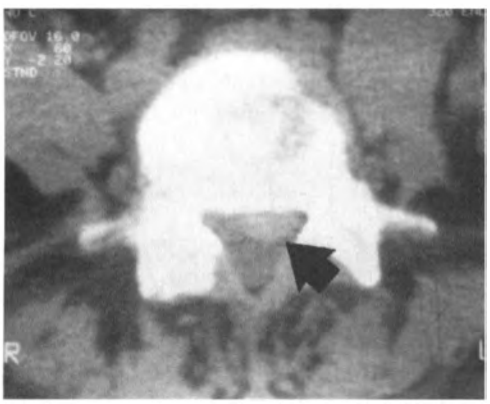

Figure 2.49. Computed tomography scan of the patient in Figure 2.47 shows a large left disc herniation at the L4–L5 disc level. All cases should be so easily diagnosed!

lem is a medial, central, or lateral disc protrusion. Therefore, two facts are of primary importance in the evaluation of a patient: the side of sciatic pain distribution and the side of antalgic inclination (i.e., whether the patient leans toward or away from the side of pain) (Fig. 2.51).

I have found that the level of disc involvement usually is the site of vertebral rotational and lateral flexion changes; this level may be observed on visual examination of the patient's spine. Often it is noted only on x-ray examination; x-ray studies can be made with the patient in both recumbent and standing positions, because no difference is noted in these disc cases. The site of lateral flexion and rotation change may be quite noticeable or only slightly discernible on radiographs; therefore, close correlation with the history and clinical examination is needed to pinpoint the site of disc protrusion. In other cases, the x-ray finding is striking regarding the amount of flexion and rotational change that results in a sciatic scoliosis. Some cases of disc prolapse requiring surgery, however, often reveal minimal change in functional spinal unit relationships. An interesting observation is that sciatic scoliosis often appears as a Lovett failure or as reverse scoliosis (i.e., a failure of body rotation or a rotation to the convexity of the scoliosis by the vertebral bodies instead of toward the side of concavity).

RADIOGRAPHIC STUDY OF LATERAL FLEXION DISC MECHANICS OF THE LUMBAR SPINE AND PELVIS

The biomechanics of the lumbar spine and pelvis are well shown radiographically by use of the dynamic lateral bending study. Without it, one of the most important tools of diagnosis of lumbar mechanics is lost. Weitz (176) revealed the accuracy of lateral bending studies by comparing his findings with those of myelography and surgery. Of 46 patients, he found 12 had normal bending studies; of these, six had midline disc protrusions and two had stenosis. Of the 34 patients with abnormal bending studies, 28 had disc protrusions confirmed at both myelography and surgery. Two of the 34 had abnormal bending studies that were confirmed at surgery despite negative myelography. Both patients had lateral disc protrusions, with normal bending away from the protrusion and impaired bending toward the protrusion. No instance has been reported of a patient with an ipsilateral list and a negative myelogram.

Bending Study Accuracy

Van Damme et al. (177) compared the relative efficacy of clinical examination, electromyography, plain film radiography, myelography, and lumbar phlebography in the diagnosis of low back pain and sciatica. They found that *the bending studies had diagnostic reliability equal to that of myelography* and lumbar phlebography.

In 1942, Duncan and Haen (178) stated that the postural attitude assumed by a patient with a disc protrusion was such to avoid further compression of the disc: "This posture entails a list of the spine away from the side of the lesion and since the mass is extruding posteriorly, an attitude of forward flexion is assumed." They took films of the patient in lateral flexion to each side and in flexion and extension and found that, "in the majority of our cases, these films have demonstrated a lack of spinal mobility localized to the involved joint" (178). They also found that, in patients with laterally placed herniation, the myelograms were consistently normal. (*Note:* We know that lateral discs can be so far lateral as not to contact the dye-filled subarachnoid space, thus giving a false-negative myelogram—

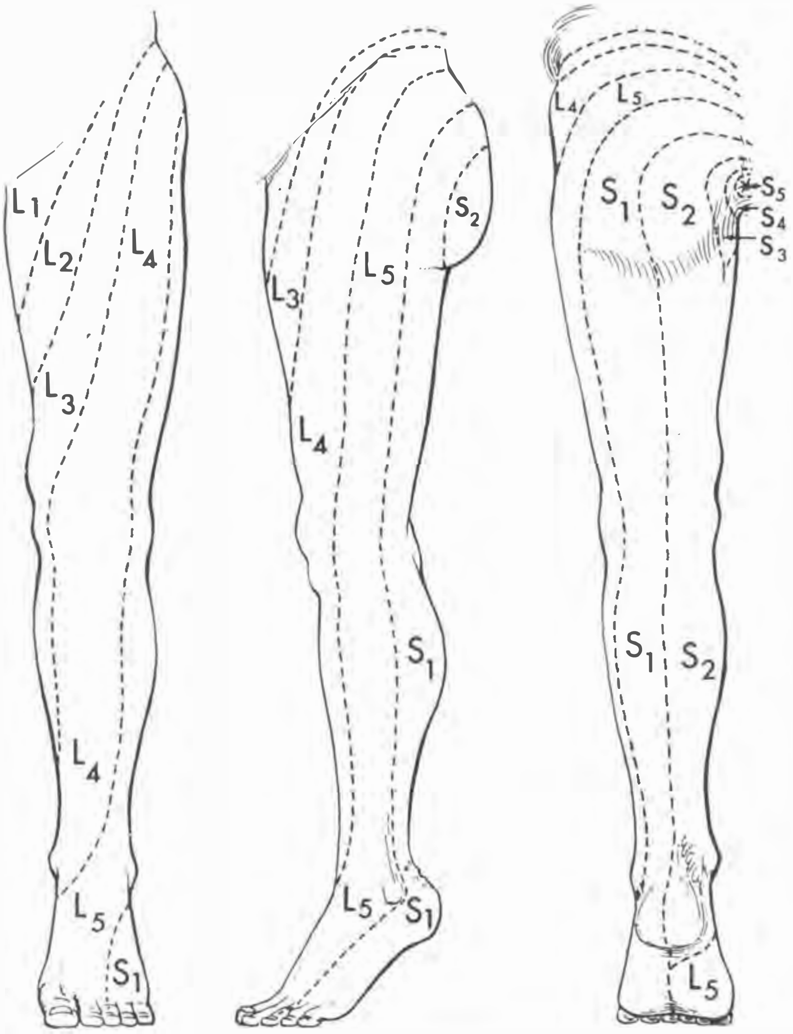

Figure 2.50. Dermatome chart of lower extremity.

one reason for the 30 to 40% inaccuracy of myelography.) They also took postoperative bending films, which showed that once the sequestrum had been removed from the involved joint, mobility was immediately restored to the joint.

In 1948, Falconer et al. (179), in a study of 25 patients with ipsilateral list and 17 with contralateral list, with the summit medial or lateral to the nerve root in both subgroups, discussed the importance of list in lumbar disc disease. They found that scoliosis was caused by spasm designed to exert the least possible "strain" on the surrounding structures, but they were unable to correlate the direction of the curvature with the side of the symptoms. One year later, Hadley (180) noted that in certain patients with nerve root pressure, the foramen is not allowed to become smaller on lateral flexion toward the affected side, although normal wedging can take place at this level when the patient bends to the opposite direction.

Schalimtzek (181) and Hasner et al. (182) performed motion studies to diagnose herniated discs. Hasner et al. discovered that if lateral bending is inhibited, either normal angulation between vertebral bodies may be less pronounced or a state of parallelism may be noted. They (181, 182) also found that the vertebral bodies may even be divergent from each other on the side where the lateral bending takes place.

Breig (183) states that the patient's posture in an acute back disorder represents a compromise between the need to minimize tension in the dura and the root and the need to reduce the bulge of the prolapsed disc. He believes that it is not uncommon to see a patient with a flattened lumbar region flex the spine forward to minimize the herniation and ipsilaterally to relieve tension on the root, such as occurs in a patient with an axillary herniation (a medial disc).

Controversy Regarding Lateral Flexion Accuracy

Porter and Miller (184) do not find lateral flexion as diagnostic as other authors and state that, in a study of 100 patients with trunk list and back pain, they found 49 who fulfilled the criterion of a symptomatic lumbar disc lesion, and 20 of these required surgical excision of the disc. The side of the list was not

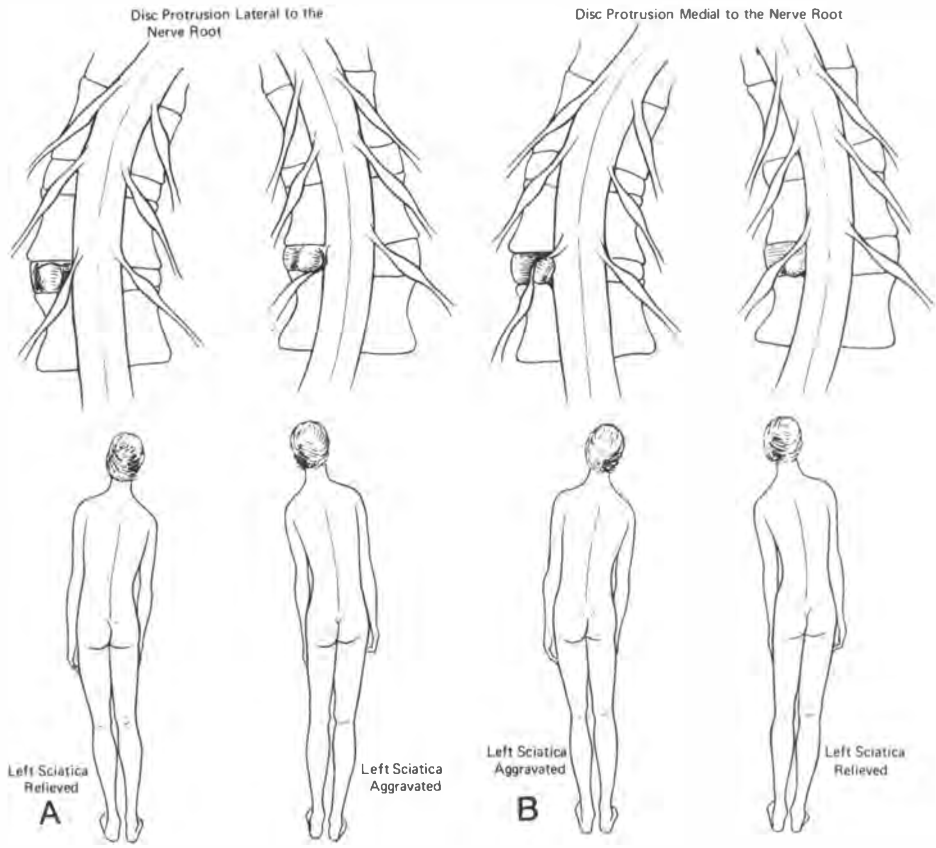

Figure 2.51. Relief or aggravation of pain with lateral flexion may indicate whether the disc protrusion is lateral or medial to the nerve root. (Reprinted with permission from Finneson BE. Low Back Pain. 2nd ed. Philadelphia: JB Lippincott, 1980:302.)

related to the side of the sciatica or to the topographic position of the disc in relation to the nerve root. Twice as many patients listed to the left as to the right, and some evidence was found that the side of the list may be related to hand or leg dominance.

Finneson (185) has demonstrated both ipsilateral and contralateral listing caused by the relationship of the protrusion to the nerve root. Nachemson believes that "the information obtained from ordinary x-rays is . . . mostly irrelevant" (186). Weitz, therefore, states, "It is with this impetus that we urge lateral bending (dynamic) x-ray studies rather than static films in patients clinically suspected of having lumbar disc herniations" (176).

RADIOGRAPHIC STUDIES

Lateral bending studies are performed to determine aberrant lateral flexion of a functional spinal unit in relation to its adjacent segments. These studies are most beneficial in determining subluxation, as in hypomobility of the static subluxation accompanying intervertebral disc protrusion. For study of the L4–L5 level, routine anteroposterior (AP) views in lateral flexion are adequate, but for study of the L5–S1 level, the tilt view must be used because the sacral angle and lumbar lordosis make viewing of the lateral flexion of L5 on the sacrum impossible.

For the tilt view, take the lateral lumbar view as shown in Figure 2.52. Next, draw the sacral promontory line and measure the angle made by this line with the horizontal. Then tilt the x-ray tube to match this angle (Fig. 2.53), with the center ray 1.5 inches inferior to the intercrestal line centered to the midline. In addition to the neutral posteroanterior (PA) view, the lateral bending studies are performed by having the patient slide his hand down his thigh while keeping his feet flat on the floor directly beneath the hip joints and keeping his knees straight (Fig. 2.54). These studies can be performed with the patient either sitting or standing, depending on the doctor's preference, and they provide information on the following:

1. Fixation (hypomobile) subluxation caused by either disc protrusion or facet incongruity;
2. Relief of disc or facet lesions following manipulation, as normal physiologic mobility returns.

Figure 2.55 is an illustration of lateral bending antalgic postures and their effect on the medial and the lateral discs.

Figures 2.56 through 2.58 are the radiographic studies that correlate with the schematic representations in Figure 2.55 of the disc protrusion causing nerve root compression. They are lateral bending studies of the L5–S1 level in a patient with pain

down the right first sacral dematome, and they provide clinical evidence of a right lateral fifth lumbar disc protrusion. Figure 2.56 is the PA neutral view. Note the left lateral flexion subluxation of L5 on the sacrum and the tropism at L5–S1, with the right facets being sagittal and the left facets being obliquely coronal in their planes of articulation. Dye from prior myelography can be seen in the dural root sleeve.

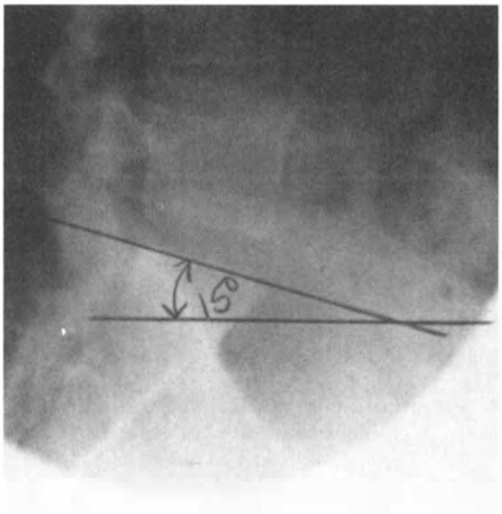

Figure 2.52. An upright lateral spot view. The sacral angle measured 15°.

Figure 2.57 is the left lateral bending study. Note spinous process deviation to the left. Figure 2.58 is the right lateral bending study, and it shows failure of right lateral movement of L5 on the sacrum. L5 is a hypomobile fixation subluxation, as evidenced by failure of lateral flexion or spinous process motion beyond the midline, which occurs in lateral disc protrusion.

Howe (personal communication, 1980) has said that the disc lesion is an area of hypomobility on the cineradiography study. Movement occurs above or below the disc lesion subluxation, but the disc is a hypomobile segment.

Figures 2.59 and 2.60 demonstrate the mechanics of the antalgic leans shown in Figures 2.56, 2.57, and 2.58.

Figures 2.61–2.63 are studies of a patient with right medial disc protrusion at L5–S1. Figure 2.61 is the neutral PA view of the L5–S1 interspace in this patient with pain down the right first sacral dermatome. Note the right lateral flexion of L5 on the sacrum and the tropism present, with the L5–S1 left facets being sagittal and the right facets being coronal. Figure 2.62 shows right lateral bending of the lumbar spine. Note a Lovett-positive scoliosis. Figure 2.63 shows attempted left lateral bending of the lumbar spine with failure of lateral flexion of L5 on the sacrum and with hypomobility of the segments above to laterally flex left. This subluxation pattern is compatible with the motion studies observed during physical examination.

Figure 2.64 is a schematic representation of the antalgia in the patient in Figures 2.61–2.63.

White and Panjabi (141) state that lateral bending produced 2° to 3° of motion at L5–S1, and Tanz (187) has found that lat-

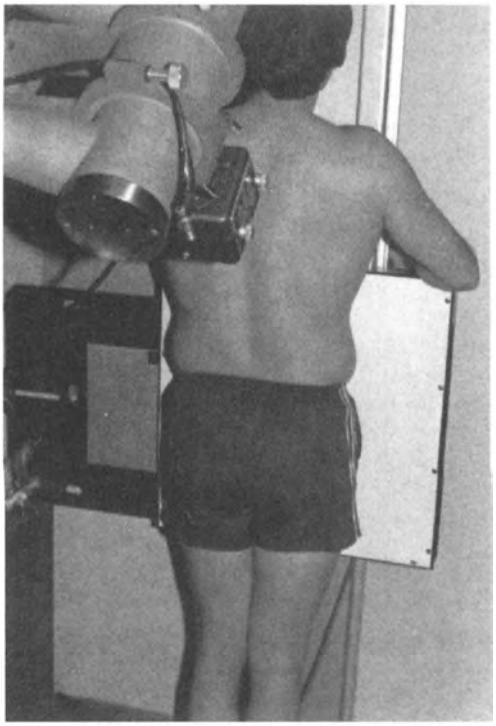

Figure 2.53. Tube tilted to match sacral angle and centered to L5–S1 level.

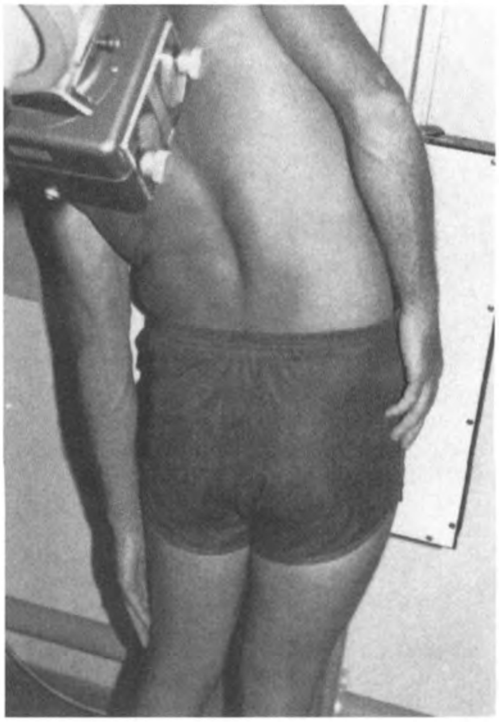

Figure 2.54. Lateral flexion is performed by having the patient slide his hand down his thigh while bending laterally.

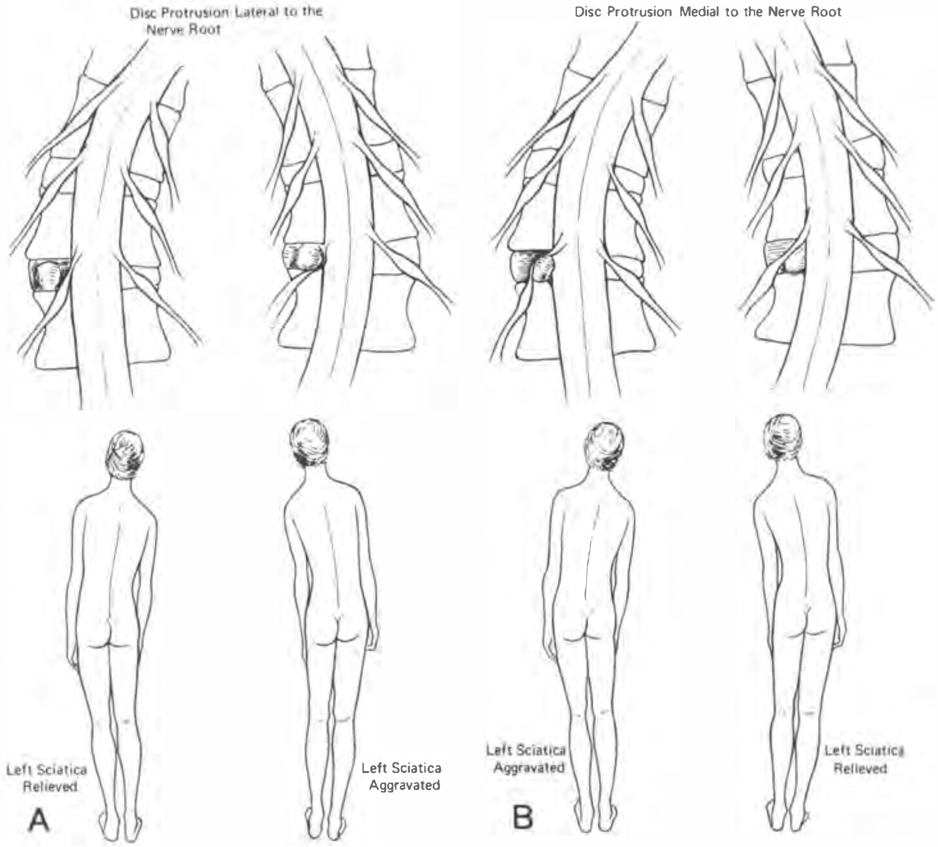

Figure 2.55. Relief or aggravation of pain with lateral flexion may indicate whether the disc protrusion is lateral or medial to the nerve root. (Reprinted with permission from Finneson BE. Low Back Pain. Philadelphia: JB Lippincott, 1973:302.)

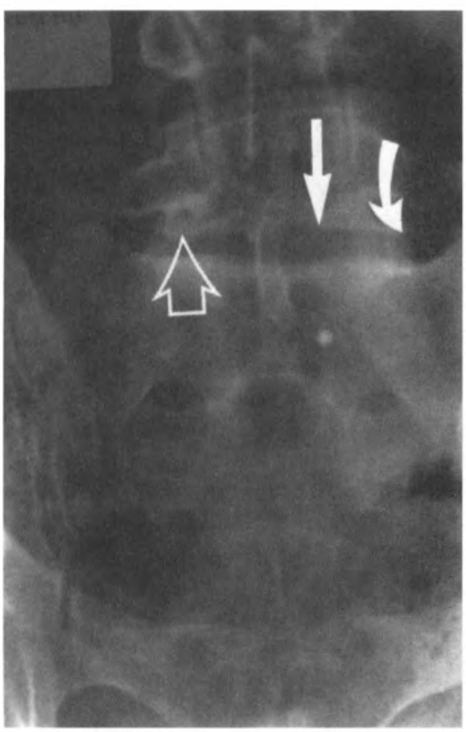

Figure 2.56. Left lateral flexion subluxation of L5 on the sacrum is shown (*straight arrow*). Tropism can be seen at the L5–S1 level, with the right facets faced sagittally (*curved arrow*) and left facets faced coronally (*open arrow*).

Figure 2.57. Left lateral flexion view of the patient seen in Figure 2.56. Good lateral mobility of each functional spinal unit is shown. Note the left lateral flexion subluxation of L5 on the sacrum (*arrow*).

Figure 2.58. Right lateral flexion view of the patient seen in Figure 2.56. Note static subluxation of L5 on the sacrum (*straight arrow*) and lateral movement of L3 on L4 and L4 on L5 (*curved arrow*).

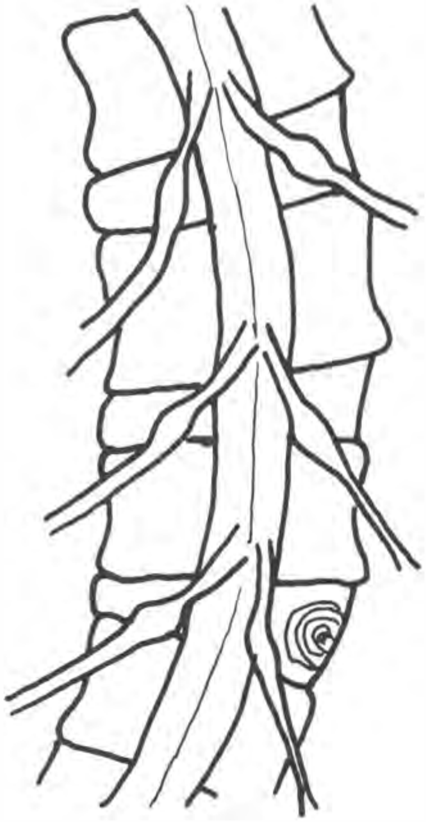

Figure 2.60. Left lean to relieve pressure caused by disc protrusion lateral to nerve root.

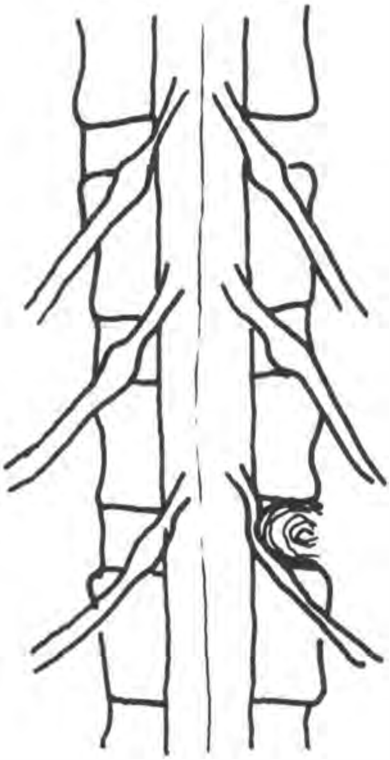

Figure 2.59. A disc protrusion lateral to the nerve root.

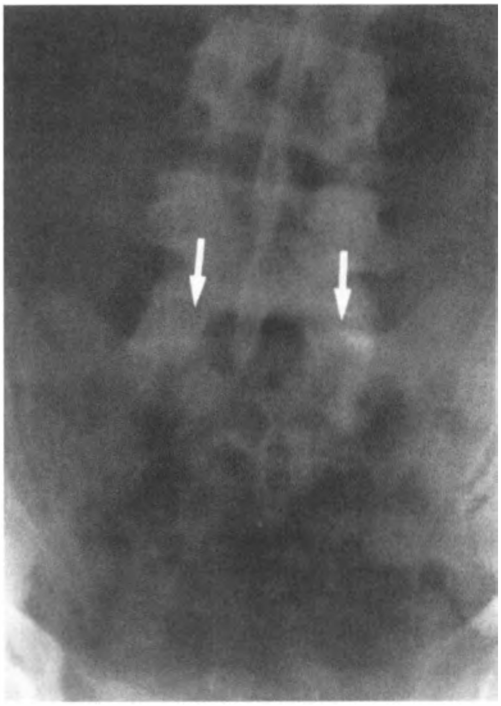

Figure 2.61. Neutral posteroanterior view of L5–S1. Note right lateral flexion of L5 on the sacrum (*arrow*). Tropism can be seen at L5–S1.

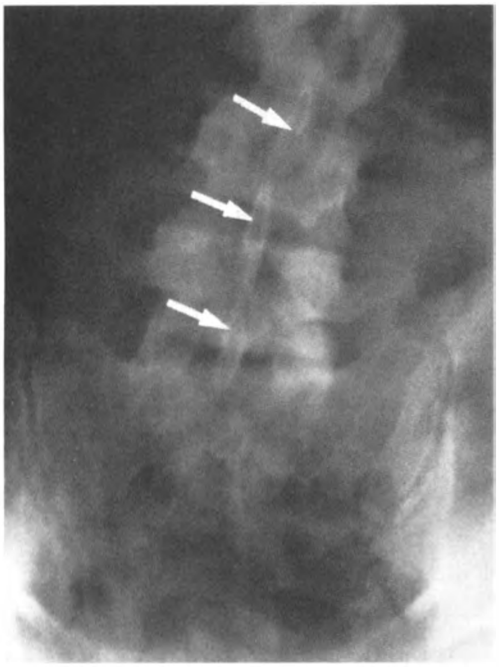

Figure 2.62. Right lateral flexion view showing Lovett-positive curve with spinous deviation into the concavity of the curve (*arrows*).

eral bending produces 7° to 8° of motion at L4–L5 and L3–L4. The greater mobility at the L4–L5 level than at the L5-S1 level would help to account for the greater lateral subluxation occurring in disc protrusions.

Figures 2.65–2.67 are studies of an L4–L5 left medial disc protrusion. Figure 2.65 is the neutral PA view of L4–L5 and shows L4 in left lateral flexion subluxation on L5. The patient has left L5 dermatome pain indicative of a left L4–L5 medial disc lesion. Figure 2.66 is the left lateral bending study of the lumbar spine, with good lateral bending shown above L4–L5. Figure 2.67 is the right lateral bending study, and it shows failure of lateral flexion of L4 on L5. This is a fixation hypomobile discogenic subluxation. Note the motion of the lumbar levels above L4–L5 to the right.

Figures 2.68–2.70 are schematic representations of antalgia in the patient in Figures 2.65–2.67.

Lovett Reverse Scoliosis

Figure 2.71 is a standing AP lumbopelvic view of a patient who has had two myelograms for persistent low back and right leg first sacral dermatome pain. This radiograph, if read alone, might be interpreted as being relatively erect, with no spinal unit subluxation patterns. Tropism is seen at L5–S1, with the right facet being sagittal and the left facet being coronal.

Figure 2.72 reveals normal lateral bending to the left. The spinous processes deviate to the concavity on the left and the bodies on the right.

Figure 2.73, however, is most informative; without it, misinterpretation of this spine would have occurred. In this right lateral flexion study, a Lovett reverse curve is shown. The spinous processes deviate to the convexity on the left, and the bodies deviate to the concavity on the right. Some right lateral flexion of L4 on L5, of L3 on L4, and of L2 on L3, but also marked inferiority of the right hemipelvis, is found on right lateral bending.

Figure 2.74 is a repeat right lateral bending study of the same patient as in Figure 2.73 following 2 weeks of flexion-distraction manipulation. Now the spinous processes deviate to the midline, and the pelvis no longer is inferior on lateral bending.

Study of lateral flexion in patients with herniated discs causes reflection of the role of the triple joint complex (the intervertebral disc and two facet joint pairs at a given spinal level) in low back pain (188). Disc herniation is the single greatest cause of disability; disc herniation is the most common cause of low back pain and acute sciatica (189); and disc problems are by far the most common cause of back ailments (190).

Nerve Root Origin from Cauda Equina

A discussion of the normal anatomic relationship of the nerve root origin from the dural sac and its ultimate exit via its intervertebral foramen is in order before proceeding. The adult spinal cord ends at the level of L1–L2 at the conus medullaris, continuing caudally as the filum terminale to attach at the back of the coccyx. The filum terminale is encased in dura mater to the level of S2. At each vertebral level, a pair of nerve roots leave the dural sac, with each enclosed by dural nerve root

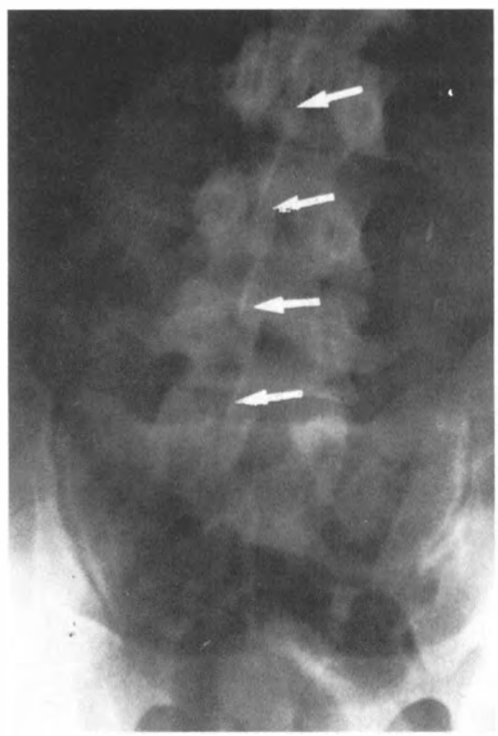

Figure 2.63. Left lateral flexion view showing Lovett failure curve with failure of the lumbar bodies to flex left and the spinous processes rotating left (*arrows*).

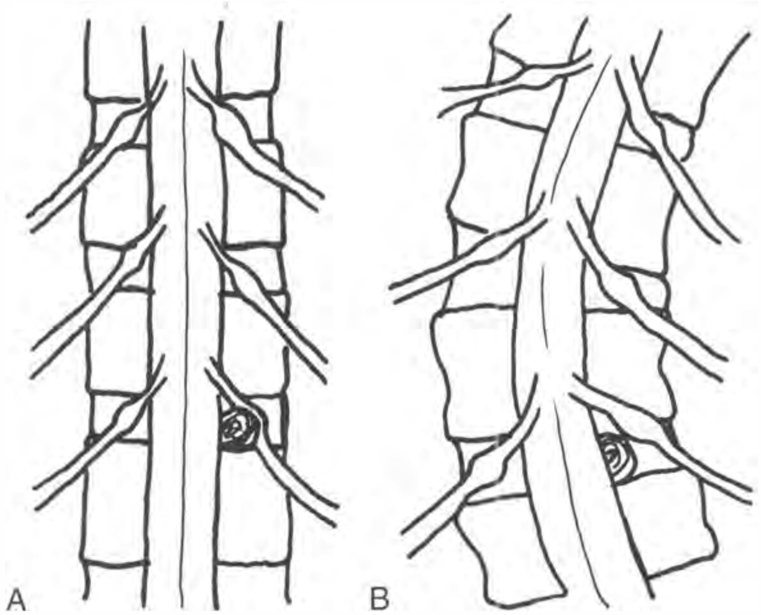

Figure 2.64. **A.** Schematic of L5 right medial disc protrusion in relation to the right S1 nerve root. **B.** The patient leans right to move the nerve root away from the disc (i.e., the patient leans into the side of the pain [right side] to relieve the pressure from an L5 disc protrusion medial to the S1 nerve root).

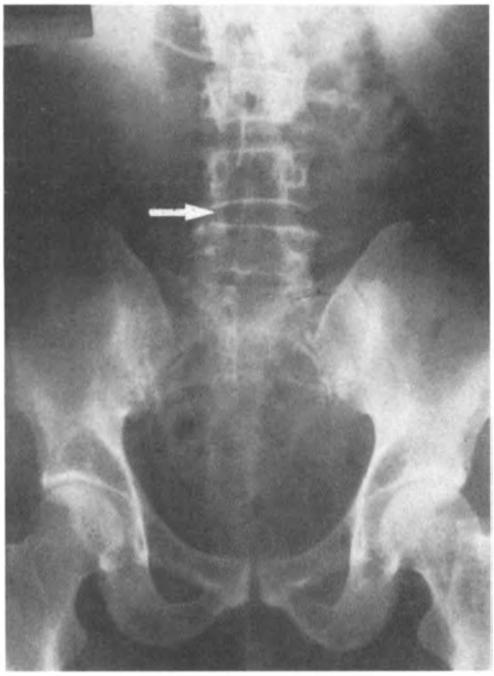

Figure 2.65. Posteroanterior view of the lumbar spine shows left lateral subluxation of L4 on L5 (*arrow*).

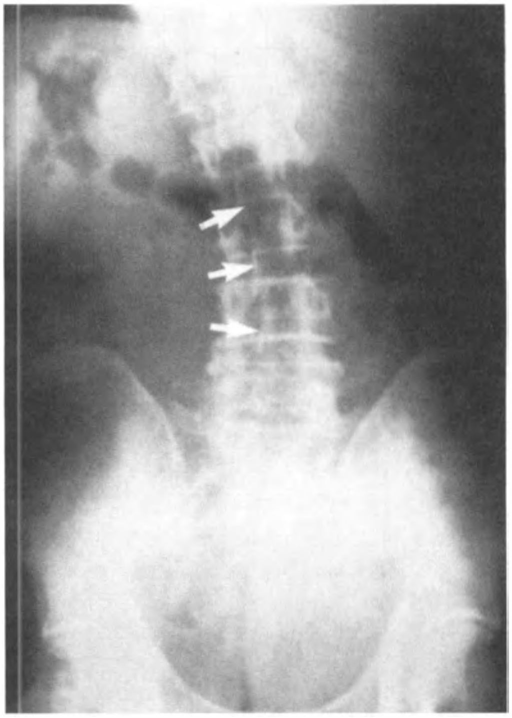

Figure 2.66. Left lateral flexion view showing normal lateral flexion mechanics. All segments have spinous process rotation into the concave side (Lovett-positive motion) (*arrows*).

Figure 2.67. Right lateral flexion view. L4 fails to laterally flex right on L5. The spinous processes do not rotate right (Lovett motion failure) (*arrows*).

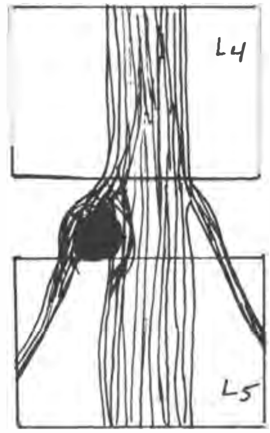

Figure 2.68. Illustration demonstrating how standing erect pulls the L5 nerve root into the L4 medial disc protrusion.

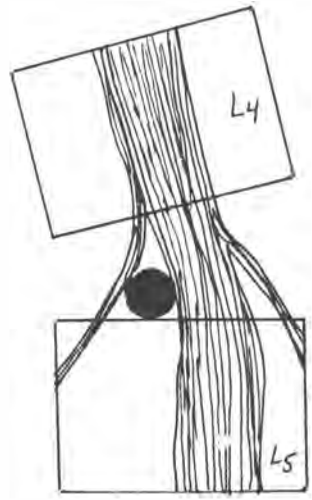

Figure 2.69. Illustration demonstrating how left lateral bending pulls the L5 nerve root away from the L4 medial disc protrusion and relieves pain.

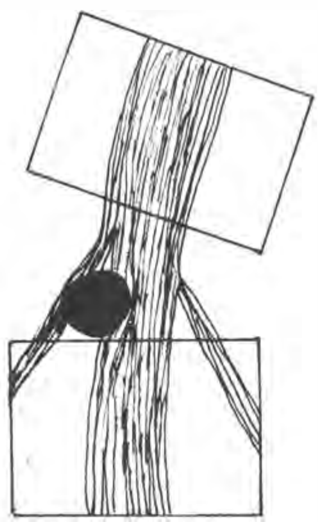

Figure 2.70. Illustration demonstrating how right lateral bending pulls the L5 nerve root into the left L4 medial disc protrusion and aggravates pain.

sleeves. In the lumbar spine, these nerve roots pass directly downward, forming the caude equina surrounding the filum terminale, until their eventual exit from each respective intervertebral foramen. The origin of the nerve root from the dural sac (cauda equina) is about one segment above the exit from its IVF. The nerve root runs down laterally to the IVF from which it exits. Specifically, the fourth lumbar root exits the dural sac at the level of the third lumbar disc to exit the IVF one vertebra below; the fifth lumbar nerve root exits the dural sac at the level of the fourth lumbar disc to exit the IVF one vertebral segment below; the first sacral root exits the dural sac at the fifth lumbar disc level, passing down to the first sacral IVF; and the second sacral nerve root lies medial to S1, originating at the lower border of the fifth lumbar disc.

From Figure 2.75, it can be seen that the L4 nerve root can be compressed at its origin and course by the protrusion of the third lumbar disc, that the L5 nerve root can be compressed by the fourth lumbar disc, and that the S1 and S2 nerve roots can be compressed by the fifth disc protrusion.

Intradiscal Pressure Changes

Pressure changes within the nucleus pulposus as they relate to postural and physiologic stresses are shown in Figures 2.76–2.78. From Figures 2.76 and 2.77, it can be noted that Déjérine triad and sitting raise the intradiscal pressure six times higher than does recumbency.

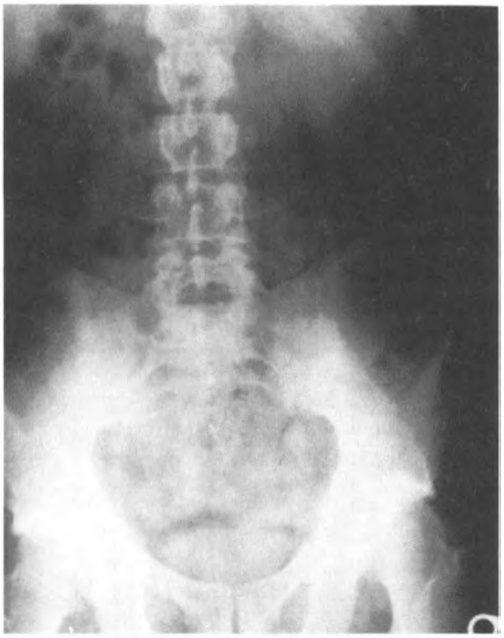

Figure 2.71. Posteroanterior neutral view of the lumbar spine that appears free of lateral curvature.

Figure 2.72. Left lateral flexion view of the patient seen in Figure 2.71. Normal Lovett-positive motion is shown with spinous processes rotated to the concave side (*arrows* on spinous processes).

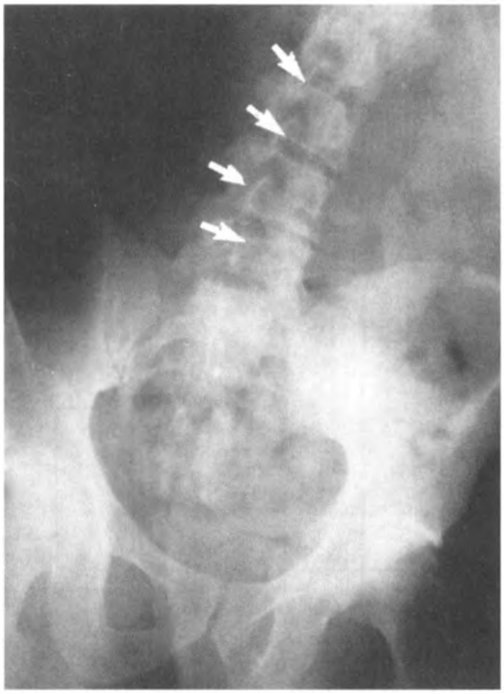

Figure 2.73. Right lateral flexion of the patient shown in Figure 2.72. Abnormal lateral movement with spinous process deviation to the convex side (Lovett negative) is shown (*arrows*). The right hemipelvis drops markedly.

Figure 2.74. Repeat view of the patient seen in Figure 2.73 following 2 weeks of Cox distraction manipulation. The right hemipelvis is level now. The spinous processes rotate to the midline instead of the convexity (*arrows*).

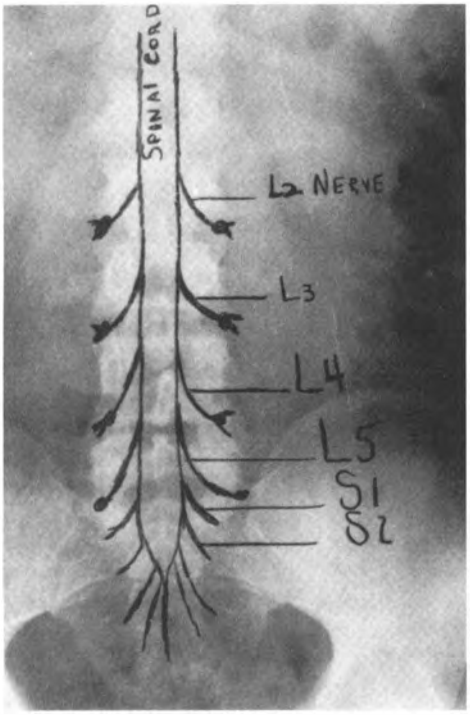

Figure 2.75. Schematic overlay of exiting cauda equina nerve roots in relation to the vertebral column and disc level.

Changes in the Intervertebral Disc

Turek (191) states that the anulus fibrosus begins to show concentric cracking and cavitation in children as young as age 15. This dehydration and cracking of the anulus can progress silently for many years, with the nucleus bulging through these cracks, causing the anulus to be thinned and weakened at its periphery. Relatively little force can cause the anulus to tear, allowing the nucleus to burst forth. Ritchie and Fahrni (192) mention that an ingrowth of vascular tissue takes place through the end plates, from the cancellous bone of the vertebral body into the nucleus pulposus. The fluid content of the nucleus decreases with increasing age until approximately 70% of the nucleus is fluid in a person at age 77, as compared with 88% in a newborn. This disc degeneration is accompanied by remodeling of the vertebral bodies. Herniation of the nucleus into the vertebral end plate at the site of vascular proliferation is termed "Schmorl's node." Rupturing of the nuclear material anteriorly and laterally results in periosteal proliferation or osteophyte formation.

This thinning of the intervertebral disc is accompanied by changes in the facet articulations as well. The facet joints lose their spacing as their articular cartilage shows degenerative changes because of the stress encountered by disc degeneration. The facets lose their gliding motion of one upon another, and the synovium undergoes hypertrophic proliferation, typically known as "osteoarthrosis." This former condition, involving the loss of intervertebral disc height and the accompanying osteophytic and subchondral sclerotic changes, has been termed "discogenic spondylosis." The latter condition, involving facet arthrosis, is consequently termed "discogenic spondyloarthrosis." Changes of the two articular facets and the disc (triple joint complex) are outlined in Figure 2.79.

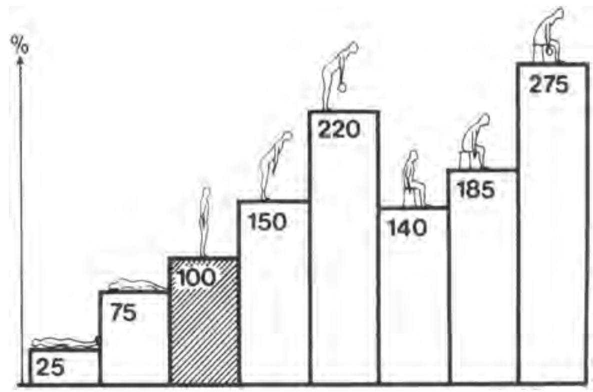

Figure 2.76. Relative change in pressure (or load) in the third lumbar disc in various positions in living subjects. (Reprinted with permission from Nachemson AL. The lumbar spine, an orthopaedic challenge. Spine 1976;1(1):61.)

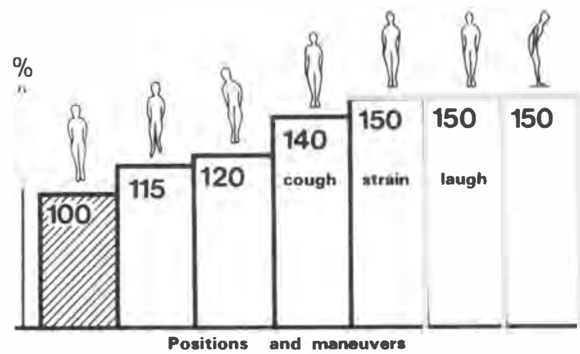

Figure 2.77. Relative change in pressure (or load) in the third lumbar disc in various maneuvers in living subjects. (Reprinted with permission from Nachemson AL. The lumbar spine, an orthopaedic challenge. Spine 1976;1(1):61.)

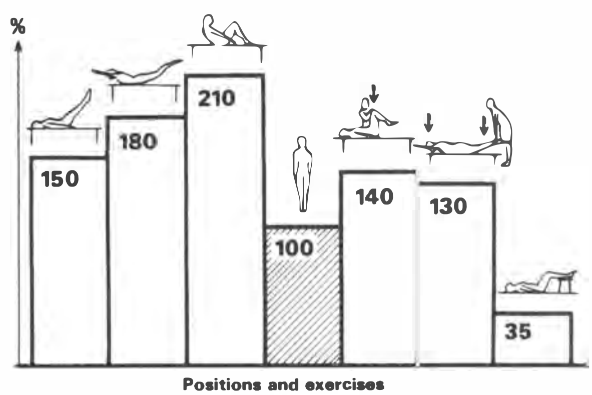

Figure 2.78. Relative change in pressure (or load) in the third lumbar disc in various muscle-strengthening exercise in living subjects. (Reprinted with permission from Nachemson AL. The lumbar spine, an orthopaedic challenge. Spine 1976;1(1):61.)

Clinical Picture of Disc Degeneration

Yong-Hing and Kirkaldy-Willis (193) describe three clinical stages in the natural history of spinal degeneration.

1. *Dysfunction.* In the beginning little pathology is demonstrated. Findings are subtle or absent, and conservative care is highly successful. Lumbago and rotatory strain are commonly diagnosed.
2. *Instability.* Abnormal movement of the motion segment of instability exists. Patient complaints are more severe, and objective findings are present. Conservative care is used and sometimes surgery is required.
3. *Stabilization.* Severe degenerative changes of the disc and facets reduce motion, and improvement may be experienced. Stenosis is now probable.

Nachemson's (5) findings agree with the second and third stages; he says that histologic signs of arthritis have been demonstrated in the facet joints late in life and always secondary to degenerative change in the disc.

It should be remembered that both the disc and the articular facet are capable of producing low back pain. It is interesting to study the work of Lora and Long (194), who were able to trace scleratogenous pain when various facet levels of the lumbar spine were irritated. L5–S1 facet stimulation resulted in referred pain to the coccyx, hip, posterior thigh, groin, inguinal ligament, and perineum; L4–L5 facet stimulation resulted in pain to the coccyx, posterior hip, and thigh; and it was less intense than that following irritation of the L5–S1 facets. L3–L4 facet stimulation resulted in pain radiating upward into the thoracic area, flank, and anterior thigh. Irritation of the articular facets at T12, L1, L2, and L3 produced no leg or coccyx sensation.

Arns et al. (195) state that the first stage of a disc lesion begins with nucleus pulposus protrusion into the outer rings of the anulus fibrosus, resulting in low back pain. This lesion is characterized by local pain that is increased by coughing and sneezing, paravertebral muscle spasm, and antalgia of the lumbar spine. Neurologic symptoms are not present. The next stage involves penetration of the nucleus pulposus into the outer rings of the anular fibers, producing pressure on the spinal nerve roots, which creates radiating pain down the leg. Neurologic signs are now present.

Farfan (51) has defined three stages of disc disease:

1. Anular bulge (protrusion).
2. Facet arthrosis as the disc thins and extrudes.
3. Stenosis if stages 1 and 2 are severe, with tautening of nerve root.

Discal thinning allows the pedicles of the superior vertebra to lower, thus compressing the nerve roots as they course toward the intervertebral foramen for emergence (196, 197). Figure 2.80 shows the normal pedicle-nerve root relationship, and Figure 2.81 shows the relationship between the narrowed disc and the pedicle compression of the nerve root. Thus, another reason can be seen for the constant back and sciatic pain before and after a surgical procedure. Note, also, the effect of short, thickened pedicles in conjunction with disc thinning, which further narrows the vertebral canal.

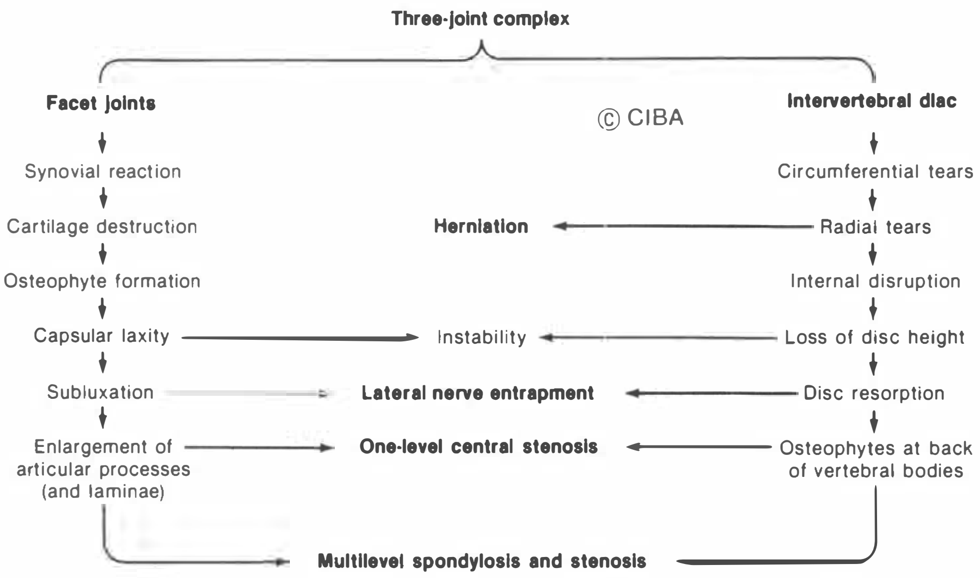

Figure 2.79. Pathogenesis of the nerve root entrapment syndrome. (Reprinted with permission from Keim HA, Kirkaldy-Willis WH. Clinical symposia. Ciba Found Symp 1980;32(6):89. Copyright 1980. Novartis. Reprinted with permission from clinical symposia, 32/6, illustrated by Frank H. Netter, MD. All rights reserved.)

Figure 2.80. Normal pedicle to nerve root distance.

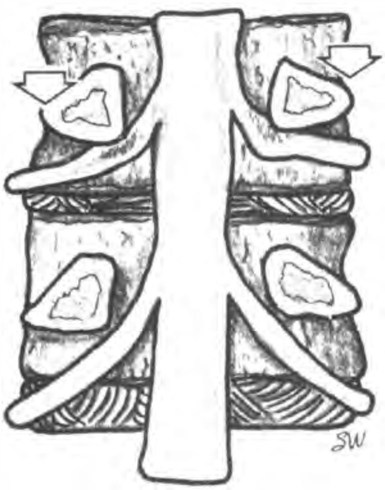

Figure 2.81. Tethering of the nerve root as the pedicle settles down upon it and as the disc space narrows or hyperextension subluxation of the superior vertebral arch occurs.

PHYSIOLOGIC AND ABNORMAL LUMBAR MOTIONS

Centrode Locations in Lumbar Kinematics

To start the discussion of lumbar mechanics, let us begin with axis motion study. The path traced by the instantaneous axis of rotation of the intervertebral disc, termed its "centrode," was studied in varying stages of disc degeneration. The centrodes of normal discs were compared with the degenerative state in 47 cadaveric spines, 22 of which were also evaluated with axial loading. The normal disc centrode fell within the posterior half of the disc space (Fig. 2.82) and averaged 21 mm in length in 10 specimens. In the earliest stages of degeneration, the centrode lengths increased significantly (average, 116 mm) (Fig. 2.83). In specimens with moderate disc degeneration, the centrode also migrated inferiorly into the L5 vertebra (Figs. 2.84–2.87). Axial loading did not appear to influence centrode length or position. This technique detected 94% of unstable spines, as compared with flexion and extension radiographs, which detected 25% of unstable spines by excessive mobility (198).

From centrode location changes in discal degeneration discussed above, one would question the concept that the nucleus pulposus moves within the anular restraints as a marble, or that it can be moved about under manipulation as such. The nuclear material seems to move out of its confines through radial anular tears in an amoeboid or pseudopodialike fashion, and its return to the interstices of the anular disc fibers must be through this same rent or tear. The escape of nuclear fluid through the tear in the anular fibers is similar to the formation of a vascular aneurysm.

The nucleus pulposus is located centrally within the posterior compartment of the disc at the juncture of the central and posterior thirds. It contains various mucopolysaccharides in the form of glucosaminoglycan, which has the ability to imbibe fluids to nine times its own volume. The nucleus fills 40 to 50% of the total disc area, and because of imbibition of fluids, it takes on a stiffness within its cells (turgor). At birth, the water content of a person's disc is 70 to 90%; the content decreases as a person ages. The intradiscal pressures drop with loss of fluid; thus, disc herniation occurs most often when the person is between 20 and 50 years of age and the intradiscal pressures are greatest.

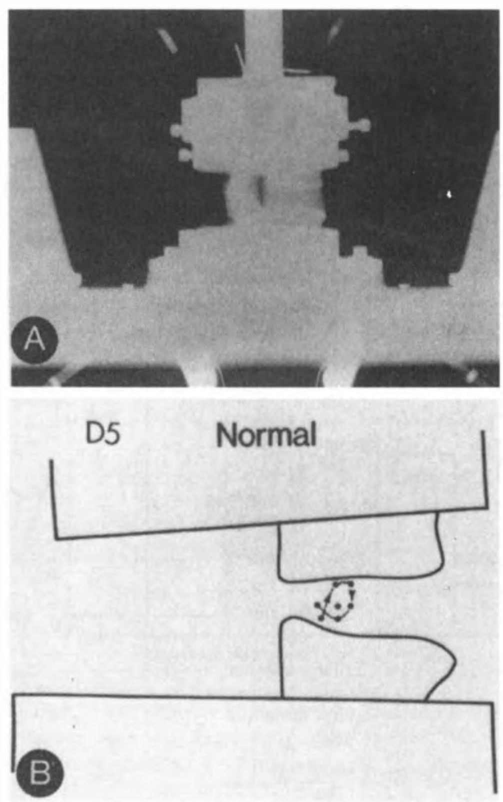

Figure 2.82. Normal spine. **A.** Radiograph. **B.** Centrode. (Reprinted with permission from Seligman JV, Gertzbein SD, Tile M, et al. Computer analysis of spinal segment rotation in degenerative disc disease with and without axial loading. Spine 1984;9(6):569.)

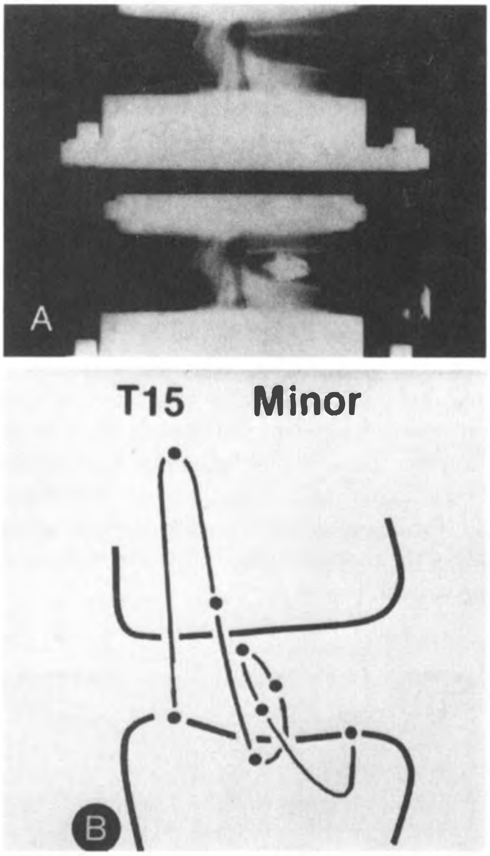

Figure 2.83. Minor degenerative disc disease. **A.** Radiograph and discogram. **B.** Centrode. (Reprinted with permission from Seligman JV, Gertzbein SD, Tile M, et al. Computer analysis of spinal segment motion in degenerative disc disease with and without axial loading. Spine 1984;9(6):569.)

The anulus fibrosus contains the nucleus pulposus by concentric laminated bands of fibrous tissue, which gradually form at the boundary of the nucleus without a sharp area of differentiation (Fig. 2.88). Sharpey's fibers attach the anular fibers to the end plates in the inner area and to the osseous tissue in the periphery.

Rotation Mechanics of the Lumbar Spine

Rotation of the lumbar spine is precluded by the action of the facet processes aligned across the path of rotation, blocking the movement. The knowledge that rotation occurs invites an explanation. Although the effective rotation contributed by each lumbar segment in the total vertebral movement not great, it adds up to a marked capability, often acknowledged only when a patient experiences its loss. Rotation primary spin is expected to occur about a center of motion dominated by the disc until the opposing facet makes contact and resists further movement across the facet plane. With increased torque, one expects a migration of the center of motion to occur toward the resisting facet and, using this as a fulcrum, a pseudospin would tend to occur as a result of lateral shear or displacement of the disc (Fig. 2.89) (199).

Rotation is felt to be a complex motion facilitated by the effective shape of the articular surface of the disc—an arcuate motion that occurs across the disc and is associated with swing in both the lateral and anteroposterior planes. The intervertebral disc is the primary articulation in the vertebral column, composed of a joint with about three degrees of freedom. This allows both spin about a mechanical axis and swing of the mechanical axis in two mutually independent directions, for example, in the anteroposterior and lateral planes (Fig. 2.90) (199).

The posterior complex, particularly the architecture of the posterior facet joints, acts as a control mechanism both to restrict the motion of the primary articulation and to control the motion to satisfy the anatomic requirements for motion of the segment while retaining segment strength and stability.

A centrode, rather than a single point, indicates that the articular surface has a varying curvature; one would expect the disc to articulate as if it were flat, and avoid diarthrosis, as shown schematically in Figure 2.91.

The disc has a potential for three degrees of freedom. Lateral flexion is accompanied by rotation in a monodal move-

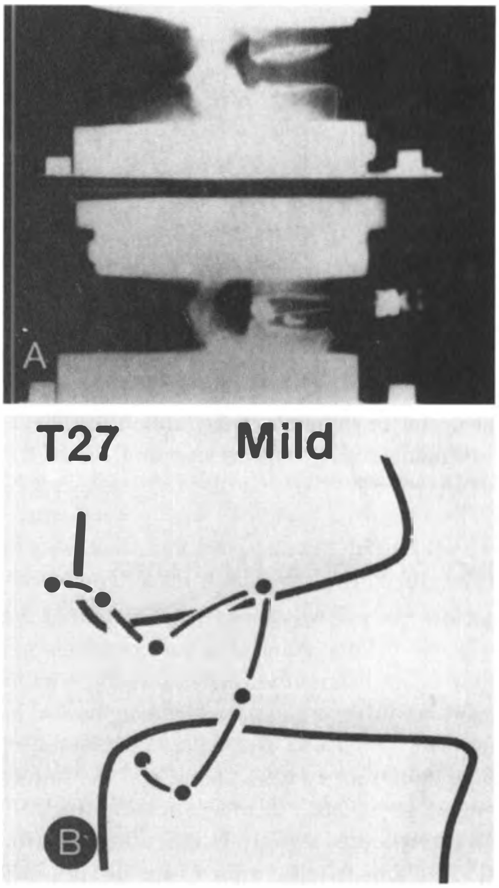

Figure 2.84. Mild degenerative disc disease. **A.** Radiograph and discogram. **B.** Centrode. (Reprinted with permission from Seligman JV, Gertzbein SD, Tile M, et al. Computer analysis of spinal segment motion in degenerative disc disease with and without axial loading. Spine 1984; 9(6):570.)

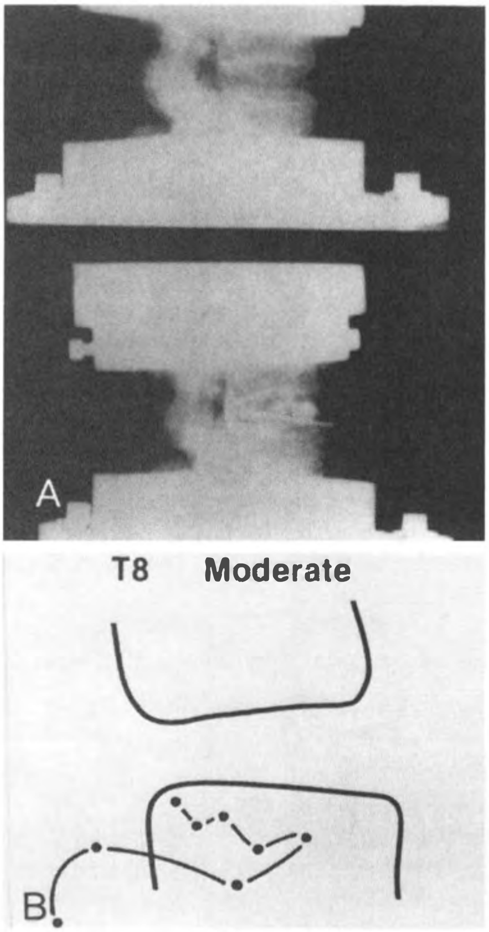

Figure 2.85. Moderate degenerative disc disease. A. Radiograph and discogram. B. Centrode. (Reprinted with permission from Seligman JV, Gertzbein SD, Tile M, et al. Computer analysis of spinal segment motion in degenerative disc disease with and without axial loading. Spine 1984;9(6):570.)

ment. The posterior elements of the motion segment cause rotatory movement during both flexion and lateral flexion, as shown in Figure 2.92.

Summary of Lumbar Mechanics

The bony parts and soft tissues of a cross section of the lumbar spine can be divided into anterior and posterior elements. The dividing line is just behind the vertebral body, with the body, the disc, and the anterior and posterior longitudinal ligaments lying anteriorly. The neural arch with its processes, the intervertebral (apophyseal or facet) joints, and the different ligaments attached to the bony elements lie posteriorly. The back muscles are distributed mainly lateral and posterior to the neural arch, but anterolateral muscles are also present (200).

Division is not merely anatomic but has a functional (mechanical) purpose. The anterior elements provide the major support of the column and absorb various impacts; the posterior structures control patterns of motion. Together they protect the dural content, which is surrounded by the neural arch.

Being synovial in nature, these joints undergo degenerative changes with aging. These changes are usually secondary to disc degeneration and, therefore, occur later in life. It is obvious that the decrease of intervertebral disc height accompanying degeneration has an effect on the apophyseal joints in stress distribution. It is germane, therefore, to postulate on the importance of mechanical factors in degenerative changes. The importance of mechanical factors to these changes is also indicated by the fact that severe osteoarthritis of the apophyseal joints is common in the presence of scoliosis, kyphosis, blocked vertebrae, spondylolisthesis, and vertebral body collapse.

Normal function of the apophyseal joints is important in stabilizing the motion segment and in controlling its movement, thus protecting the discs and ligaments. Loads applied to the lumbar spine are normally shared between the joints and discs. This load sharing can be influenced by the type of loading, the geometry of the motion segment, and the stiffness of the participating structures (200).

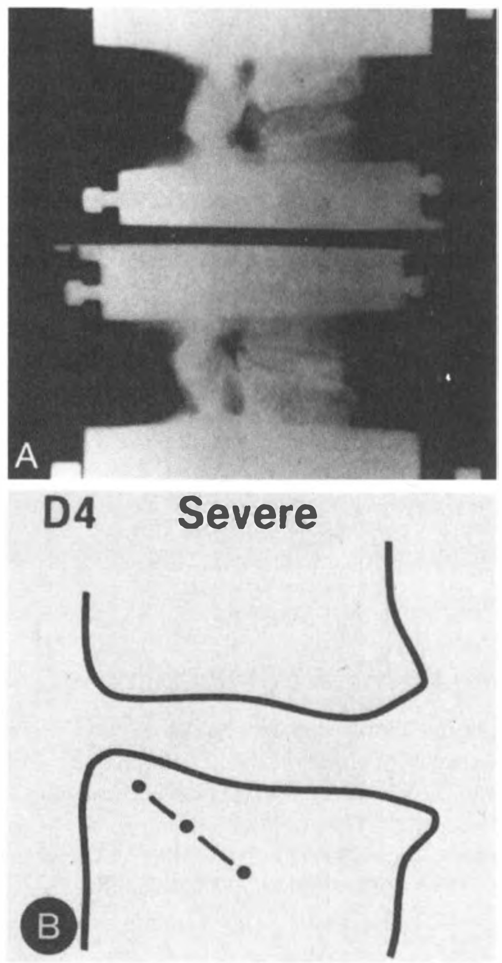

Figure 2.86. Severe degenerative disc disease. A. Radiograph and discogram. B. Centrode. (Reprinted with permission from Seligman JV, Gertzbein SD, Tile M, et al. Computer analysis of spinal segment motion in degenerative disc disease with and without axial loading. Spine 19849(6):571.)

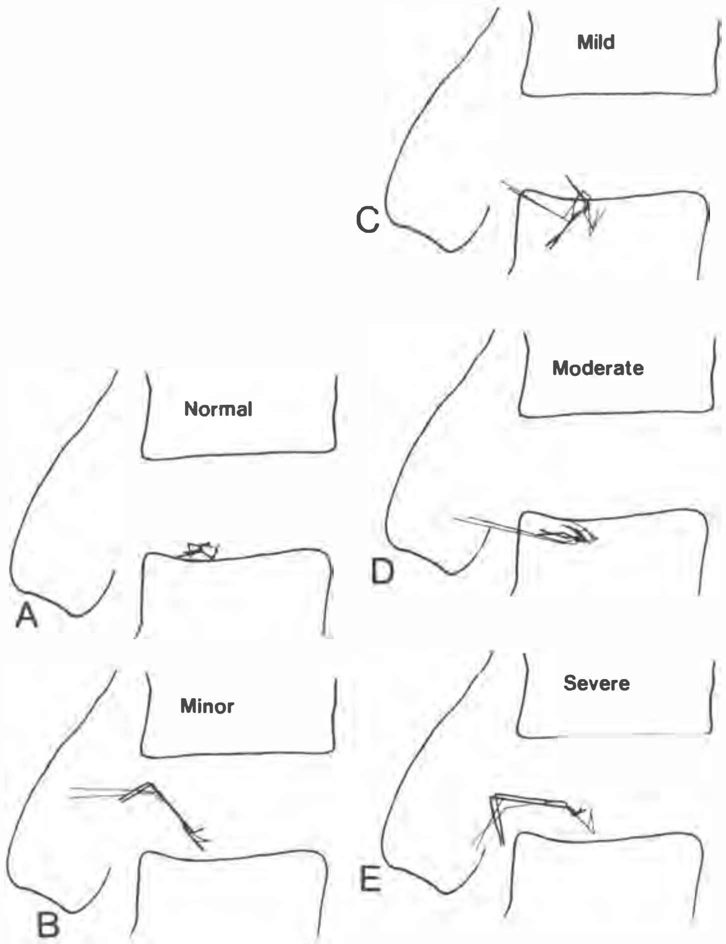

Figure 2.87. Axial loading. *Thick lines* represent centrodes from unloaded runs. *Thin lines* represent centrodes from axial-loaded runs (70 lb). On each figure all four centrodes are from the same spine. **A.** Normal spine. **B.** Minor spine. **C.** Mild spine. **D.** Moderate spine. **E.** Severe spine. The terms minor, mild, moderate, and severe refer to the degenerative state of the disc. (Reprinted with permission from Seligman JV, Gertzbein SD, Tile M, et al. Computer analysis of spinal segment motion in degenerative disc disease with and without axial loading. Spine 1984;9(6):572.)

Miller et al. (201) have reported on the manner in which the intervertebral disc and the posterior elements share loads placed on the lumbar motion segment. For their report they used a two-dimensional biomechanical model to examine this load sharing. The model incorporated two rigid bodies to represent the vertebrae and six elastic springs to represent the tissues of the IVD and the posterior elements. Compression loads were resisted almost totally by the model IVD, but both the intervertebral disc and the posterior elements contributed substantially to resisting anteroposterior shear and flexion-extension loads. Motion segment morphology was a major determinant of load sharing in the model disc response to anteroposterior shear.

Both the intervertebral disc and the apophyseal (facet) joints of low lumbar motion segments are suspected sources of low back pain. When a low back disorder occurs, pain is aggravated by some physical activities but not by others. Different physical activities impose different loads on both the disc and the facets; perhaps pain aggravation is related to those loading patterns. Hence, it is important to know how much of a load imposed on a motion segment is distributed to the IVD, how much is distributed to the apophyseal joints, and what the determinants of that distribution are.

Range of Internal Loads

Miller further states that provided facets were present, a shear force applied to the motion segment was resisted primarily by a combination of intervertebral disc shear and facet compression or tension. The portion of the overall shear resistance contributed by disc shear versus that contributed by facet tension compression depended little on how far posterior to the disc the facets were but depended greatly on their superior-inferior location. When the facets were low, almost all of that resistance was provided by shearing of the IVD. When the facets were high, each mechanism contributed substantially to

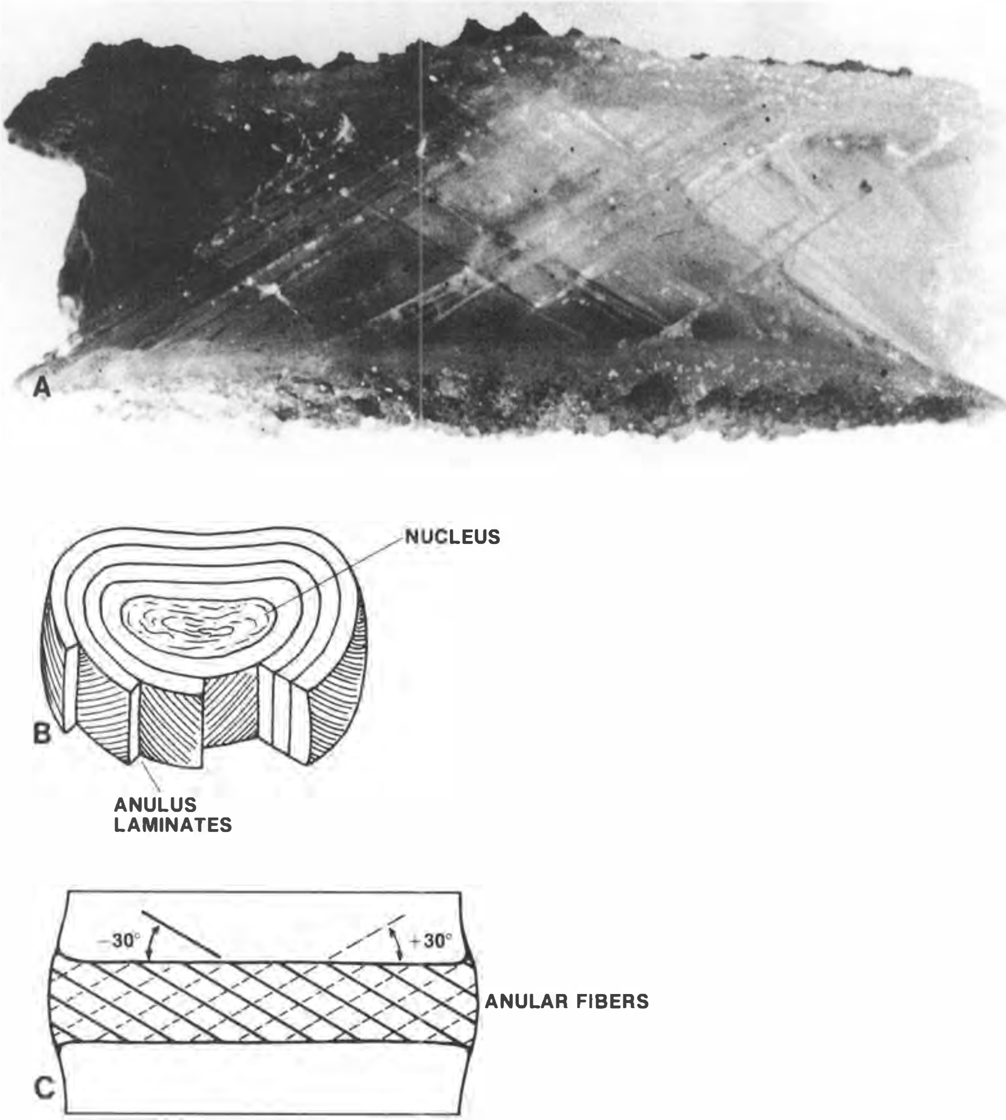

Figure 2.88. Intervertebral disc. **A.** This photograph of a disc clearly shows the annular fibers and their orientation. **B.** The disc consists of a nucleus pulposus surrounded by the anulus, which is made of concentric laminated bands of annular fibers. In any two adjacent bands, the fibers are oriented in opposite directions. **C.** The fibers are oriented at about $\pm 30^\circ$ with respect to the placement of the disc. (Photograph courtesy of Dr. Leon Kazarian.) (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: Lippincott-Raven, 1978:3.)

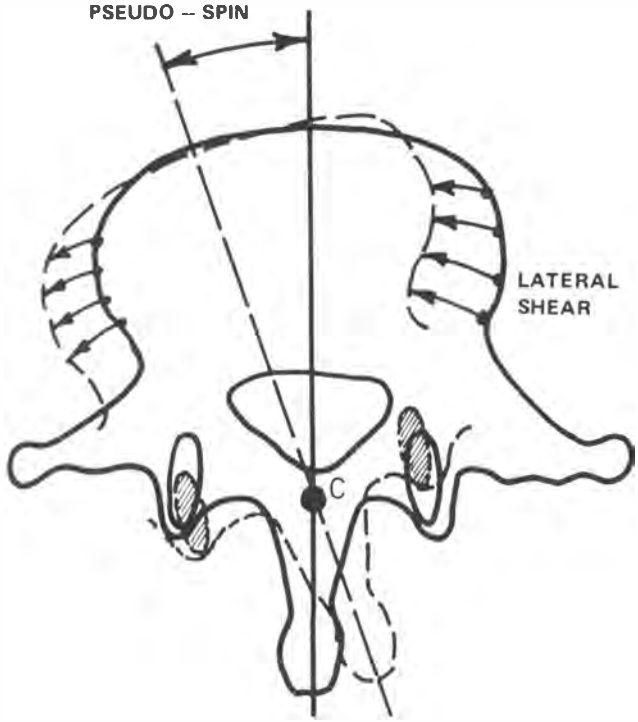

Figure 2.89. "Accessory rotation" due to lateral disc shear. (Reprinted with permission from Scull ER. Joint biomechanics and therapy: contribution or confusion? In: Glasgow EF, Twomey LT, Scull ER, et al., eds. Aspects of Manipulative Therapy. New York: Churchill-Livingstone, 1985:9–12.)

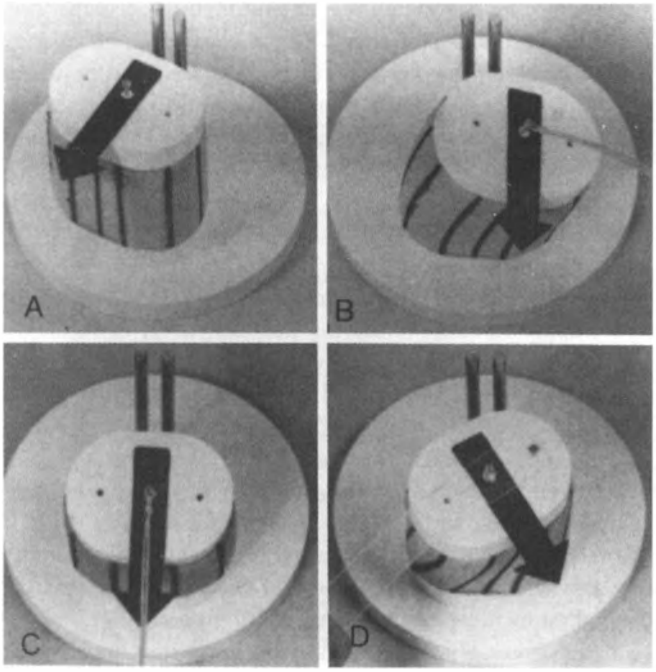

Figure 2.90. Three degrees of freedom of the isolated IV disc. **A.** Model of the isolated disc without posterior elements. **B.** Lateral swing. **C.** Anterior/posterior swing. **D.** Rotation or spin. (Reprinted with permission from Scull ER. Joint biomechanics and therapy: contribution or confusion? In: Glasgow EF, Twomey LT, Scull ER, et al., eds. Aspects of Manipulative Therapy. New York: Churchill-Livingstone, 1985:9–12.)

the total resistance. Thus, in response to a large anteroposterior shear force, both the IVD and the facet joints can be loaded lightly to moderately, or they can be loaded heavily. Which circumstance occurs seems to depend primarily on the location of the facets relative to the disc in the superior–inferior direction (201).

Facet inclination angle did not seem critical to motion segment response. When the facets were tilted 20° from the frontal plane, they were compressed 300 N at most in response to the 2500 N compression force. When the facets were tilted only 5°, they were compressed 120 N at most. That is, *facet inclination angle had only a modest effect on compression response.* In response to the 500 N shear force, changing the superior–inferior location of the facets by 2 cm caused about three times the change in load sharing between disc shear and facet inclination of 15° (201).

Conclusions

Findings (201) suggest that when loads typical of those experienced in vivo are applied to a lumbar motion segment, the following occur:

1. The apophyseal joints are not loaded heavily by compression or flexion-extension loads but can be heavily loaded by anteroposterior shear loads.

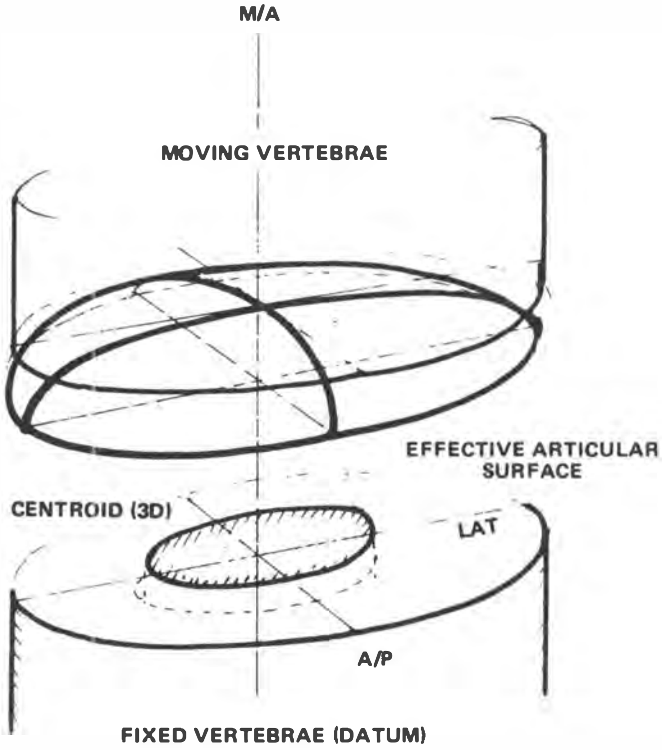

Figure 2.91. The effective articulation of the intervertebral disc. (Reprinted with permission from Scull ER. Joint biomechanics and therapy: contribution or confusion? In: Glasgow EF, Twomey LT, Scull ER, et al., eds. Aspects of Manipulative Therapy. New York: Churchill-Livingstone, 1985:9–12.)

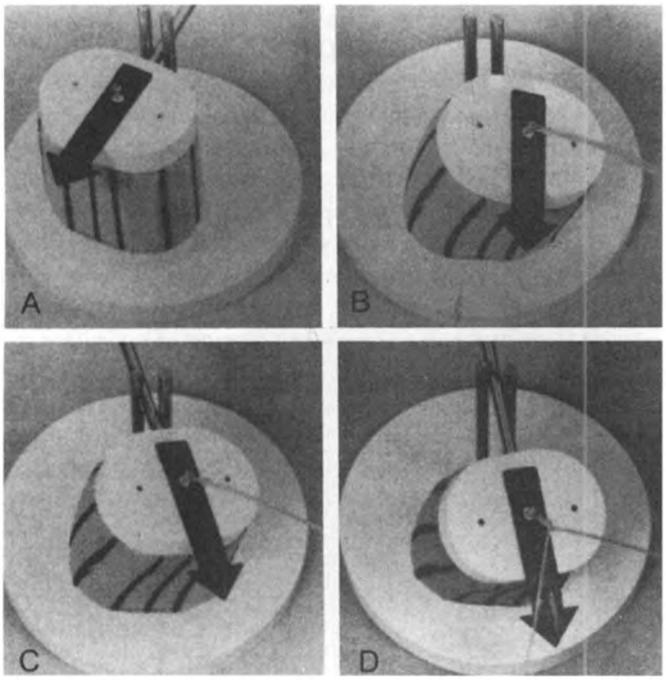

Figure 2.92. Motion of the IV disc with posterior coupling. **A.** Model of the disc with posterior coupling. **B.** Lateral chordal swing without coupling. **C.** Lateral chordal swing and adjunct rotation initiated by posterior coupling. **D.** Combined lateral and anterior/posterior (A/P) swing movement with posterior coupling. (Reprinted with permission from Scull ER. Joint biomechanics and therapy: contribution or confusion? In: Glasgow EF, Twomey LT, Scull ER, et al., eds. Aspects of Manipulative Therapy. New York: Churchill-Livingstone, 1985:9–12.)

2. Resistance developed by the apophyseal joints is not effective in relieving loads on the intervertebral disc when the motion segment is compressed. It can be effective in relieving the disc, however, when the segment is flexed, extended, or anteroposteriorly sheared.
3. In response to anteroposterior shear loads, the location of the facet joints relative to that of the intervertebral disc in the superior–inferior direction is a major determinant of what loads each structure will bear.

Pathologic, experimental, and clinical studies indicate that excessive strain concentration can occur in the posterior elements of the spine, and they can be increased by extension. These strains can cause small fractures in this region and can be responsible for episodes of back pain. Diagnosis of these fractures is usually missed.

Under compressive load, the highest compressive strains were recorded near the bases of the pedicles and deep surfaces of the pars interarticularis (202).

Experiments carried out on cadaveric lumbar spines to determine the mechanical function of the apophyseal joints (203) found that in lordotic postures the apophyseal joints resist most of the intervertebral shear force and share in resisting the intervertebral compressive force. Apophyseal joints prevent excessive movement from damaging the discs. The posterior annulus is protected in torsion by the facet surfaces and in flexion by the capsular ligaments.

Recent experiments performed on cadaveric spines have determined the mechanical properties of the apophyseal joints when they are subjected to loading regimens calculated to simulate movements and postures in life. This experimental evidence has been collated to give a concise account of the mechanical function of the apophyseal joints and to indicate under what circumstances they might sustain damage.

NORMAL DISC AND APOPHYSEAL JOINT ANATOMY AND PHYSIOLOGY

Normal Kinematics of the Lumbar Spine

Structural physiology begins with an understanding of normal spinal mechanics. Panjabi and White measured ranges of active flexion and extension, axial rotation, and lateral bending have been measured in the lumbar spines of normal volunteers in vivo, and assessed the relation between the primary and accompanying movements in the other planes (204).

Movements of flexion and extension of the L5–S1 level were greater than at the other levels. On inspection, it was apparent that some subjects flexed more than they extended at L5–S1, whereas the others extended more than they flexed. L5–S1 does not demonstrate a consistent range of motion patterns, although the total range of flexion plus extension remains similar. Lateral bending at L4–L5 is markedly limited compared with the upper three lumbar levels.

During voluntary flexion and extension, little accompanying rotation or lateral flexion is found. In axial rotation, a consistent pattern of accompanying lateral flexion is seen. At the upper three lumbar levels, axial rotation is accompanied by lateral flexion in the opposite direction. That is, if the voluntary axial rotation is to the right, the accompanying lateral bend is to the left, and vice versa. Any lateral bending occurring at L5–S1 is always in the same direction as the axial rotation (204).

Magnitude of accompanying axial rotation during lateral bending suggests that the lumbar spine is also twisted to its limit in the opposite direction during this maneuver. In voluntary axial rotation, the accompanying lateral bends were generally one half to two thirds of the full range seen in voluntary lateral bending.

The L4–L5 level is a transition point for coupled axial rotation and lateral bending. Because L4–L5 also has the greatest degree of flexion and extension in the lumbar spine, it is felt that this joint experiences higher stresses than the other lumbar levels, which provides a mechanical reason for L4–L5 to have the highest incidence of intervertebral joint pathology.

Ten degrees of lateral bending occurs in the upper three lumbar levels, whereas significantly less movement—6° and 3°—is found at the L4–L5 and L5–S1 levels, respectively.

In flexion and extension, accompanying axial rotation of 2° or greater and lateral bending of 3° or greater occur rarely, and any greater degree of rotation should be considered abnormal (204).

Weightbearing Changes in the Disc

The disc bears vertical axis weight and distributes it tangentially to the anular fibers. It also bears tensile stresses at the anular fibers during rotation motion. The nucleus bears the vertical load and the anular fibers bear the tangential load in a normal disc. Degeneration causes redistribution of the loading mechanism, with the anular fibers bearing most of the vertical load.

On compression loading, the cartilaginous end plate is most susceptible to fracture, allowing rupture of nuclear material into the cancellous bone (Schmorl's nodes). The vertebral body (Fig. 2.93) is next most susceptible to fracture. An audible crack is heard as the body gives way, occurring at compression loads of 1000 to 1700 pounds in young specimens and at as low 300 pound loads in older specimens. With the anulus intact, the disc will not compress without vertebral compression. (134).

Others (204) also observed that even if posterolateral incisions were made in the anulus fibrosus all the way to the nucleus and then loaded in compression, little change would be seen in the elastic properties of the anulus and definitely no disc herniation would occur.

Rotational Changes in the Disc

In the lumbar spine, the axis of rotation is between the articular facets in the arch of the vertebra, with the anular fibers resisting the axial shearing stresses (Fig. 2.94). On flexion and extension, the axis of rotation passes close to or within the nucleus pulposus, so that for the most part the nucleus pulposus can be considered the center of motion in a sagittal plane.

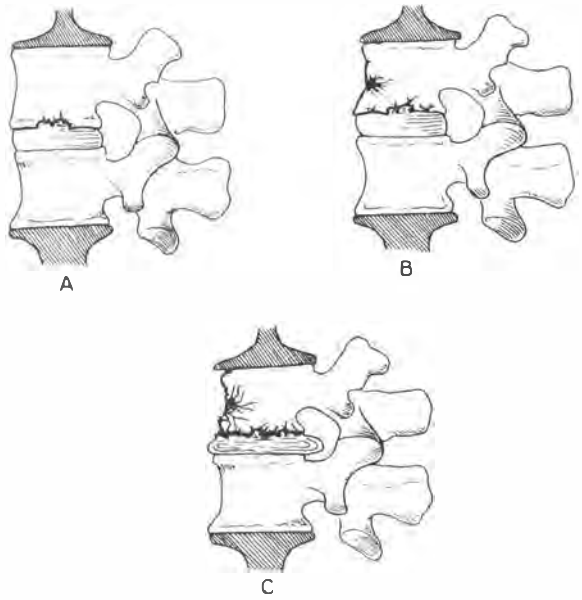

Figure 2.93. **A.** The cartilaginous end plates are most susceptible to spinal compression. **B.** The vertebral body is the second most susceptible unit of the spine. **C.** The normal nucleus pulposus and anulus fibrosus are least susceptible to pressure. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:39.)

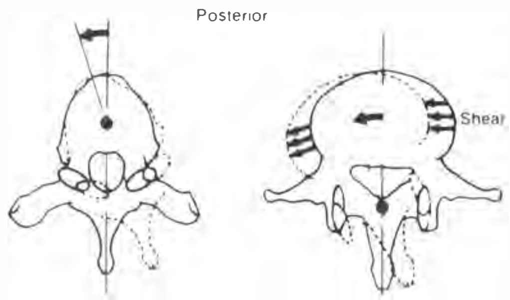

Figure 2.94. Mechanism of axial rotation in a thoracic (*left*) and a lumbar (*right*) vertebra. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:34.)

Gregersen and Lucas (206) studied axial rotation of the spine while the trunk was rotated from side to side. Approximately 74° of rotation occurred between T1 and T2, and the average cumulative rotation from the sacrum to T1 was 102°. Little rotation occurred in the lumbar spine, as compared with that in the thoracic spine; again, this is a reflection of the orientation of the facet joints. Measurements of rotation obtained during walking indicated the following (206):

1. Pelvis and the lumbar spine rotate as a functional unit.
2. In the lower thoracic spine, rotation diminishes gradually up to T7.
3. T7 represents the area of transition from vertebral rotation in the direction of the pelvis to rotation in the opposite direction, that of the shoulder girdle.
4. Amount of rotation in the upper thoracic spine increases gradually from T7 to T1.

Lumsden and Morris (207) measured axial rotation at the lumbosacral level in vivo and found that approximately 6° of rotation occurred at this level during maximal rotation. Approximately 1.5° of rotation occurred during normal walking. Rotation at L5–S1 was not measurably affected by asymmetrically oriented facets (tropism); it has always been associated with flexion of L5 on the sacrum.

White and Panjabi (141) state that the disc anulus supports two types of stress—the normal or perpendicular and the shearing or parallel. Shear stresses are greater in magnitude, and no provision is made for resisting shear stress in the way that anular fibers resist normal perpendicular stresses by the alternating anular layers. Thus, the risk of disc failure is greater with tensile loading than with compression loading.

When a disc is subjected to torsion, shear stresses occur in the horizontal as well as the axial plane. The magnitude of these stresses varies in direct proportion to the distance from the axis of rotation (Fig. 2.95). The stresses at 45° and 60° to the horizontal are shown in Figure 2.95. Shear stresses that are perpendicular to the fibers' direction may produce disc failure. The application to proper lifting (Fig. 2.96) can be considered with the above tensile stress failures.

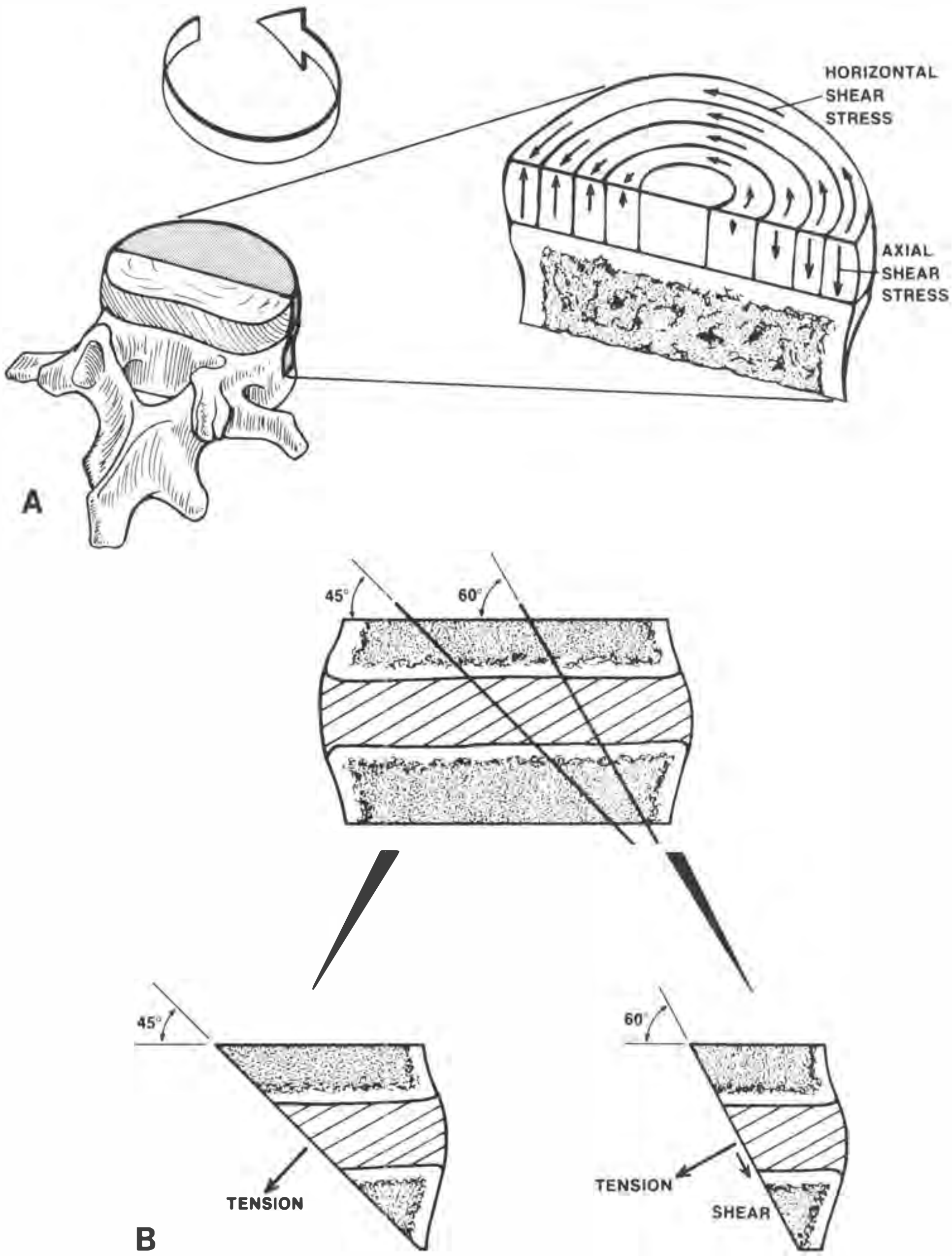

Figure 2.95. Disc stresses with torsion. **A.** Application of a torsional load to the disc produces shear stresses in the disc. These are in the horizontal plane as well as in the axial plane, and both are always of equal magnitude. They vary, however, at different points in the disc in proportion to the distance from the instantaneous axis of rotation. **B.** At 45° to the disc plane, the stresses are normal (i.e., no shear stresses). At 60° to the disc plane, perpendicular to the annular fibers, however, both types of stresses are present, normal as well as shear. The normal stresses are efficiently taken up by the annular fibers. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:16.)

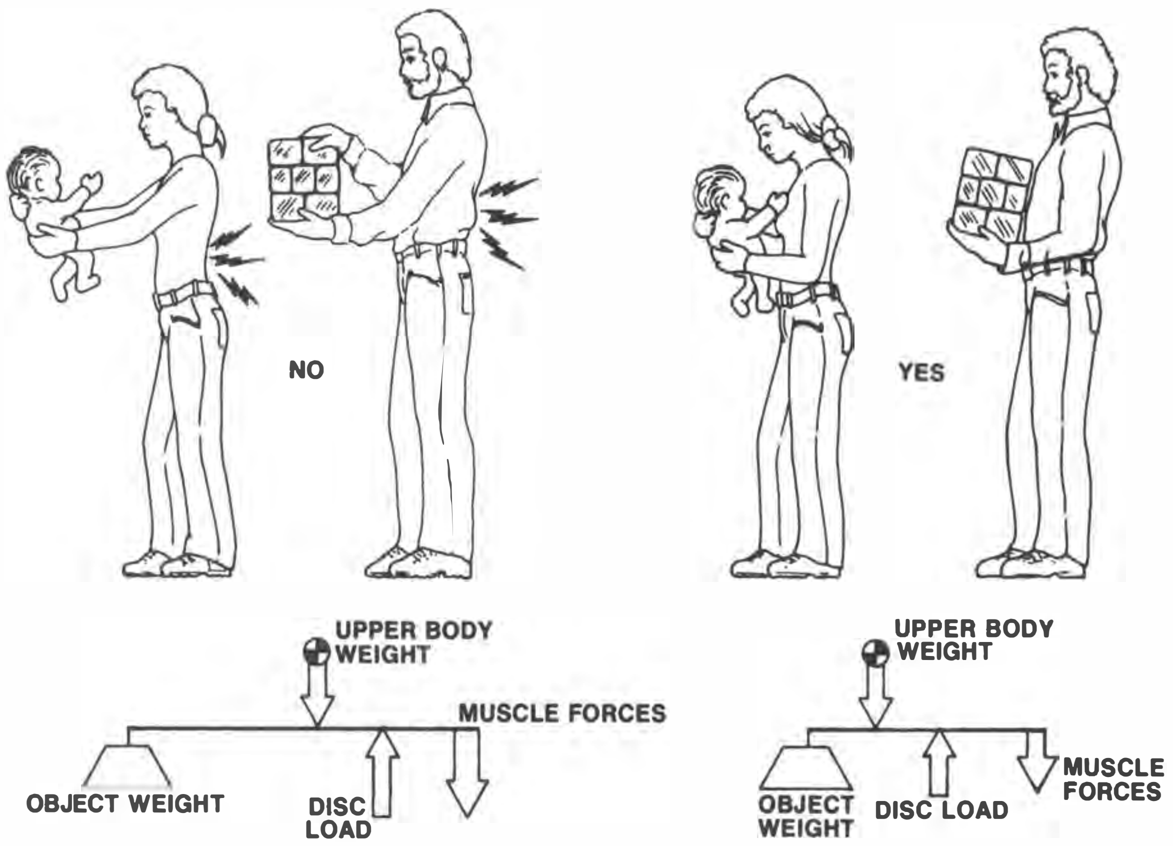

Figure 2.96. Diagram of the ergonomics of proper lifting. The load on the discs is a combined result of the object weight, the upper body weight, the back muscle forces, and their respective lever arms to the disc center. On the *left*, the object is farther away from the disc center, compared with the object on the *right*. The lever balances at the *bottom* show that smaller muscle forces and disc loads are obtained when the object is carried nearer to the disc. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:331.)

Disc Resistance to Force

Resistance to Intervertebral Shear Force

Adams and Hutton (208) report that when an intervertebral joint is loaded in shear (Fig. 2.97*A*), the apophyseal joint surfaces resist about one third of the shear force, and the disc resists the remaining two thirds. However, this passive resistance to shear is complicated by two features. First, when an intervertebral disc alone is subjected to sustained shear, it readily creeps forward. In an intact joint, this readiness to creep would manifest as stress relaxation, thus placing an increasing burden on the apophyseal joint surfaces until, in the limit, they would resist all of the intervertebral shear force. Second, the muscle slips attached to the posterior part of the neural arch brace it by pulling downward. This prevents any backward bending and brings the facets more firmly together. This means that, in the intact joint, the intervertebral disc is subjected only to pure compression and that the intervertebral shear force is resisted by the apophyseal joints, producing a high interfacet force.

Resistance to Intervertebral Compressive Force

Absence of a flattened articular surface in the transverse plane at the base of the articular facets clearly suggests that apophyseal joints are not designed to resist intervertebral compressive force. Experiments (208) confirm that, provided the lumbar spine is slightly flattened (as occurs in erect sitting or heavy lifting), all the intervertebral compressive force is resisted by the disc. However, when lordotic postures, such as erect standing, are held for long periods, the facet tips do make contact with the laminae of the subadjacent vertebra and bear about one sixth of the compressive force (Fig. 2.97*B*).

Contact may well be of clinical significance, because it will result in high stresses on the tips of the facet and, possibly, nipping of the joint capsules (Fig. 2.98). Perhaps this is why standing for long periods can produce a dull ache in the small of the back that is relieved by sitting or by using some device, such as a bar rail, to induce slight flexion of the lumbar spine. Disc narrowing results inasmuch as 70% of the intervertebral compressive force being transmitted across the apophyseal joints.

With increasing extension of an intervertebral joint, the compression force transmitted across the apophyseal joints increases, and it is likely that the extension movements are limited by this bony contact. Thus, it is possible that hyperextension movements could cause backward bending of the neutral arch, eventually resulting in spondylolysis, but again only as a fatigue fracture.

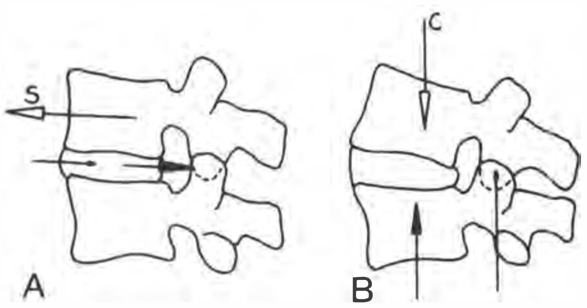

Figure 2.97. The apophyseal joint and the intervertebral disc share in resisting shear (S) and compression (C). (Reprinted with permission from Adams MA, Hutton WC. The mechanical function of the lumbar apophyseal joints. Spine 1983;8(3):328.)

Resistance to Flexion

Capsular ligaments of the apophyseal joint play the dominant role in resisting flexion of an intervertebral joint. In full flexion, as determined by the elastic limit of the supraspinous and interspinous ligaments, they provide 39% of the joint's resistance. The balance is made up by the disc (29%), the supraspinous and interspinous ligaments (19%), and the ligamentum flavum (13%) (208).

Effects of Posture on the Lumbar Spine

Current ideas on what constitutes "good posture" are rather vague. The usual advice, possibly based on esthetic and military traditions, is to "sit up straight" and "don't slouch." Paradoxically, sitting up straight is taken to mean sitting with a lumbar lordosis and not allowing the lumbar spine to flex and flatten its curve (209).

As far as the lumbar spine is concerned, no reliable evidence indicates that sitting up straight is, in fact, beneficial. On the contrary, population studies have shown that lumbar disc degeneration is rare among people who habitually sit or squat in postures that flatten the lumbar spine. Such postures are instinctively assumed by children and by many adults. If these natural preferences are to be discouraged and advice given on posture, then such advice should be founded on scientific evidence.

Posture and the Loading of the Apophyseal Joints

Apophyseal joints stabilize the spine and protect the discs from both excessive flexion and axial rotation. They also play a major role in resisting shear and compressive forces, although this varies considerably with posture.

In the erect posture, the apophyseal joints resist most of the shear force acting on the spine, as well as about 16% of the compressive force. The resulting stress between the articular surfaces is concentrated in the lower margins of the joint. If the disc is unusually narrow and degenerate, the facets can come into close apposition and then resist up to 70% of the compressive force on the spine.

In the flexed posture, the apophyseal joints resist the shear force but now play no part in resisting the intervertebral compressive force. Stress between the articular surfaces is lower than in the erect posture, and it is concentrated in the middle and upper parts of the joint. In the flexed posture no extra-articular impingement occurs (209).

Posture and the Loading of the Intervertebral Disc

Intervertebral discs and vertebral bodies comprise the main weightbearing column of the lumbar spine. Posture affects the way this column resists the loads applied to it but has little effect on the magnitude of these loads.

Under load, an unwedged disc tends to behave as a hydrostatic body exerting a uniform compressive stress on the vertebral end plates. By wedging a disc, this is complicated slightly: young nondegenerate discs remain hydrostatic, but mature and degenerate discs sustain pressure gradients. This means that when a mature disc is wedged in the erect posture, the highest compressive stresses are transmitted through the posterior anulus and the lowest through the anterior anulus. Similarly, in flexed postures the highest compressive stresses are transmitted through the anterior anulus and the lowest through the posterior anulus (209).

Fluid flow is caused by pressure changes on the disc. High pressure causes fluid to be expelled from the disc, whereas low pressure (e.g., lying down) allows the proteoglycans in the disc to suck in fluid from surrounding tissue. Flexed postures increase this fluid exchange because they cause more fluid to be expelled from the disc than do erect postures.

Flexion Effects on the Facet and Disc

Advantages and disadvantages of flexing the lumbar spine are summarized here. Let us first consider the advantages (209).

Advantages

Reducing the high stresses that can be found on the tips of the facet joints may well be significant. In a lordotic posture, the stress between the facet surfaces can exceed the peak levels found in the articular cartilage of the hip and knee, and it may be responsible for the high incidence of osteoarthritis in these joints. Advantages of flexion include:

- Reduced stresses at the apophyseal joints
- Reduced compressive stress on the posterior anulus
- Improved transport of disc metabolites
- High compressive strength of the spine

Disadvantages

The disadvantages of flexion include:

- Increased compressive stress on the anterior anulus
- Increased hydrostatic pressure in the nucleus at low load levels

How Do Discs Absorb Compressive Loads?

Discs absorb shock by squeezing fluid out of the nucleus and by allowing the fibers of the outer shell to stretch. Studies of disc

fibers suggest that they have only limited elasticity and can only stretch to 1.04 times their initial length before suffering irreparable damage. When the disc is compressed, for instance when we lift a heavy object or jump from a great height and land on our feet, this limited elasticity does not present a major problem. Indeed, when we are standing upright, the disc fibers can take 10 times as much compression as can the vertebrae themselves, so a heavy load will crush bones before it ruptures a disc.

Disc fibers are less able to cope with torsion than with compression because with torsion the stress concentrates at points of maximal curvature. Because the disc shell is made of layers of fibers that lie obliquely to each other in a crisscross pattern, torsion tends to shear one layer from another, further weakening the total structure. As a result, we stand a much greater risk of damaging our discs when we try to lift an object and twist our body around at the same time.

Sitting and Its Effects on the Intervertebral Disc

Intradiscal pressure within the nucleus pulposus is lowest when the patient is recumbent and is highest when the patient is sitting in a flexed position. Nachemson (5) has measured the relative pressure within the third lumbar disc of people in various positions and has found that these pressures range between 25 and 275 as the person moves from the recumbent to the sitting flexed posture.

Fahrni (210) studied a jungle population in India who squat rather than sit and sleep on the ground rather than in beds. These people had no concept of posture principles whatsoever but had a zero incidence of back pain. Furthermore, radiographs of the lumbar spine in 450 of these people, aged 15 to 44 years, showed no incidence of disc narrowing. Thus, sitting is to be avoided in treatment of low back pain, especially with intradiscal involvement.

Fromelt et al. (211) found that bending, twisting, and lifting were the most common causes of low back pain and disc injury. The effect of rotational instability on the lateral recess is shown in Figure 2.99.

Osmotic Principles of the Disc

The human intervertebral disc acts as an osmotic system. Water, salt, and other low-molecular weight substances penetrate the cartilage plates and anulus fibrosus. Content of water, sodium, potassium, and ashes in different regions of 69 human

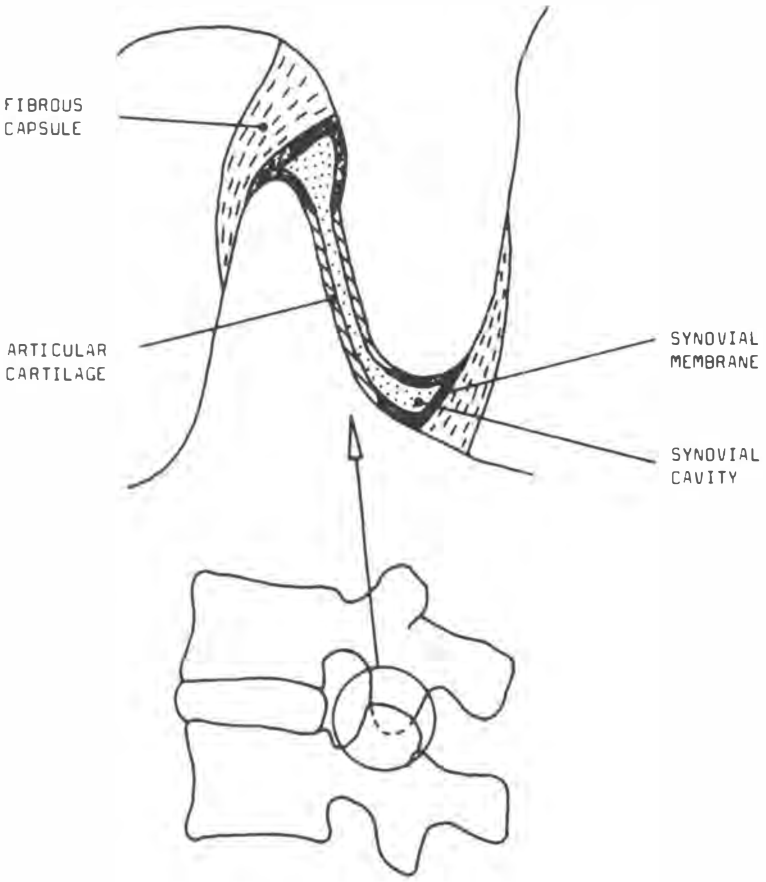

Figure 2.98. An apophyseal joint cut through in the sagittal plane. (Reprinted with permission from Adams MA, Hutton WC. The mechanical function of the lumbar apophyseal joints. Spine 1983; 8(3):328.)

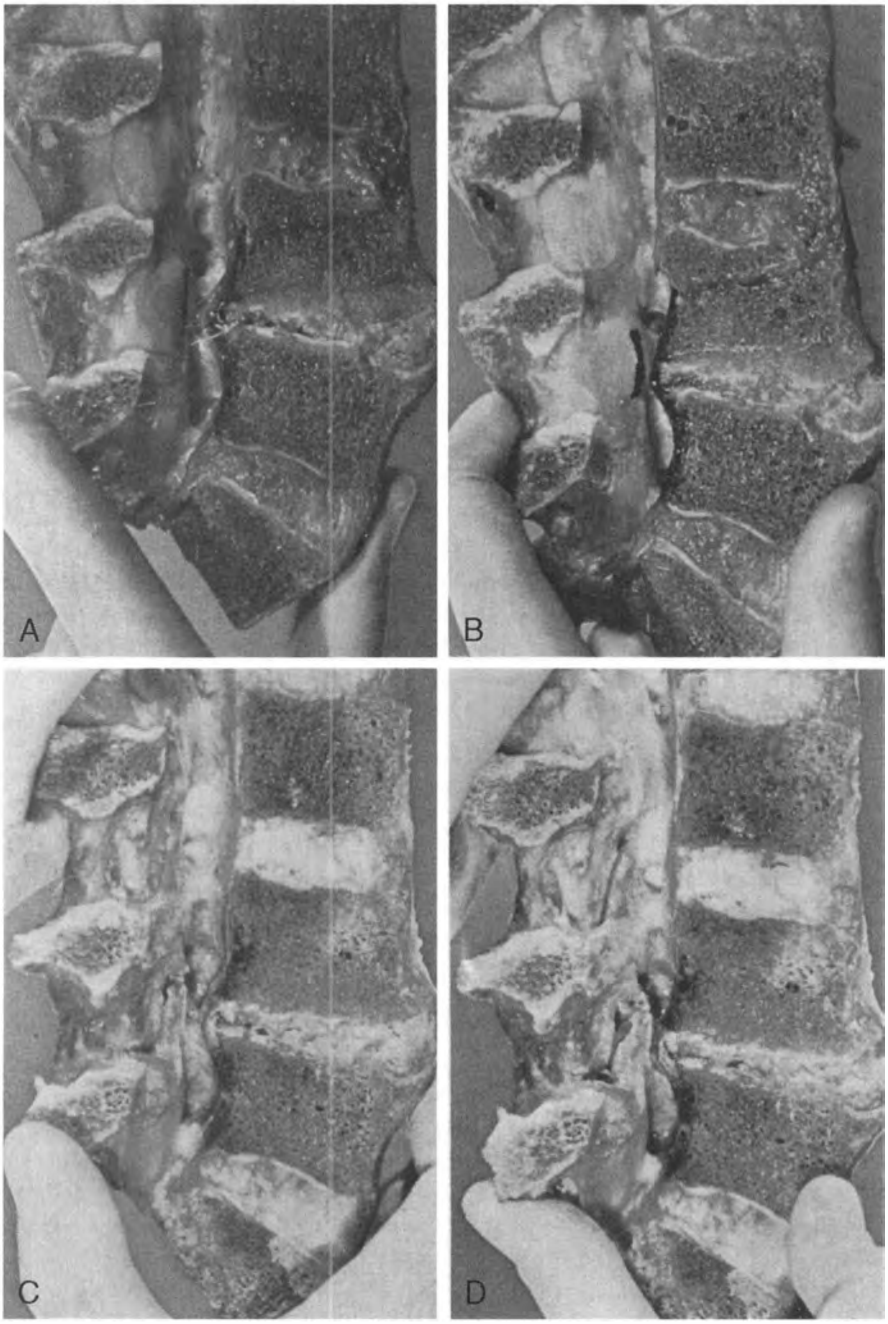

Figure 2.99. Longitudinal section of the lumbar spine. The posterior joint and disc at L3–L4 are normal. Those at L4–L5 show marked degenerative changes with rotational instability. **A.** Before rotation. The *black line* on the left is placed over the front of the superior articular process. Note the size of the lateral recess. **B.** Same specimen. The spinous process of L5 has been rotated out of the picture (toward the viewer). This rotation displaces the superior articular process forward with narrowing of the lateral recess. **C.** Same as **A,** with the lateral extension of the ligamentum flavum removed. Note the marked degeneration of the posterior joint and disc and the size of the lateral recess. **D.** Same as top right, with the lateral extension of the ligamentum flavum removed. The spinous process of L5 has again been rotated as in **B.** The posterior joint surfaces are separated. The lateral recess is narrowed by forward displacement of the superior articular process. (Reprinted with permission from Yong-Hing K, Reilly J, Kirkaldy-Willis WH. The ligamentum flavum. Spine 1976;1(4):232.)

lumbar intervertebral discs was examined before and after being loaded with certain weights. Under load, the disc loses water (anulus, 11%; nucleus, 8%) and gains sodium and potassium. The higher concentration of electrolytes in the disc after a long period of loading increases its osmotic absorption force and enables the disc to hold back the remaining water, even against a considerable pressure. After reduction of pressure, water is quickly reabsorbed, and the disc gains height and volume. The pumping mechanism maintains the nutritional and biomechanical function of the intervertebral disc (212).

Suspension Effects on the Lumbar Spine

Radiographic investigation of the lumbar spine was done in the standing and suspended position in 100 healthy adult male volunteers. Spinal and external morphology were studied. The aim of this work was to identify correlations between the modifications of shape and size of the suspended lumbar spine and external morphology. Such correlations were sought to establish a functional approach to anthropometry. This study demonstrated that the suspended position led to lengthening of the spine in 70% of the subjects examined, shortening of the spine in 22%, and mainly straightening of the spine in 8% (213).

The phenomenon of elongation of the lumbar spine when the body is placed in the suspended position is dependent on tonic muscle activity. Shortening of the lumbar spine in the suspended position was seen in apparently longitypic and thin subjects. This somatotype has been linked to "tonic" temperament. Straightening of the lumbar spine without lengthening under the effect of suspension was observed in subjects with relatively high body weight and accentuated lumbar curvature.

In 70% of the subjects studied, increased size of the intervertebral spaces was seen when the body was placed in the suspended position (i.e., by a traction force of approximately 40 to 50% of body weight). The results may have practical applications in the use of therapeutic traction. Indeed, in this respect our results underline the need to obtain muscle relaxation and show that mild traction may be effective. Furthermore, elimination of lordosis is not proof of the efficacy of traction on the intervertebral discs. Longitypic subjects may be more resistant to traction compared with other somatotypes (213).

ANATOMIC AND DEMOGRAPHIC FACTORS IN LOW BACK PAIN

Standardized tests were administered to 321 men, aged 18 to 55 years, to determine height, weight, Davenport index, leg length inequality, flexion and extension torques, flexion/extension balance, range of motion, straight leg raising, and lumbar lordosis. A total of 106 (33.0%) had never experienced low back symptoms; 144 (44.9%) had or were having moderate low back pain (LBP); and 71 (22.1%) had or were having severe low back symptoms. These three subgroups showed no significant differences in height, weight, Davenport index, lumbar lordosis, or leg length inequalities. LBP patients had less flexor and extensor strength and were flexor overpowered; they had diminished range of motion for spinal extension and axial rotation ($P = 0.003$, $P = 0.0005$), and had diminished straight leg raising capacity ($P = 0.004$). A multivariate correlation matrix demonstrated no typical pattern of associated abnormalities except that a diminished spinal range of motion in one plane was associated with the anticipated diminishment in all other planes of motion, and often with greater restrictions of straight leg raising tests (214).

In another study, men with a height of 180 cm or more showed a relative low back pain risk of 2.3 (95% confidence limits, 1.4 to 3.9), and women with a height of 170 cm or more showed a relative risk of 3.7 (1.6 to 8.6), compared with those who were more than 10 cm shorter (1.0). In men, but not in women, increased body mass index proved to be an independent risk factor for herniated lumbar disc, whereas triceps skin fold thickness had no predictive significance. Height and heavy body mass may be important contributors to the herniation of lumbar intervertebral discs (215).

Measurements made from plain lumbar radiographs were used to compare the size and shape of the lumbar vertebral canals between various categories of occupation and work load among 77 men and 118 women with a history of low back pain. The mean anteroposterior foraminal diameters proved to be wider in female farm workers than in other women, especially in the vertebrae L1 to L3 (17.1 versus 15.4 mm). However, men performing heavy manual work had smaller anteroposterior foraminal diameters than the men whose work involved less physical labor (difference at L5, 9.4 versus 10.8 mm). Female farm workers were found to have shorter interarticular distances than females in other occupational groups. In the men who reported working in stooped postures or who reported lifting and carrying heavy objects at work, the interarticular distances were wider than in men who had no such exposures (216).

Correlation of Age, Weight, Height, and Body Curve to Low Back Pain

Correlations of age, height, weight, lordosis, and kyphosis with noninvasive spinal mobility measurements were studied in 301 men and 175 women, aged 35 to 55 years, who suffered from chronic or recurrent low back pain. Correlations of the different spinal movements with the degree of LBP were analyzed, with corrections for these relationships. Age had significant indirect correlations with most of the mobility measurements, but the effect of height was minor. Weight had considerable negative correlations with the mobility measurements, except lateral flexion. Lordosis and kyphosis has significant relationships with mobility in the sagittal and frontal planes. Thoracolumbar mobility had a higher correlation with LBP than mobility of the lumbar spine. Thoracal spinal mobility alone also correlated with LBP. Lateral flexion and rotation, except for rotation in women, had stronger relationships than forward flexion and extension with LBP (217).

Eighty percent of all 30- to 60-year-old inhabitants of Glostrup, a suburb of Copenhagen (449 men and 479 women) participated in a general health survey, which in-

cluded a thorough physical examination relating to the lower back. The examination consisted of anthropometric measurements, flexibility/elasticity measurements of the back and hamstrings, and tests for trunk muscle strength and endurance. The main findings were that good isometric endurance of the back muscles may prevent first-time occurrence of low back trouble (LBT) in men and that men with hypermobile backs are more liable to contract LBT. Recurrence or persistence of LBT correlated primarily with the interval since last LBT episode: the more LBT, the shorter the intervals had been. Weak trunk muscles and reduced flexibility/elasticity of the back and hamstrings were found as residual signs, particularly among those with recurrence or persistence of LBT in the follow-up year (218).

In all, 28.9% of the sample (29.8% of the men and 27.9% of the women) had leg length discrepancies equal to or greater than 1 cm. The leg length discrepancy showed no significant predictive power for first-time occurrence of LBT in the follow-up year or for recurrence or persistence of LBT. When tested in relation to whether that subject ever had LBT prior to the initial examination, however, the group with prior LBT was found to contain significantly more participants with unequal leg length than the group with no prior LBT ($x^2 = 9.19$, $df = 1$, $P = 0.0025$). Of those with LBT, 46% (264 of 569) had unequal leg length. This figure was of the same magnitude in all eight sex/age groups. Neither the magnitude of the inequality nor whether the right or left side was shortest was found to provide any additional information regarding LBT (218).

Children's Incidence of Low Back Disc Herniation

Herniated discs in children and adolescents can be extremely disabling and difficult to diagnose because of the paucity of neurologic abnormalities and the consequent suspicions of hysteria.

One percent of patients operated on for discal herniation are between 10 and 20 years of age. Spinal fusion should be considered when discal herniation is complicated by transitional vertebrae and spondylolisthesis, which because of instability contribute to the persistence of back pain (219).

One study reported on 25 teenagers with herniated lumbar intervertebral discs with accompanying structural anomalies (219). Three had transitional vertebrae; 11 had spinal stenosis confirmed at surgery; one had tropism; and one had spondylolysis. The unusual frequency of transitional vertebrae dominates all of the cases reviewed. When associated with hyperlordosis, which also seriously compromises the mechanical efficiency of the spine, the result is often residual back pain and disability.

The exact incidence of lumbar disc herniation in children is unknown. In white patients, the percentage varies from 0.8% in one series to 3.8% in another. In Japan, the frequency is unusually high, from 7.8% to 22.3%, possibly related to earlier ages of employment (219).

Disc Anular Fiber Damage and Pain Production

Discs of 25 specimens of human lumbar motion segments were subjected to an internal division of the anulus fibrosus, sparing only a peripheral layer 1 mm thick. Thus, an attempt was made to simulate an internal disruption of the anulus caused by a traumatic episode or a degenerative process. The disc bulge that developed at the site of the injury was observed under axial compression fracture and after intradiscal injection. Under a 1000 N load, the bulge amounted to less than 0.5 mm; typically, it increased to less than 1.0 mm after fracture. An extrusion of disc material at the site of the anulus injury was never observed. The results suggest that a radial division of the anulus is not sufficient to produce a clinically relevant disc herniation; further prerequisites are a fragmentation of the disc material and a separation from the end plates (220).

Anular tears can, by nociceptor nerve endings in the anulus fibrosus, cause pain referral to the low back, buttock, sacroiliac region, and lower extremity even in the absence of neural compression (221).

WHAT ARE THE LIMITING ANATOMIC STRUCTURES (DISC, FACET, LIGAMENTS) TO TORSIONAL ROTATION MOTION OF THE LUMBAR SPINE?

Intact Anulus Fibers Restrict Axial Rotation More Than Facets

Intact anulus fibers in young discs without degeneration restricted axial rotation more than the facets in a study on 12 lumbar motion segments in which six had the anular fibers dissected in one direction while the oppositely directed fibers were left intact. In six segments, bilateral facetectomy was performed and the segment loaded in torsion. The effects of rotation were studied in each situation. The fibers behave as tendons, which explains the formation of peripheral rim lesions and circumferential tears which are considered to be the first signs of disc degeneration (222).

Axis of Rotation Determines Limiting Force to Rotation

The anulus fibrosus is the most effective structure in resisting torsion in an intact lumbar spine (223). The axis or rotation of the lumbar motion segment determines whether the disc or facet is the primary resistant structure to rotational stress. Figure 2.100 shows that the axis of rotation near the facet joints and the anulus would be the primary structure in resisting rotational forces. Figure 2.101 shows the axis of rotation to be within the disc anulus, which means the facet structures resist rotation (223).

Figure 2.102 shows the entire lumbar spines (T11 to S1) of cadavers placed into a torsion system with vertical markers

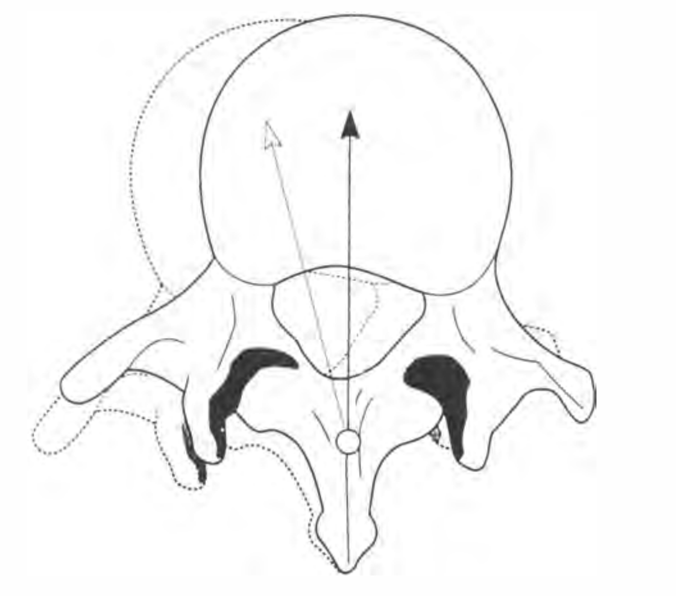

Figure 2.100. Diagram of the spine with the axis of rotation near the facet joints. The distance (*moment arm*) from the axis of rotation to the anulus can be appreciated. Note the rotatory motion of the anulus (*dotted lines*) when the axis is posterior to the anulus. If the axis of rotation were in this location, the anulus would be the primary structure in resisting rotation. (Reprinted with permission from Haher TR, Felmy W, Baruch H, et al. The contribution of the three columns of the spine to rotational stability, a biomechanical model. Spine 1989;14(7):663–670.)

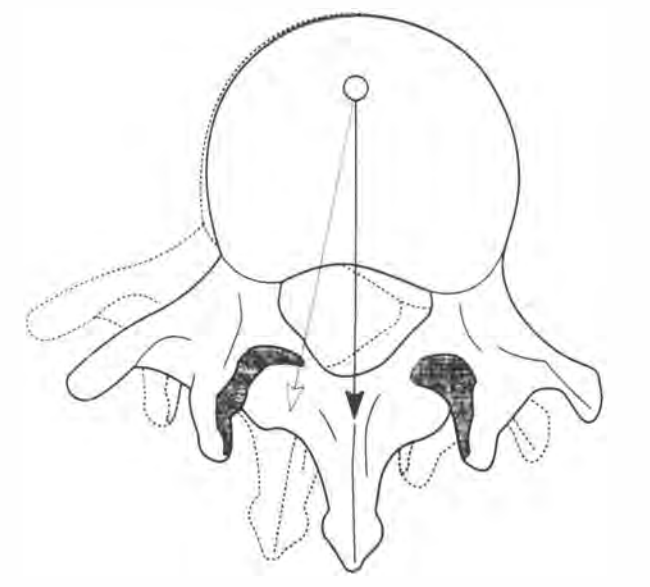

Figure 2.101. Diagram of the spine with the axis of rotation in the anulus. The distance from the axis to the facet joints can be seen. In this case, the facet joint shows the greatest rotatory motion (*dotted lines*). If the axis of rotation were in this location, the compressive facet joint would be the primary structure in resisting torsion. (Reprinted with permission from Haher TR, Felmy W, Baruch H, et al. The contribution of the three columns of the spine to rotational stability, a biomechanical model. Spine 1989;14(7):663–670.)

placed on the spinous processes. Using the entire lumbar spine instead of a single level vertebral model allows the intermediate segments of the spine to rotate about their anatomic axis of rotation. Torsional loads were applied to the spines and axial rotations were recorded for intact spines and for those having the anterior column destroyed (to include the anterior longitudinal ligament and 67% of the anterior anulus), middle column destroyed (posterior longitudinal ligament and the posterior 33% of the anulus), and posterior column destroyed (facet joints and posterior interspinous ligaments) (223).

An average loss of rigidity of 75% was seen with anterior column destruction, whereas only 25 to 35% was seen with posterior column destruction. Progressive destruction of the anulus was proportional to the loss of torsional stiffness. The posterior ligaments consisting of the supraspinous and interspinous ligaments also had little effect on resisting rotation.

The instantaneous axes of rotation constitute the centers about which lumbar spine muscles exert their moment during flexion, extension, and torsion (224). When the lumbar spine undergoes rotation, lateral bending also occurs. The reverse is also true: lateral bending of the spine results in rotation. This relationship is referred to as "coupled motion," and it is defined as the consistent association of one motion about an axis with another motion about a second axis.

The intervertebral disc is considered one of the structures of major importance in spinal motion, stability, and spinal disorders; it is also of major importance in resisting torsion. *Injury to the disc can lead to altered sharing of the load between the disc and the apophyseal joints.*

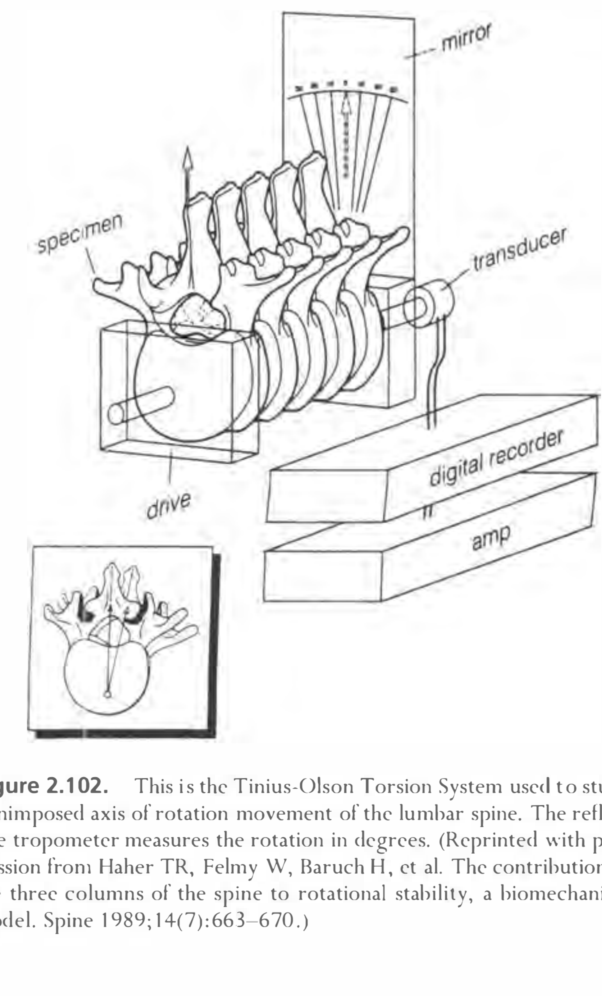

Figure 2.102. This is the Tinius-Olson Torsion System used to study nonimposed axis of rotation movement of the lumbar spine. The reflective tropometer measures the rotation in degrees. (Reprinted with permission from Haher TR, Felmy W, Baruch H, et al. The contribution of the three columns of the spine to rotational stability, a biomechanical model. Spine 1989;14(7):663–670.)

If the instantaneous axis of rotation is at the facet joints, the disc resists rotation. If the axis of rotation is at the disc, the facet joints resist rotation (224). Axial rotation of the lumbar spine was found to be less when combined with forward flexion, It is hypothesized that, in this position, the posterior anulus and posterior longitudinal ligament are being stretched with a small component of rotation being sufficient to damage these structures. In flexion, the instantaneous axis of rotation is possibly moved posteriorly, inducing greater displacements anteriorly, thus causing rim lesions or peripheral anular tears. This mechanism could explain the frequent clinical finding of patients reporting that bending-rotation movements coincided with the onset of their low back pain (225).

Adams and Hutton (98) state that the posterior facet joints limit rotation strain on the disc, and that torsional stresses great enough to damage the posterior element facet joints would be needed in order to harm the disc. However, this is in a patient with normal disc tissue, not a patient with a protruding disc in whom the anular fibers are strained to contain the bulging, high-intradiscal nucleus pulposus. The difference may be likened to that between the strength of a rubber band that has been stretched many times versus one that has never been stretched.

Three Degrees Torsion Is Maximum Before Anular Damage

The maximal torsion angle for isolated discs that will not damage anular fibers found is 3° (226). Because twisting the torso has been cited as a significant risk factor for low back pain, common industrial trunk motions and recorded trunk loading via an electromyogram (EMG) assisted model were studied. Figure 2.103 shows the twisting frame for measuring axial trunk torque on 320 individuals under various static and dynamic twisting tasks. Figure 2.104 shows the trunk muscle vectors determined from their origins and insertions.

The body's ability to produce a twisting moment is far more limited that its ability to produce a lifting moment. Pure torsional moment is not possible without also generating extension (20% extension maximal voluntary contraction) and lateral moments (79% lateral maximal voluntary contraction). *It is easy for task demands to exceed the capacity of the trunk in torsional exertions* (227).

Velocity increases compression forces, placing an individual at a higher risk of exceeding tolerance to such forces. The final result is that the risk of low back pain is related to exertion load, velocity, and twisting angle (227).

LUMBAR SPINE MOTION DYNAMICS AND ABERRANCIES

A nonlinear three-dimensional finite element program (Fig. 2.105) has been used to analyze the response of a lumbar L2–L3 motion segment subjected to axial torque alone and to axial torque combined with compression. Torsion is primarily resisted by the articular facets that are in contact and by the disc anulus. The ligaments play an insignificant role in this respect.

Figure 2.103. A twisting reference frame was employed to control and monitor static posture and dynamic motions of the subjects during torsional exertions. (Reprinted with permission from Marras WS, Granata KP. A biomechanical assessment and model of axial twisting in the thoracolumbar spine. Spine 1995:20(13);1440–1449.)

For the intact segment, with an increase in torque, the axis of rotation shifts posteriorly in the disc so that under maximal torque it is located posterior to the disc itself (Fig. 2.106). Loss of disc pressure increases this posterior shift, whereas removal of the facets decreases it. Torque, by itself, cannot cause failure of disc fibers, but it can enhance the vulnerability of those fibers located at the posterolateral and posterior locations when it acts in combination with other types of loading (e.g., flexion). The most vulnerable element of the segment in torque is the posterior bony structure (228).

Further analysis by the L2–L3 three-dimensional study showed (229):

1. The motion segment exhibits stiffening effects with increasing sagittal plane movements. It is found to be stiffer in extension than in flexion. The segmental stiffness reduces slightly in flexion with the loss of disc pressure, and it reduces considerably in extension with the removal of the facets.
2. In contrast to the case under flexion moment, when relatively high intradiscal pressures are generated, under extension movement negative pressures (suction type) of low magnitude are predicted.
3. Great intradiscal pressure resists the inward bulge of the inner anulus layers. However, when the disc loses its pres-

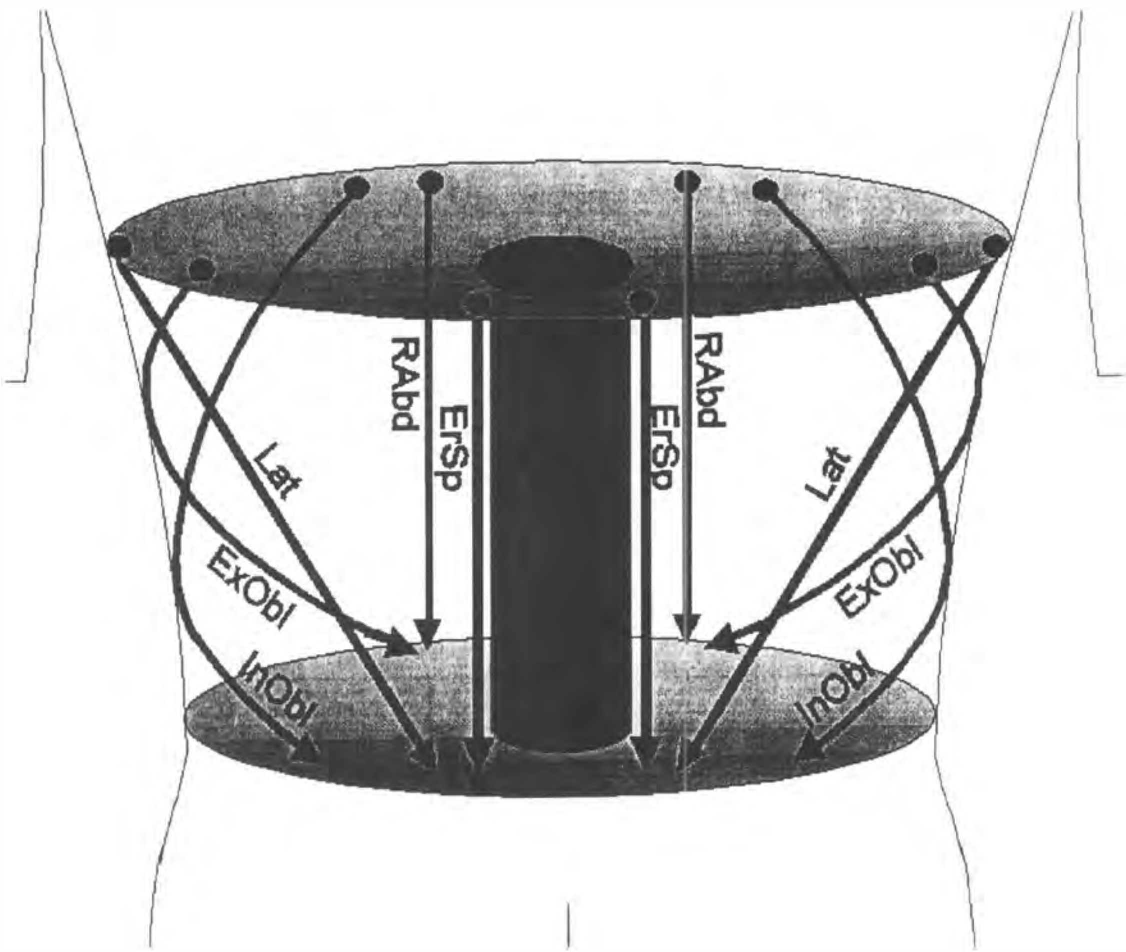

Figure 2.104. Trunk muscles are modeled as vectors determined from three-dimensional locations of muscle origins and insertions. Vector directions, lengths, and velocities are computed as a function of the instantaneous positions of the end points that move with the trunk posture. *ErSP*, erector spinae; *ExObl*, external oblique; *InObl*, internal oblique; *Lat*, lateral; *Rabd*, rectus abdominis. (Reprinted with permission from Marras WS, Granata KP. A biomechanical assessment and model of axial twisting in the thoracolumbar spine. Spine 1995:20(13);1440–1449.)

sure, the inner anulus layers at the anterior region bulge inward markedly under flexion movement.

4. Comparison of the predicted stresses and strains in the various segmental materials with their reported ultimate values leads to the following conclusions: (*a*) Ligaments: Only the vulnerability of the interspinous ligament to rupture at its medial site can be concluded. (*b*) Anulus ground substance: Large tensile radial strains are computed to occur at anterior and posterior locations under flexion and extension movements, respectively. Such strains can cause circumferential clefts between the anulus layers, the frequency of occurrence of which is reported to increase with age. Large tensile axial strains are also predicted, which may correspond with the observation of horizontal splits in the anulus as parallel to the end plates (228). (*c*) Anulus collagenous fibers: The maximal fiber strains are computed to be larger in flexion than in extension, and they occur posterolaterally in the innermost anulus layer of a normal disc. Loss of disc pressure reduces these strains. The fact that the magnitude of the maximal fiber strain is greater than the fibers' reported elastic limit and that the maximal strains occur at the posterolateral fibers in the normal disc suggests that hyperflexion in combination with other types of loading might induce failure of these fibers commonly seen in conjunction with disc prolapse (229).

Effects of Flexion and Extension on the Lumbar Structures

Effects of flexion (Fig. 2.107) on the lumbar spine include:

1. Decrease in the intraspinal protrusion of the lumbar intervertebral disc.
2. Slight increase in the length of the anterior wall of the spinal canal.
3. Significant increase in the length of the posterior wall of the spinal canal.
4. Stretching and a decreased bulge of the yellow ligaments within the spinal canal.

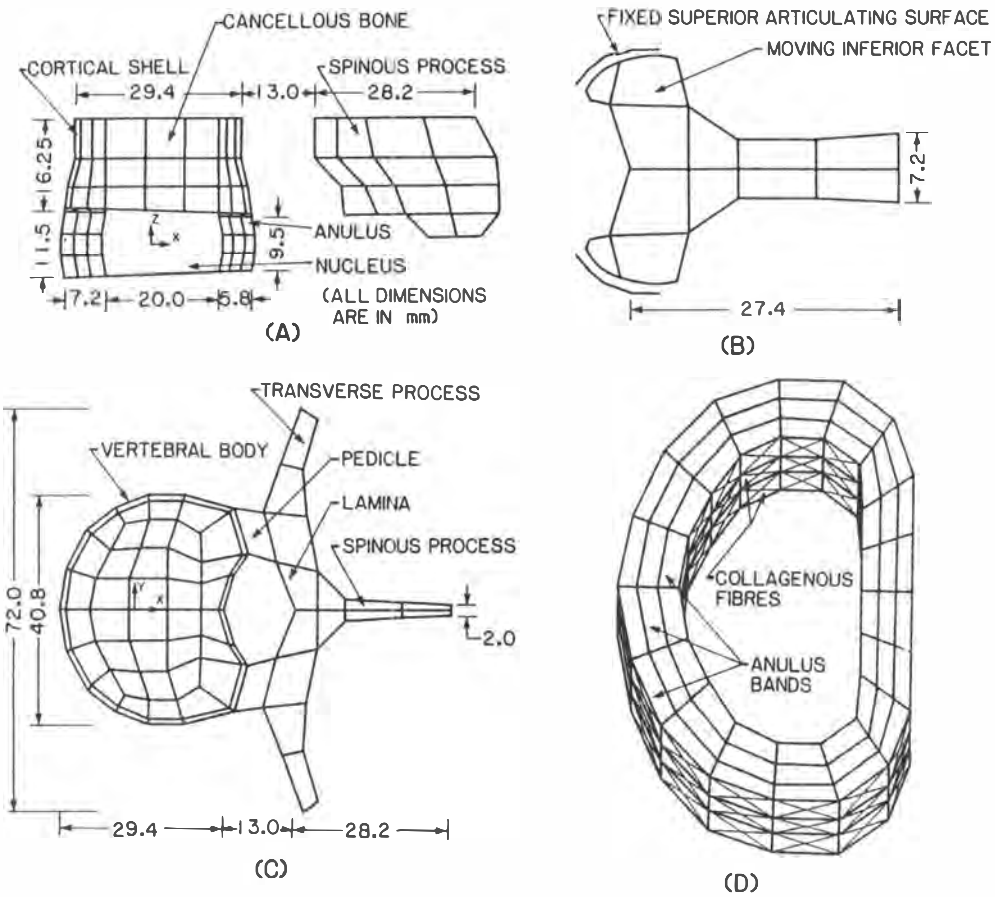

Figure 2.105. Finite element grid of the motion segment. **A.** Sagittal cross-section. **B.** Horizontal section of the posterior bony elements at $z = 5$ mm. **C.** Horizontal cross section at $z = 22$ mm. **D.** Anulus layers and fiber orientation. Measurement of z is done at the sagittal cross section at the top level of the upper vertebral body as a function of axial torque. (Reprinted with permission from Shirazi-Adl A, Ahmed AM, Shrivastava SC. Mechanical response of a lumbar motion segment in axial torque alone and combined with compression. Spine 1986;11(9):915–924.)

5. Stretching and a decreased cross-sectional area of nerve roots.
6. An overall general increase in spinal canal volume and decreased nerve root bulk.

Effects of extension (Fig. 2.108) are:

1. Bulging of the intervertebral disc into the spinal canal.
2. Slight decrease in the anterior canal length.
3. Moderate decrease in the posterior canal length.
4. Enfolding and protrusion of the yellow ligaments into the spinal canal.
5. Relaxation and an increase in the cross-sectional diameter of the nerve roots.
6. An overall decrease in the volume of the lumbar spinal canal and an increased nerve root bulk.

For these reasons, patients seek flexion for relief of back pain, and this is the premise on which the use of flexion-distraction manipulation for correction of disc protrusion is based.

Effects of Flexion and Extension on the Lumbar Canal

Extension of the lumbar spine has been shown to cause protrusion of the intervertebral disc with dorsal displacement of the cauda equina roots (230). On myelography, Ehni and Weinstein (230) showed that extension produces total block and flexion permits the contrast medium to pass through the blocked area. Reaching overhead or bending backward causes the common complaint of painful paresthesia or numbness in both legs (231). Dyck et al. (231) showed that extension promotes lumbar stenosis and forward flexion reduces it.

Raney (232) performed a series of myelograms that showed that with flexion of the lumbar spine, the posterior bulge of the posterior anulus and posterior longitudinal ligament disappeared as the anterior margin of the vertebral bodies approached each other and the posterior margins separated. The myelographic column became flat, and the dural sac closely approximated the back of the posterior longitudinal ligament and anulus. Even though the force propelling the disc posteriorly is increased by flexion, tightening of the posterior anulus and posterior longitudinal ligament in flexion

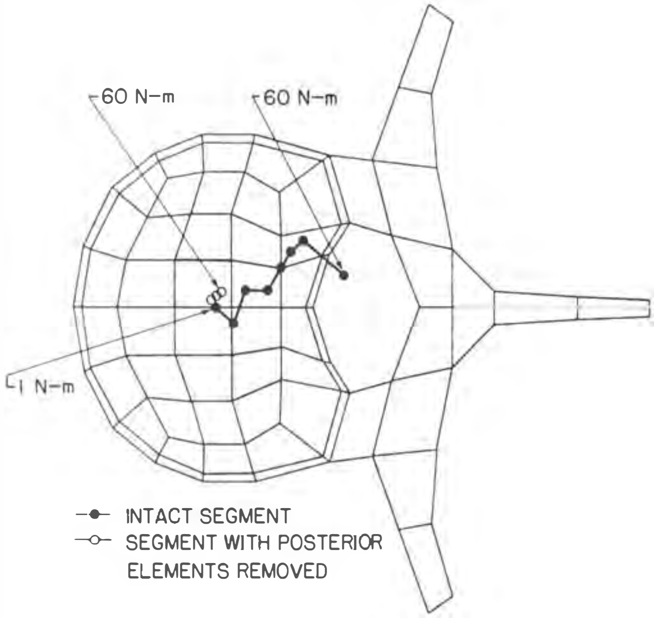

Figure 2.106. Predicted change in the location of the axis of rotation with increasing torque (1 N-m to 60 N-m) at the top level of the upper vertebral body. (Reprinted with permission from Shirazi-Adl A, Ahmed AM, Shrivastava SC. Mechanical response of a lumbar motion segment in axial torque alone and combined with compression. Spine 1986;11(9): 915–924.)

improved the barrier to a greater extent, with the net effect being reduction of the posterior protrusion. In prolapse, this relief has not been found. Therefore, the flexed position obliterates the disc bulge and relieves the irritated nerve root in the bulging disc.

Pilling (233), who performed myelography on patients in the upright position, showed that a protrusion is reduced in the flexion position because the posterior longitudinal ligament and anulus are stretched and the disc spaces are widened posteriorly. A prolapse would not show such reduction.

With epidurography, Matthews and Yates (234) showed that 120-pound distraction reduced disc protrusion.

Extension causes the ligamentum flavum, the disc, and the posterior longitudinal ligament to narrow the sagittal diameter of the vertebral canal, whereas flexion reverses this (235).

McNeil et al. (236) demonstrated that the extensor muscles are the weakest muscles in the low back. We, therefore, exercise them after the patient has had relief of low back and leg pain.

White and Panjabi (141) showed that with bending of the spine, the disc bulges on the concave side of the curve and collapses on its convex side. In flexion, the disc protrudes anteriorly and depresses posteriorly. Finneson (134) showed disc protrusion on extension and reduction on flexion (Figs. 2.107 and 2.108).

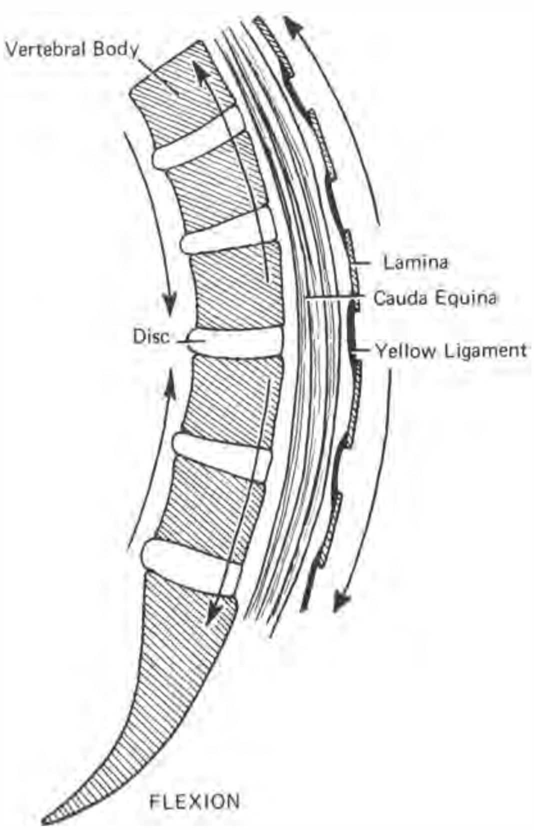

Figure 2.107. Increased spinal canal volume and decreased nerve root (cauda equina) bulk with flexion. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:432.)

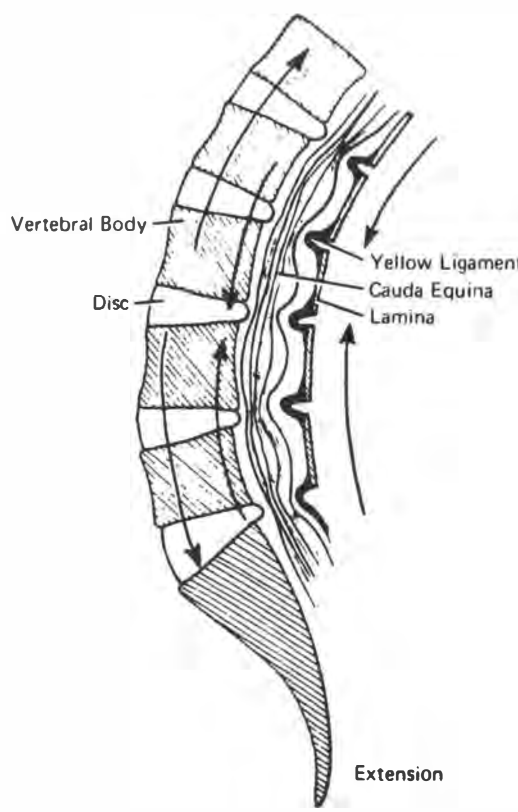

Figure 2.108. Decreased spinal canal volume and increased nerve root bulk with extension. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:432.)

Effects of Flexion and Extension on the Spine

Cadaveric lumbar motion segments (consisting of two vertebrae and the intervening disc and ligaments) were loaded to simulate forward bending movements in life, and the flexion angle at the elastic limit was measured (237). These flexion angles were then compared with flexion angles obtained from radiographs of healthy volunteers in the erect standing and fully flexed positions. The comparison showed that, when people adopt the static, fully flexed posture, the osteoligamentous lumbar spine is flexed about 10° short of its elastic limit. The results imply that the lumbar spine is normally well protected by the back muscles in the relaxed, fully flexed posture. Special mechanisms must be identified to explain forward bending injuries.

For a typical motion segment, a 2° reduction in flexion means a 50% reduction in the resistance to bending moment, and hence a 50% reduction in the bending stresses in the posterior anulus and intervertebral ligaments. At the limit of the range of flexion, the osteoligamentous spine resists a bending movement equal to about 50% of that exerted by the upper body in forward bending. This means that at the more moderate angles of flexion found in life, only about 5% of the upper body's forward bending movement will be resisted by the spine; the rest will be resisted by the lumbodorsal fascia, back muscles, and so forth. Therefore, it appears that little chance is seen of damaging the lumbar spine in the static toe-touching posture (237).

For flexion-extension mode, more mobility exists at the L4–L5 level than at the L1–L2. For lateral bending mode, more mobility is observed at the L1–L2 level than at the L4–L5 level (238).

Effects of Flexion and Extension on the Dural Sac During Myelography

Penning and Wilmink (239) performed measurements on 40 lateral lumbar myelograms in flexion and extension to analyze changes in position and shape of the dural sac in spinal movements. There proved to be an anterior displacement of the entire lumbar dural sac in lumbar extension, most likely caused by shortening and thickening of the flaval ligaments. In addition, the anterior dural surface was indented at the L3–L4 and L4–L5 interspaces by posterior bulging of the discs in extension. This encroachment was partially compensated by dual bulging into areas with a rich and compressible venous plexus behind the vertebral bodies and the L5–S1 disc. Although the patterns of dural movements showed individual variations, these trends were found in all diagnostic and anatomic subgroups. One subgroup (with root involvement at L4–L5) showed marked dorsal encroachment on the dural sac in extension at the same level.

In distinction to these posture-dependent changes in a cross-sectional area of the spinal canal, Breig (240) stressed the influence of flexion-extension movements on longitudinal spinal dimensions. In spinal flexion, marked elongation of the spinal canal is seen with concomitant stretching of the dural sac and nerve root fibers. This is thought to have a bearing on the production of nerve root symptoms in cases with disc herniation (239).

Moderate Flexion Finds Lumbar Spine Strongest

Accurate measurement of lumbar spine curvature is important because curvature affects the stresses acting on the apophyseal joints and intervertebral discs. In moderate flexion, the lumbar spine is at its strongest; in full flexion, the discs are vulnerable to fatigue damage, and in hyperflexion, the intervertebral ligaments can be sprained and the discs can prolapse suddenly. Therefore, to evaluate the risks of a job involving bending and lifting, it is necessary to measure how much the lumbar curve is flexed in each bending movement (241).

Forty-one cadaveric lumbar intervertebral joints from 18 spines were flexed and fatigue-loaded to simulate a vigorous day's activity. The joints were then bisected and the discs examined. Twenty-three of 41 discs showed distortions in the lamellae of the anulus fibrosus and, in a few of these, complete radial fissures were found in the posterior anulus (242).

Flexed Postures Improve Transport of Metabolites

A study (243) compared postures that flatten (that is, flex) the lumbar spine with those that preserve the lumbar lordosis. Flexed postures have several advantages: flexion improves the transport of metabolites in the intervertebral discs, reduces the stresses on the apophyseal joints and on the posterior half of the anulus fibrosus, and gives the spine a high compressive strength. Flexion also has disadvantages: it increases both the stress on the anterior anulus and the hydrostatic pressure in the nucleus pulposus at low load levels. The disadvantages are not of much significance, and we conclude that it is mechanically and nutritionally advantageous to flatten the lumbar spine when sitting and when lifting heavy weights (243).

Flexion Occurrence at Pelvis Versus Low Back

In normal subjects, lumbar motion accounts for 63% of gross flexion, with 37% caused by pelvic motion in up to about 90° of flexion. Low back pain subjects exhibit less gross motion than normal subjects (54%), with the ratio of lumbar flexion to gross flexion decreased from 63 to 43%. Range-of-motion exercising can significantly increase functional pain-free range in both lumbar (71%) and pelvic (39%) motion over a 3-week period (244).

Effects of Flexion and Extension on the Spinal Canal

Myelographic studies show that flexion opens the anterolateral angles of the spinal canal, creating relief of the nerve roots. Conversely, extension closes the anterolateral angles, causing bilateral root involvement, as evidenced by the conventional myelogram in extension (Fig. 2.109). In extension, nerve root involvement signs are maximal, whereas in flexion they tend to disappear (239, 245). This seems to indicate some posture-dependent narrowing of the spinal canal rather than disc herniation as the cause of the problem. Facet hypertrophy, both of the facet and its covering ligaments, has long been felt to be a possible factor in narrowing of the spinal canal (246, 247), but this is difficult to prove in plain radiography of the lumbar spine. Penning and Wilmink (248) showed the presence of facet hypertrophy, sometimes in combination with an abnormally bulging but not necessarily herniated disc.

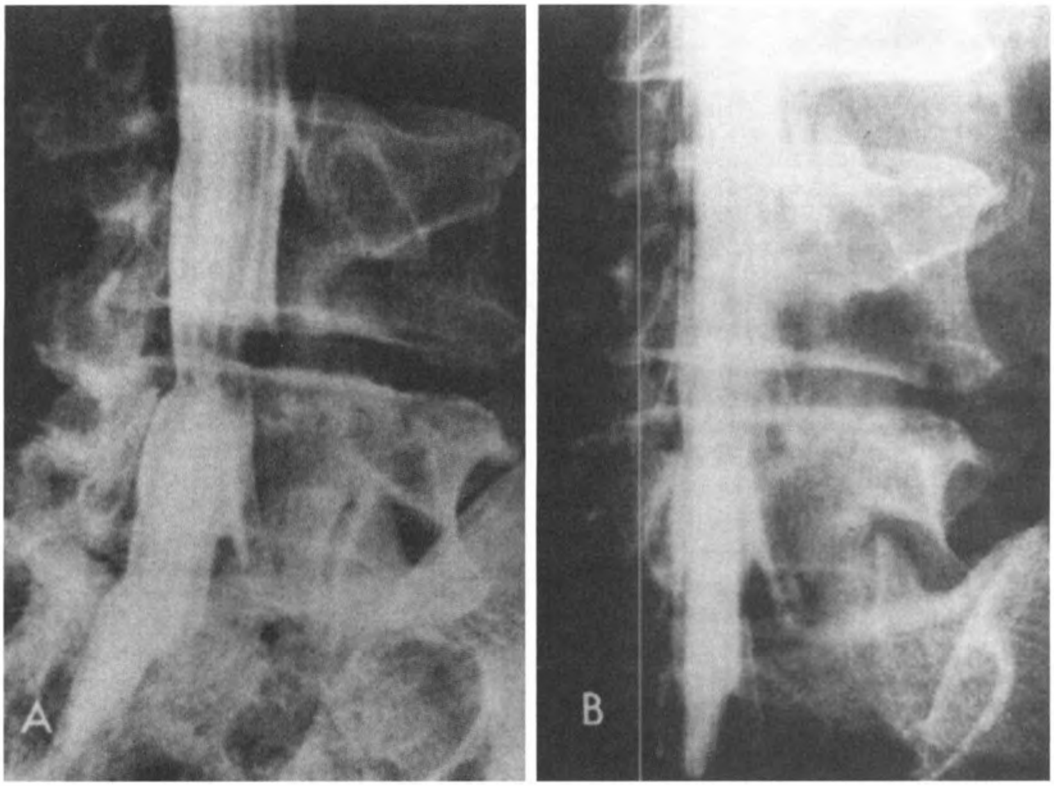

Figure 2.109. Effects of lumbar flexion-extension movements upon root involvement. **A.** Water-soluble lumbar myelogram, RPO projection, showing compression of the emerging left L5 root in extension, with swelling of nerve root and cutoff of root sheath filling. Note also dorsal indentation in dural sac at same level. **B.** Flexion view shows that compression of L5 root and dural sac has been relieved. Right L5 root (not illustrated) showed similar posture-dependent involvement. (Reprinted with permission from Penning L, Wilmink JT. Posture-dependent bilateral compression of L4 or L5 nerve roots in facet hypertrophy. Spine 1987;12(5):489.)

In 12 patients with myelographic evidence of bilateral root involvement at the L3–L4 or L4–L5 levels, postmyelographic CT studies were performed in flexion and extension. They showed concentric narrowing of the spinal canal in extension, and widening with relief of nerve root involvement in flexion (Fig. 2.110). This could be attributed to marked degenerative hypertrophy of the facet joints, narrowing the available space for dural sac and emerging root sleeves. In extension of the lumbar spine, bulging of the disc toward the hypertrophic facets causes a pincers mechanism at the anterolateral angles of the spinal canal, with the risk of bilateral root compression. This mechanism is enhanced in these cases by marked dorsal indentation of the dural sac because of anterior movement of the dorsal fat pad in extension. The authors of the study believe that the radiologically described mechanism forms the anatomic basis of neurogenic claudication and posture-dependent sciatica (248).

Closing of the anterolateral angles can be explained by bulging of the disc in extension, as shown in Figure 2.111. This bulging, described by Knuttson (249), among others, is a physiologic phenomenon caused by the approximation of the dorsal parts of the vertebral end plates. Thus, no disc pathology is needed to explain the narrowing or complete occlusion of the anterolateral angles of the spinal canal in facet hypertrophy. This facet hypertrophy may reduce the distance between disc and facet to such a degree that normal disc bulging in extension is sufficient to close the lateral recesses. In most patients, the dorsal surface of the disc had a symmetric appearance. In some of these instances, possibly disc bulging in extension was pathologically increased because of disc degeneration and subsequent approximation of adjoining vertebrae, but this was impossible to measure. More marked asymmetry was noted in a minority of patients, indicating that disc pathology (abnormal bulging, disc prolapse) in these cases might have played an additional role. Hypertrophy of the flaval ligaments has also been mentioned as a cause of additional narrowing of the spinal canal but, as with abnormal disc bulging, this is difficult to quantify (248).

Fat Pad Changes During Motion

Anterior movement of the dorsal fat pad is an additional mechanism contributing to root compression in extension. In previous studies, it has been noted that in patients with bilateral root involvement at L4–L5, marked dorsal indentation of the dural sac in extension takes place. That the dorsal indentation of the dural sac is related to anterior motion of the dorsal fat pad came as a surprise because it had always been presumed that the flaval

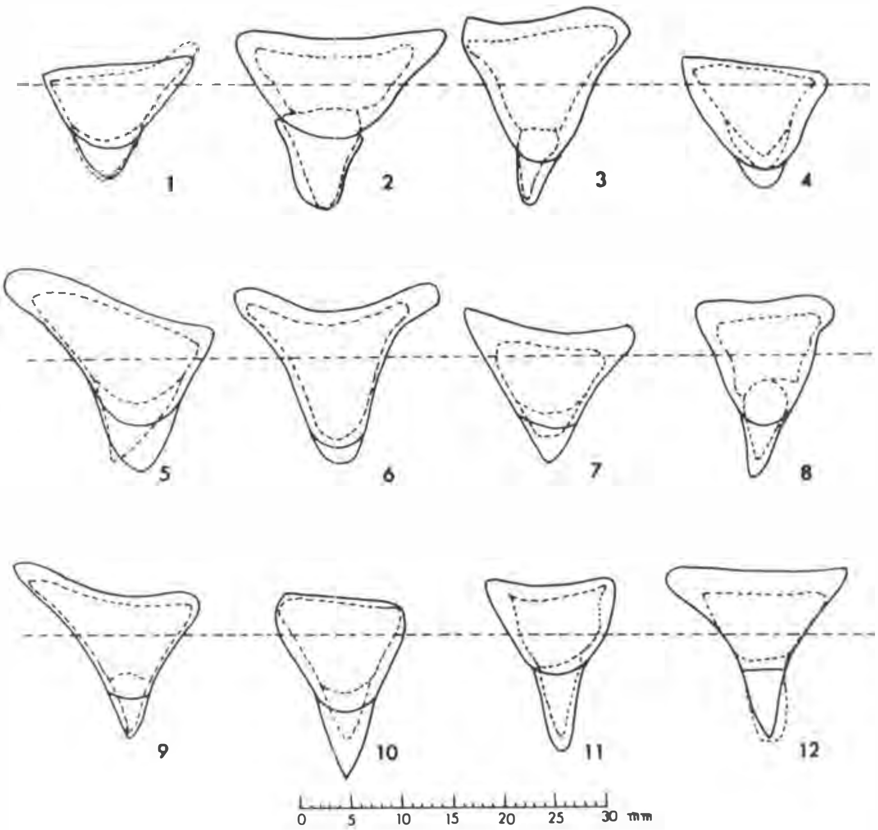

Figure 2.110. Drawings of dural sac and dorsal fat pad outlines in flexion and extension in the 12 patients. Drawings are made by tracing the outlines of dural sac and dorsal fat pad from maximally (x4) enlarged CT slices. *Solid lines,* outlines in flexion. *Broken lines,* outlines in extension. The *horizontal line* represents the interfacet line. (Reprinted with permission from Penning L, Wilmink JT. Posture-dependent bilateral compression of L4 or L5 nerve roots in facet hypertrophy. Spine 1987;12(5):495.)

ligaments would cause the dorsal indentation, as is the case in spondylotic myelopathy in the cervical spine (250). However, examples (Figs. 2.112 and 2.113 *B* and *D*) show that when lumbar lordosis increases and the laminae approach one another, the fat pad (which cannot be compressed but is easily deformed because of its semiliquid form) decreases in its longitudinal dimension and ,as a result, thickens in the transverse plane. Bordered dorsolaterally by flaval ligaments, the fat pad can expand only in an anterior direction at the expense of the dural sac. This forward expansion of the dorsal fat pad is enhanced by some thickening of the flaval ligaments in extension. This thickening by shortening was previously described in a cadaver study by Knuttson (249). Most patients present with transverse flattening and sometimes some anterior displacement of the entire fat pad because of flaval thickening in extension, probably in combination with slight anterior movement of the lamina with respect to the disc. The effect of the anterior movement of the dorsal fat pad in extension is anterior displacement of the dural sac with increased presentation of the emerging root sleeves to the pincers mechanism at the anterolateral angles (248).

Measurements were performed on 40 lateral lumbar myelograms in flexion and extension to analyze changes in position and shape of the dural sac in spinal movements. Anterior displacement of the entire lumbar dural sac in lumbar extension was found, which most likely was caused by shortening and thickening of the flaval ligaments. In addition, the anterior dural surface was indented at the L3–L4 and L4–L5 interspaces by posterior bulging of the discs in extension. This encroachment was partially compensated by dural bulging into areas with a rich and compressible venous plexus: behind the vertebral bodies and the L5–S1 disc. Although these patterns of dural movements showed individual variations, these trends were found in all diagnostic and anatomic subgroups. Marked elongation of the spinal canal with concomitant stretching of the dural sac and nerve root fibers is found in spinal flexion (239).

Velocity of Motion May Be a Factor in Back Pain

Trunk mobility, as defined by trunk angle, has long been considered an acceptable means to evaluate the degree of impairment in patients with low back pain. However, biomechanically, no reason is found to believe that patients with low back pain have significant sensitivity to trunk velocity of motion or that angular mobility factors have an impact on their condition. Thus, it is suggested that trunk velocity be used as a quantita-

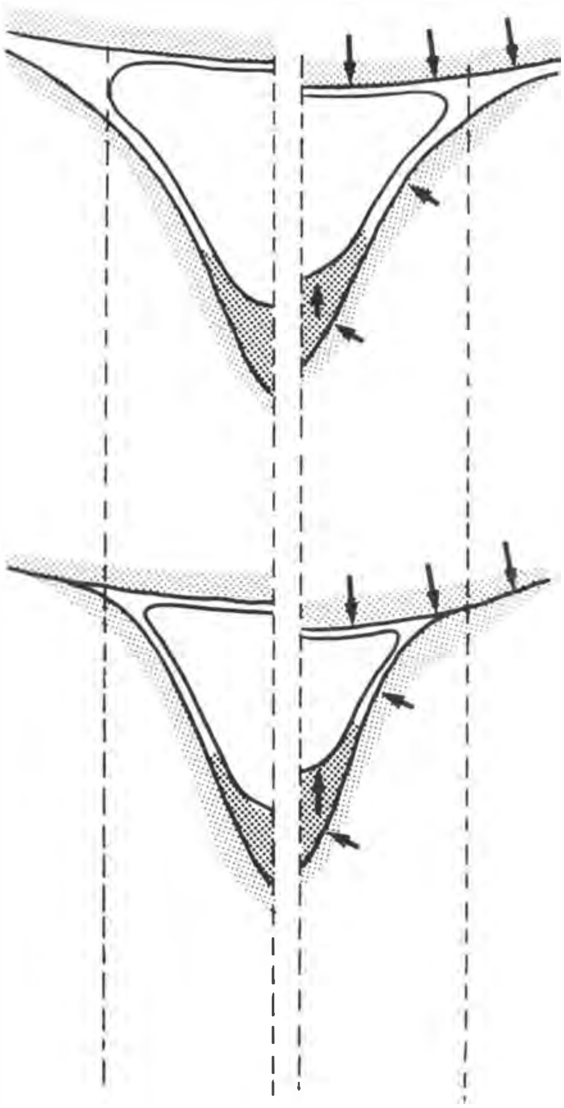

Figure 2.111. Effects of flexion extension in normal canal (*above*) and in individual with facet hypertrophy (FH) (*below*). *Left*, Flexion; *right*, extension. In a normal canal, extension causes reduction of cross-sectional area available for dural sac through combined bulging of disk, flaval ligaments, and retrodural fat pad, without endangering available space for dural sac and emerging nerve roots. In an individual with FH, loss of reserve space, already present in flexion, enhances narrowing of spinal canal and lateral recesses in extension, and pinching effect on dural sac and emerging nerve roots. (Reprinted with permission from Penning L, Wilmink JT. Posture-dependent bilateral compression of L4 or L5 nerve roots in facet hypertrophy. Spine 1987;12(5):496.)

tive measure of low back disorder and to monitor the rehabilitative progress of patients with low back pain (251).

Effect of Spinal Fusion on Adjacent Segment Motion

Effects of spinal fusion on the fused segments and the adjacent, unfused segments play a significant role in the clinical effectiveness of spinal fusion for low back pain with or without sciatica. Much of the information on this important subject is derived from clinical impressions. All types of fusion resulted in increased bending and axial stiffness. All types of fusion demonstrated stabilizing effects on the fusion and produced increased stress on the adjacent, unfused segments, especially the facet joints (252).

Resistance of Ligamentous and Bony Elements

Resistance to Flexion

Capsular ligaments of the apophyseal joints play the dominant role in resisting flexion of an intervertebral joint. In full flexion, as determined by the elastic limit of the supraspinous and interspinous ligaments, the capsular ligaments provide 39% of the joint's resistance. The balance is made up by the disc (29%), the supraspinous and interspinous ligaments (19%), and the ligamentum flavum (13%) (203).

In hyperflexion, the supraspinous and interspinous ligaments are damaged first, followed by the capsular ligaments and then the disc. Bending forward and to one side, however, could damage the capsular ligaments first because the component of lateral flexion would produce extra stretching of the capsule away from the side of bending while not affecting the supraspinous and interspinous ligaments which lie on the axis of lateral bending.

In flexion, as in torsion, the apophyseal joints protect the intervertebral disc. Once the posterior spinal ligaments have been sprained in hyperflexion, the wedged disc is can prolapse into the neural canal if subjected to a high compressive force.

Disc Changes Affect Movement More Than Facet Changes

The posterior vertebral ligamentous and bony elements' influence on the sagittal range of motion of the lumbar spine has been investigated by observing the effects of sectioning ligaments and pedicles in 17 cadavers of both sexes ranging in age from 17 to 78 years. The investigation showed that the apophyseal joints provide a greater restraint to flexion and extension movements than do the lumbar ligaments. It also showed that the age changes that most severely affect movement in elderly persons occur in the intervertebral discs rather than in the posterior elements (253).

Resistance to Intervertebral Shear Forces

When an intervertebral joint is loaded in shear, the apophyseal joint surfaces resist about one third of the shear force, whereas the disc resists the remaining two thirds (203). This passive resistance to shear is complicated by two features, however. First, when an intervertebral disc alone is subjected to sustained shear, it readily creeps forward. In an intact joint, this readiness to creep would manifest itself as stress relaxation, thus placing an increasing burden on the apophyseal joint surfaces until they resist all of the intervertebral shear force. Second, the muscle slips attached to the posterior part of the neural arch brace it by pulling downward. This prevents any backward bending and brings the facets more firmly together.

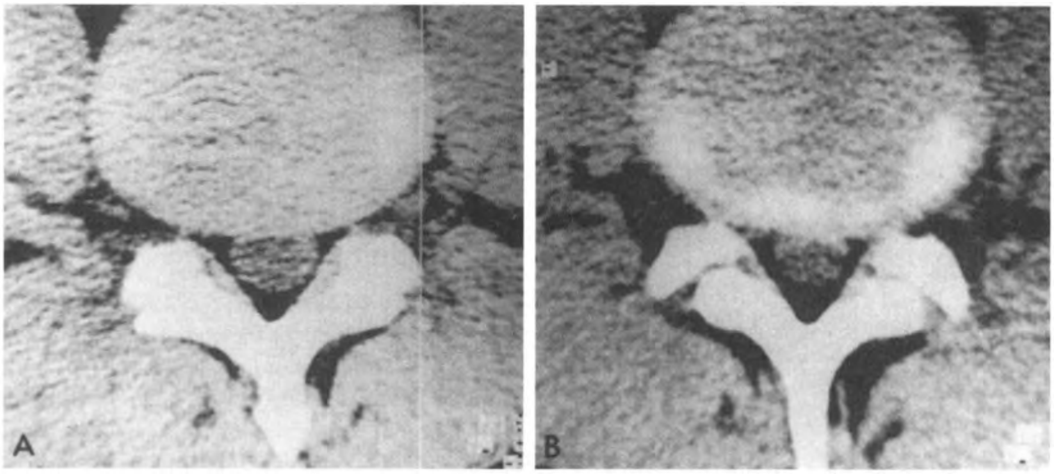

Figure 2.112. Flexion-extension CT scan in a normal individual. Note increase in depth of black epidural fat from **(A)** lumbar flexion to **(B)** extension, at the expense of the dural sac. Combination of fat thickening and disc bulging, both physiologic, causes reduction of cross-sectional area of dural sac amounting, in this example, to approximately 30%. (Reprinted with permission from Penning L, Wilmink JT. Posture-dependent bilateral compression of L4 or L5 nerve roots in facet hypertrophy. Spine 1987;12(5):497.)

In the intact joint this means that the intervertebral disc is subjected only to pure compression and that the intervertebral shear force is resisted by the apophyseal joints, producing a high interfacet force.

Resistance to Intervertebral Compressive Force

Absence of a flattened articular surface in the transverse plane at the base of the articular facets clearly suggests that apophyseal joints are not designed to resist intervertebral compressive force. Experiments confirm that, provided the lumbar spine is slightly flattened (as occurs in erect sitting or heavy lifting), all the intervertebral compressive force is resisted by the disc. When lordotic postures such as erect standing are held for long periods, however, the facet tips contact the laminae of the subadjacent vertebra and bear about one sixth of the compressive force (203).

This contact may well be of clinical significance, because it will result in high stresses on the tips of the facets, and possibly, nipping of the joint capsules. Perhaps this is the reason standing for long periods can produce a dull ache in the small of the back that is relieved by sitting or by using some device, such as a bar rail, to induce slight flexion of the lumbar spine. Disc narrowing results in as much as 70% of the intervertebral compressive force being transmitted across the apophyseal joints. Three such specimens tested exhibited gross degenerative changes in the apophyseal joints (203).

With increasing extension of an intervertebral joint, the compressive force transmitted across the apophyseal joints increases, and it is likely that the extension movements are limited by this bony contact. Thus, it is possible that hyperextension movements could cause backward bending of the neural arch, eventually resulting in spondylolysis, but again only as a fatigue fracture.

Ligament and Facet Contribution to Lumbar Spine Stability

Results from a comprehensive nonlinear finite element model of the L3–L4 motion segment study of the role of ligaments and facets in lumbar spine stability indicate that:

1. Ligaments play an important role in resisting flexion rotation, whereas facets are important in (*a*) resisting anterior shear displacements that accompany flexion and (*b*) restricting extension rotations.
2. Rotational instability in flexion or posterior displacement (retrospondylolisthesis) is unlikely to occur without prior damage of ligaments, whereas instabilities in extension rotation or forward displacement (spondylolisthesis) are unlikely before facet degeneration or removal.
3. Supraspinous and interspinous ligaments are most susceptible to failure in flexion, whereas capsular ligaments are liable to fail under extension or flexion, and shear loads may lead to facet osteoarthritis or hypertrophy. Spinal stenosis is likely under these conditions, especially under flexion-anterior shear loading.
4. High stresses occur on the articular processes.
5. Ligament strains and facet loads are sensitive to their orientations. A more sagittally oriented facet allows larger sagittal displacements, and it can be linked to the cause of spondylolisthesis, whereas a less transversely oriented facet joint allows greater extension rotation, and it can be linked to rotational instabilities in extension.
6. Localized facet excision (anterior regions of L3 inferior facets and lower anterior and posterior regions of superior L4 facets) may restore spinal canal size without compromising the segment stability (254).

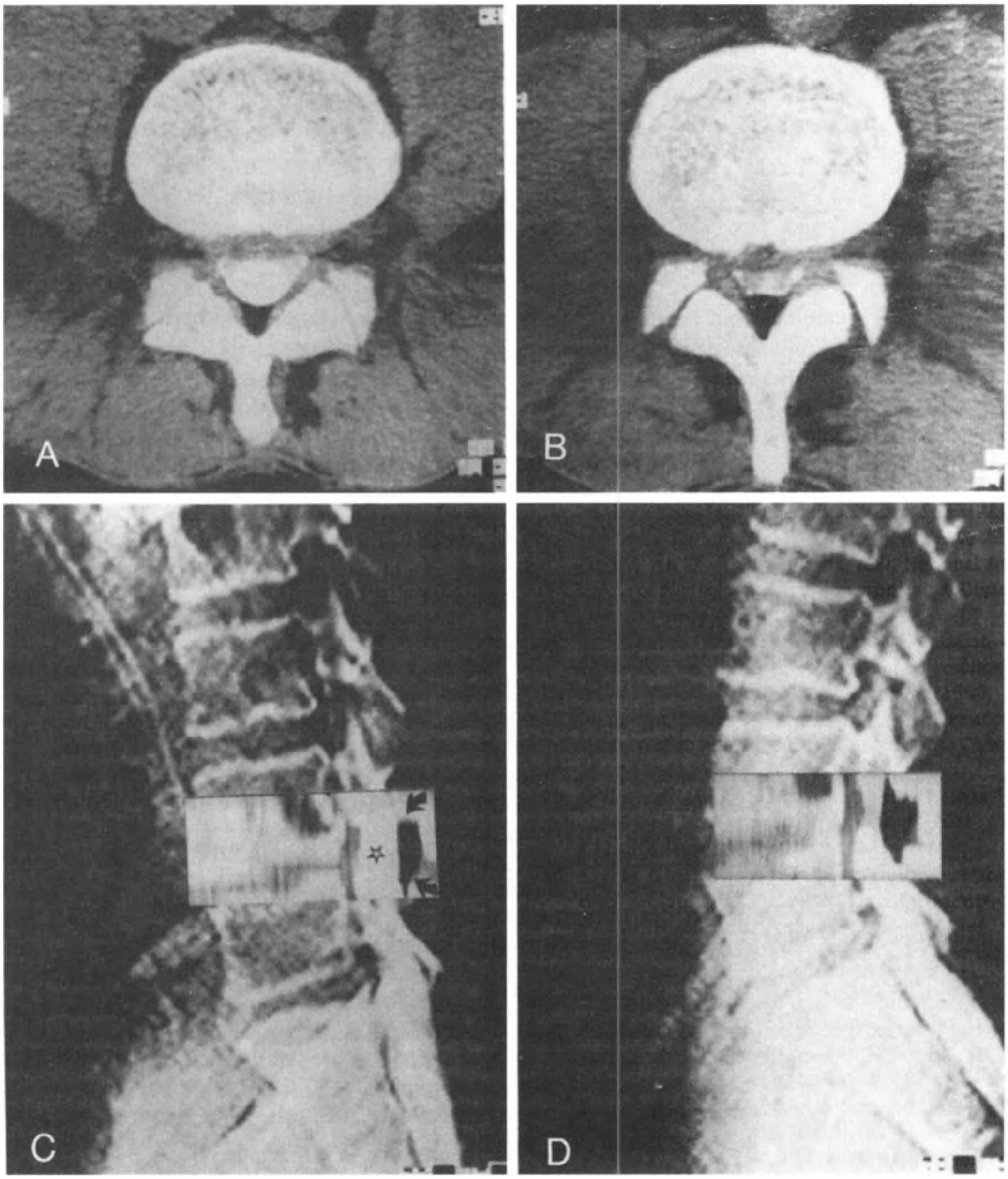

Figure 2.113. Flexion-extension CT scan. Transverse sections (**A, B**) and lateral views (**C, D**) are shown. Lateral views are composed of scanograms with midsagittal reformats of CT sections superimposed at L4–L5 (*insets*). Note marked anterior expansion of dorsal fat pad (between arrows) from flexion (**A, C**) to extension (**B, D**), indenting posterior surface of dural sac (*asterisks*) because of thickening of flaval ligaments and approximation of laminae. Note narrow lateral angle, in flexion (**A**), formed by disc surface and flaval ligaments. Thickening of flaval ligaments and increased disc bulging in extension (**B**) causes "pincers mechanism," pinching ventrolateral angles of dural sac and emerging L5 roots. (Reprinted with permission from Penning L, Wilmink JT. Posture-dependent bilateral compression of L4 or L5 nerve roots in facet hypertrophy. Spine 1987;12(5):498.)

Flexion-Relaxation Phenomenon of Trunk Flexion

At a certain position of trunk flexion, a sudden onset of electrical silence occurs in back muscles. This is called "flexion-relaxation (F-R) phenomenon." A significant difference in muscular activities of erector spinae between healthy and chronic low back pain patient groups was obtained when subjects returned to the erect position from the maximal flexion. Moreover, time lag between trunk and hip movement was much greater in patients than in healthy subjects. Neuromuscular coordination between trunk and hip can be abnormal in patients with chronic low back pain (255).

Facet Is Questioned As Limiting Factor in Rotation of Lumbar Segments

The concept that the posterior complex of the lumbar spine prevents rotation must be re-examined, according to Scull (256). Traditionally it is stated that rotation of the lumbar spine is precluded by the action of the facetal processes blocking the movement. Scull feels, however, that the disc is the primary articulation in the motion segment comprising a joint with 3° of freedom.

The intervertebral disc most resists the coupled motion of lateral rotation under the application of axial torque, whereas the articular facets most resist the coupled axial rotation under the application of lateral bending at the lumbosacral joint (257).

Farfan Research on Rotational Limiting Anatomic Structures

Farfan (258) found the average angle of rotation at failure for whole joints with normal discs was 22.6°, whereas for degenerated discs the failure was at 14.3° (Fig. 2.114). The disc and the two articular processes of the vertebral arch provide 90% of the torque strength of the intervertebral joints. Disc resistance is by the anular fibers, so the condition of the anular fibers is important and damage to them must have serious consequences for the whole joint.

Forced rotation applied to the intervertebral joints produces damage to the disc and facet joints with the first signs of injury appearing with as little as 3° of forced rotation and requiring only 100 to 200 pounds of torque. The effect of rotation is to tear anular fibers from their attachments to the end plate with the appearance of radial fissures in the anulus. Three phases of disc disease have been proposed: first, anular bulging (protrusion); then, facet joint degeneration with loss of disc thickness and disc extrusion; and finally loss of disc thickness, severely degenerated facet joints, and stenosis causing tautening of the nerve root (259).

Farfan (260) stated that from 12° to 20° of rotation, sharp cracking sounds emanate from the disc vertebra sections at failure of the specimen. These loud snapping sounds are suspected to come from anular injury. Forced rotation of a lumbar disc causes 0.1 to 0.4 inches of lateral shear of the vertebral body. Rotation of 15° for the whole lumbar spine can be expected to produce injury to the disc.

Under laboratory rotational stresses, the disc emitted sharp cracking sounds reminiscent of the loud snapping sounds that are known to accompany the sudden onset of back pain (261).

Pearcy (262) found axial rotation at L5–S1 to be less than 6°. Lateral flexion and axial rotation is a consistent pattern at the upper three lumbar levels, but it varied at the two lower levels. Lateral flexion and rotation of L4–L5 is not consistent; sometimes the axis of rotation of L4 on lateral bending is into the convexity and sometimes into the concavity of the lateral flexion curve. Such discrepancy does not seem to occur in the upper lumbar spine. As long as rotation at L5–S1 is limited, the disc itself is not strained by rotation. Torsion alone is insufficient to damage the intervertebral disc but a combination of flexion and torsion will increase the disc's vulnerability to injury (263). Degree of lumbar flexion influences risk of disc injury more than stoop or squatted posture (264). Other factors predisposing to disc prolapse are disc nutrition, degeneration, rotation, bending, compression, intradiscal pressure, and pre-existing anulus damage (265).

Disc Plays a More Significant Role in Limiting Rotation Than Does the Facet Joint

Shirazi-Adl et al. (266) found that torque, by itself, cannot cause the failure of disc fibers, but it can enhance the vulnerability of those fibers located posterolaterally and posteriorly in the disc. Axis of rotation shifts posteriorly in the disc during torsion so that maximal torque is located posterior to the disc itself. The disc plays a more significant role than the facet joint in resisting torsion with loads of less than 20 N-m.

Torsion increases the intradiscal pressure as well as the compressive load on the disc (267). The spine has a greater ability to rotate when in some degree of flexion, which may have some implication in spinal injury. Torsion affects intradiscal pressure with reduction of the pressure perhaps in some flexed posture (268). Rotational strain is one of the most important factors leading to facet joint hypertrophy and spinal canal stenosis (269).

	NORMAL DISC	DEGENERATED DISC
Average angle of rotation at failure	22.6°	14.3°
Resistance to torque	300 in-lb.	200 in-lb.

Figure 2.114. Farfan rotation failures.

Rotation Resisted by Disc, Facet, and Ligaments

Zimmerman et al. (270) find human and canine disc resistance to rotation to be similar. Normal intervertebral disc contributes 45% of the torsional resistance of the whole lumbar joint, with the facets providing 37% and the ligaments 18%. The canine study showed 45% of torsion resistance to be by the disc, 31% by the facets, and 25% by the ligaments. Facets are not the principal support structure in extension: it is the disc!

Oxland et al. (271) conclude that the intervertebral disc most resists the coupled motion of lateral rotation under the application of axial torque, whereas the articular facets most resist the coupled axial rotation under the application of lateral bending at the lumbosacral joint. The apophyseal joint capsules limit rotation both in neutral and flexed positions. In flexion, the amplitude of rotation in the lumbar spine is reduced. Of the capsuloligamentous structures, it is the posterior anulus and the posterior longitudinal ligament that seem to play the more important role in limiting axial rotation while the spine is flexed (271).

Rotation Not Found in the Low Lumbar Spine

Maigne (272) states that facet orientation at the lumbar spine permits only flexion and extension. The thoracic spine, by virtue of its facet facings, should have a high degree of mobility, especially in rotation. "*No rotation is possible in the lumbar spine by virtue of the facet orientation and form*" (emphasis added). Therefore, the highest degree of rotation and lateral flexion must take place at the level of the thoracolumbar spine.

Helfet and Gruebel-Lee (52), in discussing the instability of the lumbar spine with regard to range of motion, point out that rotation injuries affect primarily the intervertebral disc itself.

Upright Versus Recumbent Rotation of the Lumbar Spine

Case 1

In this case, a 23-year-old woman presented with a history of low back pain but no leg pain. At the time this x-ray study was done, she was being treated only for left shoulder pain; her low back was totally asymptomatic.

Consequently, we performed rotation studies on the spine to determine any differences in rotation from the standing to the recumbent non-weightbearing rotational posture. The anteroposterior view (Fig. 2.115) reveals that this patient has tropism at L5-S1, with the right facet being sagittal and the left being coronal. Hip and sacroiliac joints are adequate. The lateral view (Fig. 2.116) reveals a moderately increased lumbar lordosis. The disc spaces are normal.

Figures 2.117 and 2.118 were taken with the patient in the standing weightbearing posture. Figure 2.117 reveals left rotation, and Figure 2.118 reveals right rotation. Lateral flexion is seen of L4 and L5 with actual Lovett reverse rotation of the spinous process at L4 on left lateral flexion and with minimal Lovett positive rotation on right rotation. Normally, the spinous process would deviate to the concave side; instead, it deviates to the convex side, which is called a "Lovett reverse curve." This may indicate a minimal rotatory capability at L4; no rotation is seen at the L5 level.

Figures 2.119 and 2.120, respectively, show right and left lateral rotation of the lumbar spine with the patient in the recum-

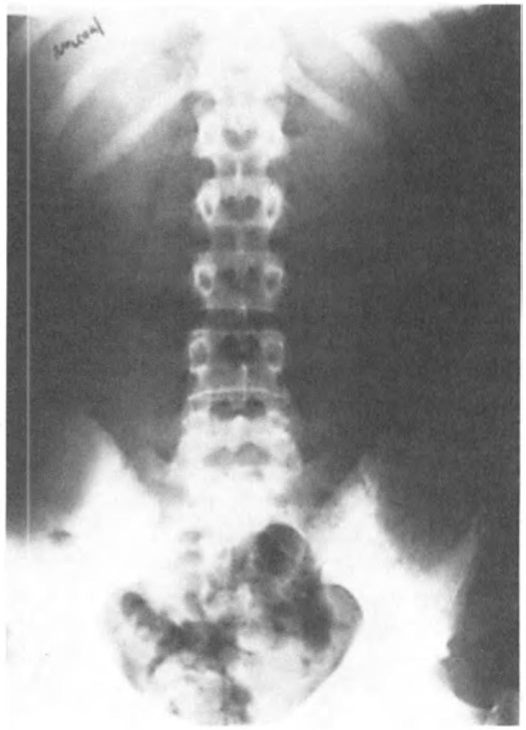

Figure 2.115. Neutral anteroposterior view. Tropism is present at L5-S1.

Figure 2.116. Lateral view—mild hyperlordosis. The disc spaces are normal.

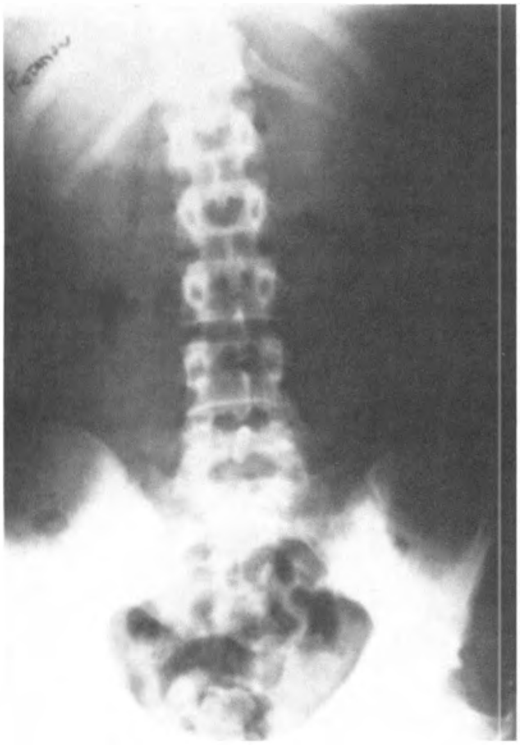

Figure 2.117. Left rotation of the lumbar spine in upright posture.

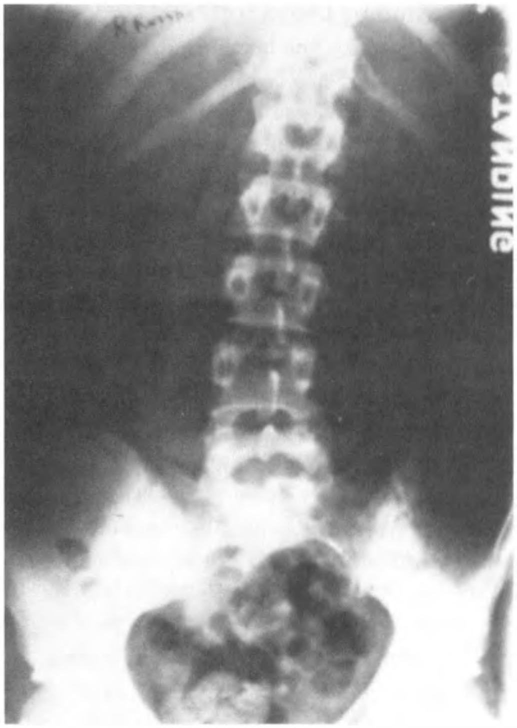

Figure 2.118. Right rotation of the lumbar spine in the upright posture.

Figure 2.119. Right rotation of the lumbar spine in the recumbent supine position.

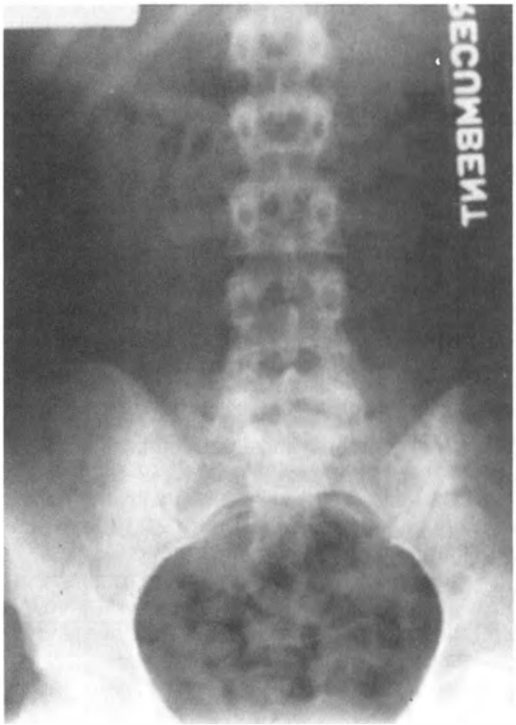

Figure 2.120. Left rotation of the lumbar spine in the recumbent supine position.

bent position. These films were taken with the patient rotating to each side while in the recumbent position and being supported with foam padding in the thoracic spine as maximal rotation is attained. This study was done to determine whether any difference could be seen between the muscular contractions causing rotatory change when the patient assumed a recumbent position and those causing change with the patient in a weightbearing position. As can be seen, no discernible rotation actually occurred with the patient in the recumbent posture. I interpret this to mean that rotation, if possible, is greatest when the patient is standing upright. This might also make sense considering that most back injuries occur during flexion in the upright posture, with either lifting or rotating in combination motion.

Comparison of Lateral Flexion and Rotation of the Lumbar Spine

Case 2

In this case, a 34-year-old woman presented with low back and left lower extremity pain. She has had numerous episodes of low back pain in the past few years, but leg pain has been present for only the past 2 weeks.

Rotation movement of the lumbar spine is studied in comparison with lateral flexion movement. Figures 1.121–2.123 are films of neutral and lateral bending showing normal motion. Figures 2.124 and 2.125 reveal a sacral angle of 33° and a lumbar lordosis of 50°. Figure 2.126 shows no stenosis present. Figure 2.127 is a posteroanterior tilt view of the same patient as in Figure 2.121.

Figures 2.128 and 2.129, respectively, show left and right rotation studies made by having the patient rotate at the waist while holding the pelvis fixed on the bucky. Note that L5 does not rotate measurably and L4 bends laterally but that rotation is no greater than the lateral bendings reveal. Definite increased rotatory movement of the upper lumbar vertebral bodies is seen, coupled with lateral flexion.

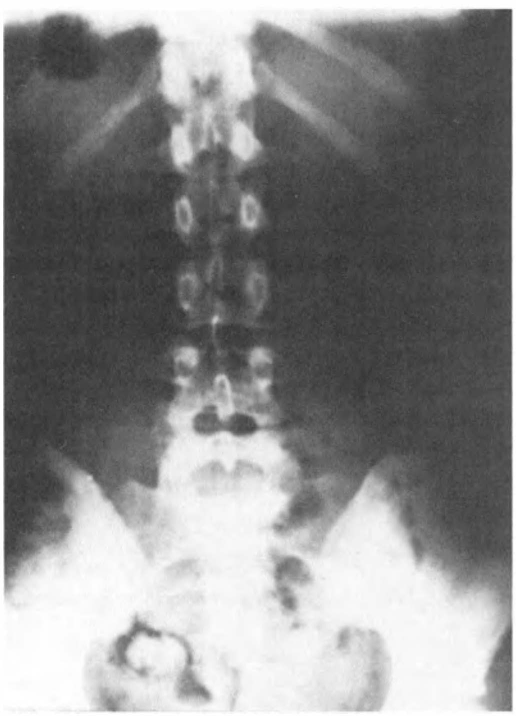

Figure 2.121. Neutral anteroposterior view of the lumbar spine and pelvis.

Figure 2.122. Right lateral flexion.

Rotational and Lateral Flexion Capabilities of the Lumbar Spine

A three-dimensional radiographic technique was used to investigate the ranges of active axial rotation and lateral bending plus the accompanying rotations in places other than those of the primary voluntary movements in two groups of normal male volunteers. Approximately 2° of axial rotation was seen at each intervertebral joint, with L3–L4 and L4–L5 being slightly more mobile. Lateral bending of approximately 10° occurred at the upper three levels, whereas significantly less movement was seen at 6° and 3° at L4–L5 and L5–S1, respectively. In the upper lumbar spine, axial rotation to the right was accompanied by lateral bending to the left, and vice versa. At L5–S1, axial rotation and lateral bending generally accompanied each other in the same direction, whereas L4–L5 was a transitional level (273).

Torsional Versus Compressive Disc Injury

Cyclic torsional load effects on the behavior of intact lumbar intervertebral joints was investigated. Failure locations occurred in such diverse regions as end plates, facets, laminae, capsular ligaments, and so forth. All specimens exhibited a synovial fluid discharge from the apophyseal joint capsule sometime during testing. Post-test examinations of all the cartilage surfaces showed fibrillation, whether or not the intervertebral joint failed (274).

Cyclic torsional fatigue loads produce undesirable effects, such as (*a*) leakage of synovial fluid at the apophyseal joints; (*b*) fibrillation of the facet cartilage surface (Table 2.5); and (*c*) fracture of various elements of the vertebra. The "failures" lead to weakening and improper functioning of apophyseal joints and disc. In the absence of synovial fluid, the apophyseal joint

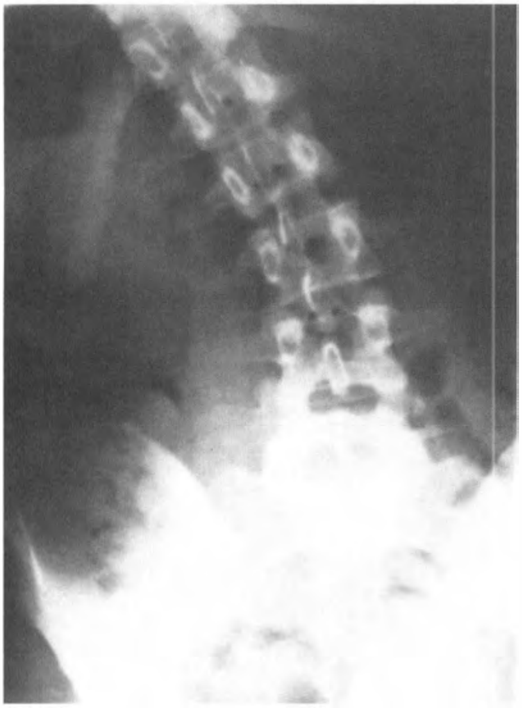

Figure 2.123. Left lateral flexion.

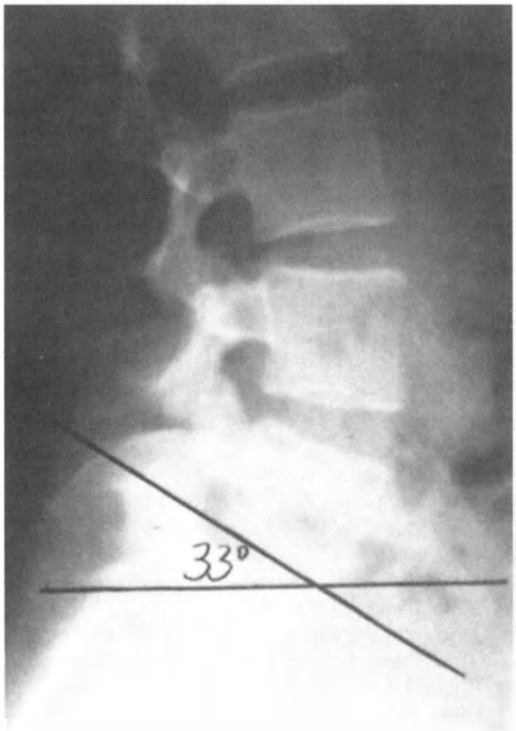

Figure 2.124. Sacral angle measurement.

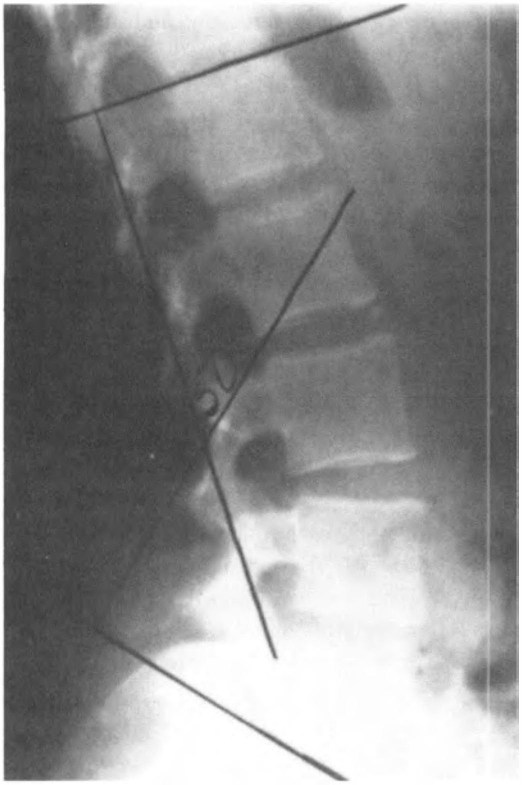

Figure 2.125. Lumbar lordosis measurement.

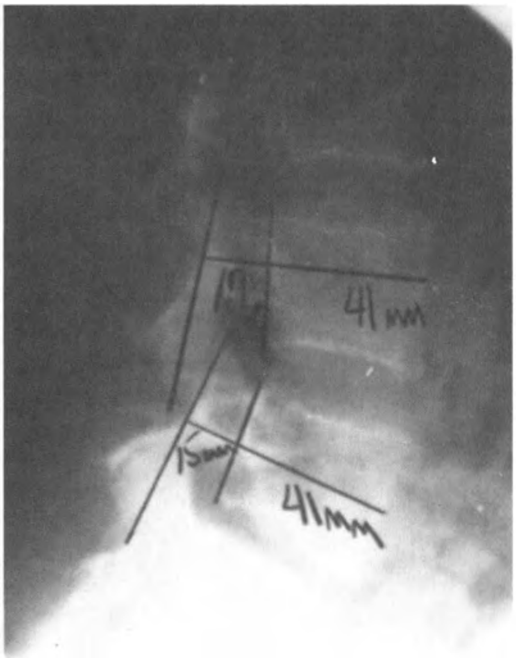

Figure 2.126. Eisenstein's measurement for stenosis.

Figure 2.127. Posteroanterior tilt view of L5-S1. Note the centered position of the spinous processes.

may exhibit more bony contact and higher friction. Under chronic in vivo loading, damage rate can exceed repair rate by the cellular mechanisms of the body. Prolonged exposure to cyclic torsional loads producing 1.5° or greater of angular displacement per segment is detrimental to elements of the lumbar spine. Because these elements contain nociceptors, their disturbance can lead to low back pain (274).

Fifteen discs were studied in pure compression, flexion and extension, axial rotation, and shear. The greatest strains were in torsion (275).

During axial rotation, the oblique fibers, running counter to the direction of movement, are stretched, whereas the intermediate fibers with opposite orientation are relaxed. Tension reaches a maximum in the central fibers of the anulus, which are the most oblique. The nucleus is therefore strongly compressed, and the *internal pressure rises in proportion to the angle of rotation* (276).

Kapandji cites Cailliet: "It has been shown by myelography that there is posterior protrusion of the intervertebral disc from hyperextension" (276). Cailliet speaks of the "concept that rotary trauma in later life causes rupture of the outer anular fibers." He goes on to say:

"Gradually the outward intradiscal pressure causes radial tears to occur in the disc. . . . Rotational forces, however, place torque upon the anular fibers which become disrupted, and the intradiscal nuclear pressure is no longer contained."

Regarding disc herniation, Caillett and Kapandji (276) agree on the effect of microtrauma: Cited by Kapandji, Cailliet states that the strain or repated stresses to discs have frequently occurred prior to the acute onset and have set the state for the ultimate herniation. Weakening of the anulus fibrosus diminishes the elastic recoil against a stress. A minimal stress applied on

Figure 2.128. Left lumbar spine rotation in the upright posture.

Figure 2.129. Right lumbar spine rotation in the upright posture.

Table 2.5

Failures Observed in Specimens Tested at Constant Torque Levels (Mode II)[a]

Number of Failures	Type of Failure Observed		Number of Specimens Failed at Torque (N-m) 11.3	22.6	33.9	45.2	Total
1	Vertebral body	Superior	1	0	1	1	3
(VC)		Inferior	1	2	1	0	4 = 7
2	Facets cracks	Unilateral	1	0	2	2	5
(FC)		Bilateral	0	1	1	1	3 = 8
3	Torn capsules	Unilateral	0	0	2	1	3
(TC)		Bilateral	1	3	0	1	5 = 8
4	Annulus in tears		0	3	0	2	5
(TA)							
5	Lamina crack	Unilateral					
(LC)		Bilateral	1	1	3	0	5
	2 + 3		1	1	2	2	6
	1 + 3		1	2	1	0	4
	1 + 4		0	2	0	0	2
	2 + 4		0	1	0	1	2
	3 + 4		0	2	0	2	4
	1 + 2 + 3		1	1	1	0	3
	2 + 3 + 4		0	2	0	2	4
	1 + 3 + 4		0	2	0	0	2
	1 + 2 + 4		0	1	0	0	1
	1 + 2 + 3 + 4 + 5		1	1	3	0	5

Reprinted with permission from Liu YK, Goel VK, Dejong A, et al. Torsional fatigue of the lumbar vertebral joints. Spine 1985;10(10):899.
[a]The fibrillation of facet articular cartilage and a discharge of fluid from apophyseal joints were observed in all the specimens regardless of whether the specimens failed or not.

the disc that is contained within a weakened defective anulus may cause the nuclear material to herniate.

The most harmful motions appear to be rotations, because these produce both compression and shear. Low back pain patients should be counseled to reduce such activities as shoveling and lifting, as well as recreational activities such as handball, squash, tennis, cycling, and gardening (277).

Anulus Fibrosus Damage by Rotation on Concave Side of Curve

Lindblom, Percy, and others (278) have reported that the injection of saline solution (or some other solution) into the appropriate disc space can reproduce the symptoms of some patients who are suffering from low back and sciatic pain. However, if Novocain is injected into these same discs, the pain is eliminated. Similarly, injection of the hormone hydrocortisone into the appropriate disc space has relieved symptoms in a substantial proportion of patients in whom rupture and actual protrusion or extrusion of a disc fragment had not occurred (278).

Under axial compression, failure occurred due to fracture of one of the vertebral end plates and collapse of the underlying vertebral body. In these tests, failure of the anulus fibrosus occurred only as the result of extremely rapid cyclic bending combined with mild axial compression.

Nucleus Pulposus Migrates to the Concave Side of Curve

It is commonly stated that the nucleus pulposus is the structure by which stresses are distributed uniformly to the anulus fibrosus and cartilaginous plates, and that it moves toward the convex side of the curve when the spine bends to either side or forward and backward. The behavior of two specimens subjected to combined axial loading and bending in this investigation did not seem consistent with this concept. The anulus invariably bulged on the concave side, apparently as the result of compression between the opposing vertebral surfaces (279).

To study the effect of increased pressure on the intervertebral disc, rats' tails were tied up and fixed in the shape of a *U*. It was found that degeneration and even rupture of the anulus fibrosus occurred on the concave side of the tails whereas the convex side remained normal (278).

Intervertebral disc herniations causing low back and sciatic pain are predominantly situated on the concavity of the curvature. The location of intervertebral disc herniation is different in men and women: the fifth lumbar IVD is more often affected in women than in men (278).

Disc and Ligament, Not Facets, Are the Principal Support in Extension Motion

Unilateral and bilateral facetectomies cause an alternate path of loading to be established, namely axial loads are transferred to the anulus and anterior longitudinal ligament to support the spine. Facet joint destruction will not produce acute instability, but it will transfer the loads to the adjacent disc and conceivably accelerate its degeneration.

Anterior anulus and anterior longitudinal ligament of the lumbar spine are the principal support structures in extension. These structures protect the facets from severe loading and degeneration (280).

DISC DEGENERATION ALLOWS ROTATION: DISC MAY NOT BE PROTECTED BY THE FACET JOINTS IN ROTATION

Triano (282) states that a side-lying posture with the lumbar region flexed is commonly used in delivering a chiropractic lumbar spine adjustment. A "positive stop" action, regardless of facet orientation, is believed to reduce risk of injury. This study examined the axial rotation and shearing translations that arise from loading of flexed motion segments with varied facet geometry and material properties. Both symmetric and asymmetric facet orientations were tested. Material properties varied, from healthy to degenerative discs. A 10.6 N-m axial torque from measurements of manipulation were applied. Facet loads increased as a function of degeneration and flexion position to a peak load of 441 N. Facet loading resulted in reduced rotation from those in facetectomized motion segments. Rotations were higher than the "stop action" effects. Rotations increased by a factor of 3.0. Combined interaction of facet geometry and stage of disc degeneration resulted in greater rotations and translations than has been commonly assumed, which could mean that the facet joints do not protect the disc during rotation stresses as much as has been previously thought.

Four Major Low Back Exercises Are Identified As Risk Factors

The four low back exercises found to be risk factors (283) include:

1. Hyperextension: Extension of between 2° and 6° can cause facet loads to be 30% of the applied compressive load. On the other hand, in flexion up to 7°, virtually no load is carried by the facets. Beyond 7° to 8° of flexion, greater contact forces are carried by the facets. To minimize the risk of facet joint injury, extension exercise postures should be performed slowly and repetitions kept to a minimum.
2. Rotation: Rotation beyond the normal average range of 2.6° causes microdamage to the discal structures and impaction of the zygapophysial joints.
3. Axial loading.
4. Increased intradiscal pressure.

NUCLEUS PULPOSUS MOTION WITHIN THE ANULUS FIBROSUS ON FLEXION AND EXTENSION

Management of patients with low back pain is often based on theorized positional changes of the nucleus pulposus (NP) during spinal extension and flexion. Data describing NP positional changes have not been reported for noninvasive measurements.

Figure 2.130 shows the flexed lumbar position of an MRI study of lumbar intervertebral disc changes. Figure 2.131 shows the extended lumbar position for the MRI study of disc changes on flexion and extension motions.

Normal and Abnormal Nucleus Pulposus Move Differently within the Anulus Fibrosus on Motion Study

The distance of the posterior margin of the NP to the posterior margins of the adjacent vertebral bodies was greater in the extended position compared with the flexed position in a study by

Figure 2.130. The flexed position. The foam bolster has been placed under the subject's knees. The surface coil is 1 inch proximal to the subject's iliac crest. (Reprinted with permission from Beattie PF, Brooks WM, Rothstein JM, et al. Effect of lordosis on the position of the nucleus pulposus in supine subjects: a study using magnetic resonance imaging. Spine 1994;19(18):2096–2101.)

Figure 2.131. The extended position. The lumbar roll has been placed under the subject's low back. The subject's legs are extended. (Reprinted with permission from Beattie PF, Brooks WM, Rothstein JM, et al. Effect of lordosis on the position of the nucleus pulposus in supine subjects: a study using magnetic resonance imaging. Spine 1994;19(18):2096–2101.)

Beattie et al. (284) who found no difference in the anterior distance. Eight of the 20 subjects had at least one degenerative disc in the lower lumbar spine. The NPs of the degenerative discs did not move in the same manner as normal discs.

A lumbar roll used under the low back when supine caused an increase in the distance from the posterior margin of the NP to the posterior portions of the vertebral bodies in normal discs of healthy young women. Degenerative discs deform differently from nondegenerative discs.

Schnebel et al. (285, 286) used a digitizing technique to measure the position change of the NP from discograms obtained from subjects with low back pain. These subjects were studied in a flexed position (knees to chest) followed by an extended position (press-up extension). A significant difference in the posterior distance of L3–4, L4–5, and L5–S1 between flexion and extension for normal NPs was reported. Measurements of the mean difference of the posterior distance between positions at L4–5 and L5–S1 were greater (2.2 and 2.9 mm compared with 1.5 and 1.7 mm by Beattie), whereas their measurements at L3–4 were less (0.8 mm compared with 1.2 mm). The difference between flexion and extension at L4–5 and L5–S1 may have been greater in the Schnebel et al. Study because a comparison of the lumbar spine between the positions of "knees to chest" and "press-up extension" would presumably result in a greater position change in the NP than the positions that were used in the Schnebel study. Thus, although Beattie et al.'s findings would not appear to represent the absolute NP position change that would occur at the extremes of spinal flexion and extension, the difference between the Cobb angle in the two supine positions was significant. These results suggest that the NP deforms, and possibly moves within the IVD.

Abnormal Discs Show Little Difference in Position on Motion Study

Nuclear Motion Is Posterior in Extension in Abnormal Discs

In those subjects with an abnormal NP, little difference was found in the shape and location of the NP between positions. Similar observations were reported by Schnebel et al. In four of the eight subjects with degenerative discs, the nucleus pulposus of the involved segment was observed to "bulge" posteriorly in the extended position (284).

The concept that a motion segment that has a degenerative NP may not move in the same manner as a motion segment that has a normal NP may be important clinically. Because NP movement appears to differ between normal and abnormal IVDs, Beattie (284) questions whether nuclear movement can be used to justify the McKenzie approach when treating individuals with degenerative disc disease. In addition to degenerative disc disease, other disorders (e.g., herniated discs, bony abnormalities, and neuromuscular impairment) may influence NP displacement as a functions of position.

My opinion on these studies of nuclear motion within the anulus on flexion and extension motion is:

- The nucleus pulposus does not move anterior in flexion or extension
- The degenerated nucleus pulposus does not move anterior in extension

Lateral Flexion Studies

Patients with a relative increase in left lateral flexion improved more, both subjectively and physically, than did those with an increase to the right or those with no increase to either side. A right side shift was twice as common as a left side shift, and right side dominance increased by 1.8° with treatment, although only average improvement was noted in the patients by the physician.

Left side dominance of lateral flexion and a shift to the same side were associated with less back pain and better physical performance. Asymmetry of spinal lateral flexion, and probably of human body mechanism in general, should be noted in back pain studies on both the pathogenesis and treatment (287).

Intradiscal Pressure Decreases Under Distraction

Ramos and Martin (288) applied pelvic traction to the lumbar spine and measured the intradiscal pressure with a pressure transducer. Nucleus pulposus pressure dropped significantly to below −100 mm Hg.

Types of Disc Degeneration (Table 2.6)

Intervertebral Osteochondrosis

Intervertebral osteochondrosis is a common degenerative process involving the nucleus pulposus. With advancing age are

Table 2.6

The Five Types of Discogram and the Stages of Disc Degeneration They Represent

Discogram Type	Stage of Disc Degeneration
1. Cottonball	No signs of degeneration; soft white amorphous nucleus
2. Lobular	Mature disc with nucleus starting to coalesce into fibrous lumps
3. Irregular	Degenerated disc with fissures and clefts in the nucleus and inner anulus
4. Fissured	Degenerated disc with radial fissure leading to the outer edge of the anulus
5. Ruptured	Disc has a complete radial fissure that allows injected fluid to escape; can be in any state of degeneration

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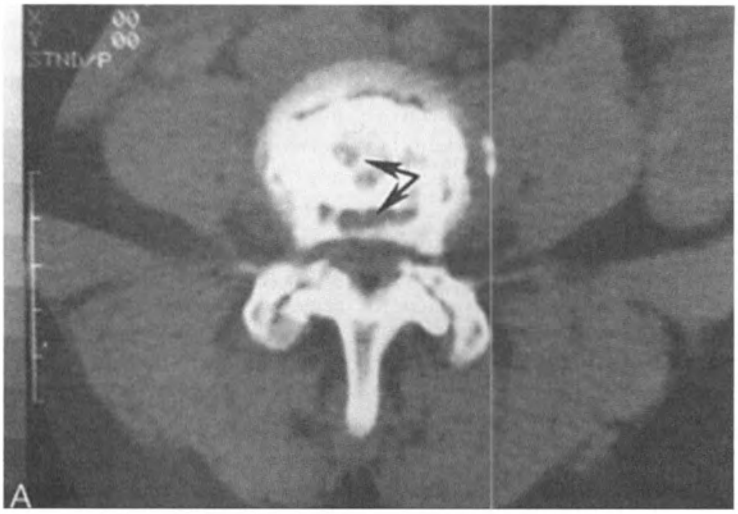

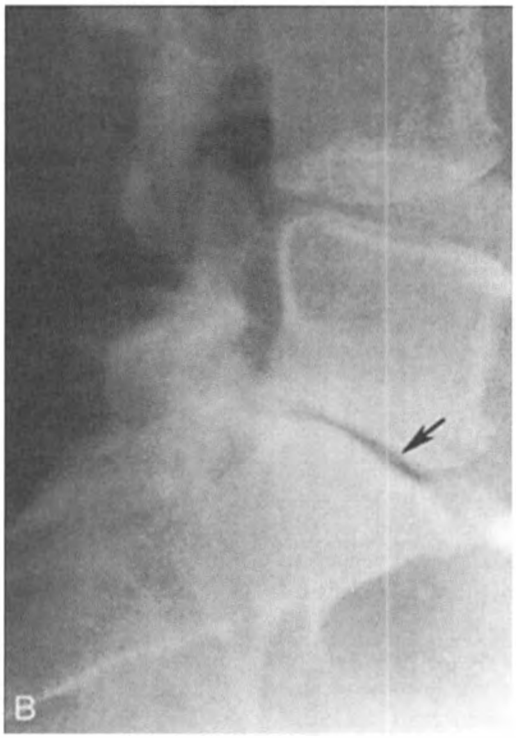

Figure 2.132. Osteochondrosis of the disc with vacuum phenomenon (*arrows*) as shown. **A.** On CT scan. **B.** On plain radiograph.

observed dehydration and desiccation of the intervertebral disc, particularly the nucleus pulposus. These changes begin in the second or third decade of life and become prominent in middle-aged and elderly individuals. The nucleus appears friable and loses the elastic quality it possessed in youth. It becomes yellow or yellow-brown in color, and the onion-skin appearance of the nucleus pulposus changes, developing cracks or crevices within its substances. The cracks produce an abnormal space into which surrounding gas, principally carbon dioxide and nitrogen, collects. The gas produces a radiolucency on radiographs or CT, an occurrence that is called a "vacuum" phenomenon (Fig. 2.132). The radiolucent collections are initially circular or oval, and they later elongate, extending into the anulus fibrosus. This vacuum phenomenon differs from the radiolucent collection at the margin of the intervertebral disc, which may accompany a different process (spondylosis deformans), and from those within the vertebral bodies (vacuum vertebral body), which may accompany ischemic necrosis of bone. A discal vacuum phenomenon generally excludes an infection (289).

As intervertebral osteochondrosis progresses, the intervertebral disc diminishes in height, the anular fibers bulge, and the cartilaginous end plates degenerate and fracture. Adjacent trabeculae in the subchondral regions of the vertebral bodies thicken. Radiographically seen at this stage are disc space loss and sclerosis of peridiscal areas of the vertebral body. The scle-

rosis is generally well defined. The discovertebral junction is usually sharply marginated, differing from the ill-defined margin that accompanies infection. An associated finding is a radiolucent focus within the vertebral body, which is a cartilaginous node (Schmorls' node) that is caused by intervertebral herniation of a portion of the disc through the degenerating cartilaginous end plate.

Pathologic and radiographic features of intervertebral osteochondrosis are most prominent in the lower lumbar region, and they are observed more commonly in men than in women.

With breakdown in Sharpey's fibers, the propulsive force of the nucleus pulposus leads to anterior and lateral displacement of the anulus fibrosus. Displacement elevates the anterior longitudinal ligament and traction at the site of ligament attachment to the vertebral body. This site is several millimeters from the discovertebral junction. Osteophytes resulting from the abnormal ligamentous traction course first in a horizontal direction before turning in a vertical one. Eventually, the osseous excrescences may bridge the intervertebral disc (289).

Nitrogen Gas Formation in Osteochondrosis Vacuum Change

Gas-containing cleftlike spaces in the intervertebral disc indicate a process of cartilage degeneration in most instances. Crevices formed in the degenerated disc cartilage become low-pressure spaces that attract gases from surrounding interstitial fluid. Chemical analysis of these gas collections in the disc space shows a 90 to 92% concentration of nitrogen. This is understandable considering diffusion gradients, solubility coefficients, and partial pressures of nitrogen, oxygen, and carbon dioxide. Nitrogen, a metabolically inert gas, is the dominant element trapped in any low-pressure space created in degenerated disc cartilage, distracted synovial joints, or aseptic bone necrosis. Similarly, when a vertebra undergoes collapse secondary to ischemic necrosis, the volume of bone is reduced, and cleftlike spaces are formed. Low pressure is found in these spaces, especially when hyperextension of the spine distracts the apposing surfaces of the cleft. Thus, the vacuum sign may become accentuated or appear only when the spine is in hyperextension (290).

Corticosteroid-Induced Aseptic Necrosis of Bone

The relationship between aseptic bone necrosis and long-term corticosteroid treatment is well established, especially in patients who have had chronic corticosteroid treatment. Biopsy-proved aseptic bone necrosis has been reported in vertebrae exhibiting the vacuum sign in these patients (290).

Criteria for Determining the Level of Disc Involvement

MacGibbon and Farfan (291) provide criteria to determine the level of disc degeneration by markings on a plain film study of the lumbar spine (Fig. 2.133). Basically, they state that when the intercrestal line passes through the upper half of the fourth lumbar vertebral body and when the transverse processes of L5 are well developed, the L4–L5 disc degenerates first. When the intercrestal line passes through the body of L5 and L5 has less developed transverse processes, the L5–S1 disc degenerates first. The higher the intercrestal line, the greater the risk of L4–L5 degeneration; the lower the intercrestal line, the greater the risk of L5–S1 degeneration. They further state that, if it is assumed that discs are injured and degenerate solely because of torsional strains, the high intercrestal line and long transverse processes become antetorsional devices, protecting the L5–S1 discs and indicating the likelihood of degeneration at L4–L5. Similarly, the low intercrestal line and small transverse processes, which provide no protection against torsion, indicate the likelihood of degeneration at either the L4–L5 or the L5–S1 disc. We would, therefore, expect a high odds ratio for L4–L5 disc degeneration in the protected spines and a more equal odds ratio with no protection.

Criteria for probable L4–L5 degeneration are:

1. A high intercrestal line passing through the upper half of L4.
2. Long transverse processes on L5.
3. Rudimentary rib.
4. Transitional vertebrae.

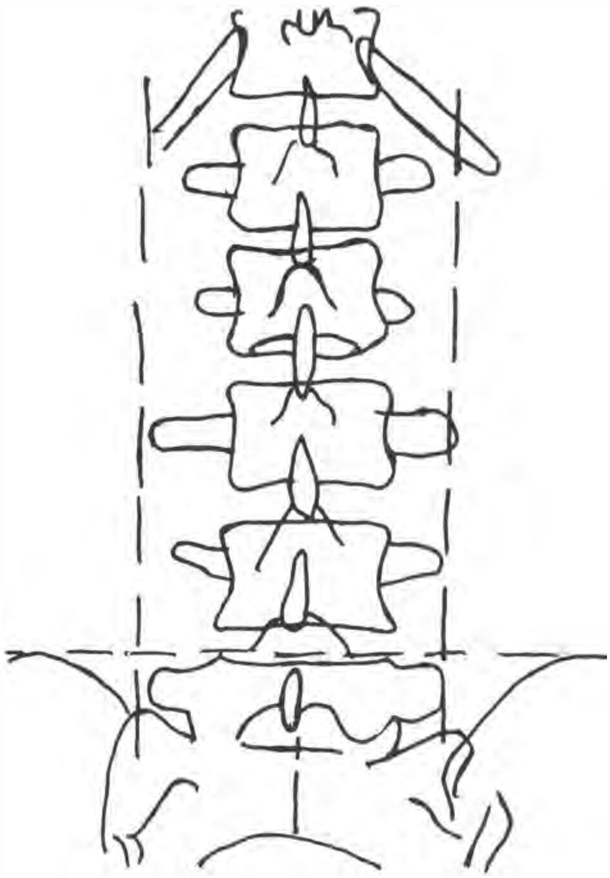

Figure 2.133. Intercrestal and transverse process lines drawn for determining probable level of disc degeneration.

The criteria for L5–S1 degeneration are:

1. An intercrestal line passing through the body of L5.
2. Short transverse processes on L5.
3. No rudimentary rib.
4. No transitional vertebrae.

Surgical fusion has an effect similar to that of the intercrestal lines described by MacGibbon and Farfan. Fusion places the mobility at the segment above the fusion, and it can cause disc degeneration at that level.

INSTABILITY DEFINED—ACUTE AND CHRONIC

Clinical instability of the spine exists when physiologic loads produce abnormal motion, major deformity, and incapacitating pain or significant neurologic deficit (292). Clinical instability can be divided into acute instability and chronic instability. Acute instability denotes an impending catastrophe, as in cases of severe trauma or tumors, that will destroy most spinal structural support. Chronic instability is the result of a prolonged degenerative process in which the pathomechanics are less clear, and the radiographic and clinical correlations are more difficult to establish.

Lumbar Vertebral Translation

Normal lumbar vertebral translation is less than 3 mm, or 8% of the adjacent superior vertebral body width. In 42% of normal subjects sagittal translation is at least 3 mm. Schaffer and Weinstein were reported to have concluded that a threshold of 4 to 5 mm of translation must exist for accurate measurement (292).

The earliest signs of degeneration in the aging spine appear at the intervertebral disc; they can start as early as the second decade of life. Torsional loading and resulting anular injuries have been especially implicated in the initiation of disc degeneration.

Strengthening exercises should be the basis for management of instability. Biomechanically, the abdominal musculature can absorb up to 30% of the load on the lumbosacral spine during lifting. Isometric abdominal exercises have been shown to improve symptoms significantly with minimal negative risk. "Low back school" has also proved effective. Manipulation has not been effective. A lumbosacral corset or brace may unload the spine by abdominal pressurization. The last resort in the management of degenerative spinal clinical instability is spinal fusion, although the indication for fusion is controversial (292).

Instability—Radiographic Findings

Lumbar instability has been reported to be most common at the L4–5 level and to be rare at L5–S1 because the facets between L5–S1 are normally coronally aligned and thus resist anterior sliding. Although instability cannot be diagnosed by plain films alone, the following findings have been associated with instability (293):

1. Retrolisthesis
2. Traction spur
3. Spondylolisthesis
4. Previous total laminectomy or fusion operation below the motion segment
5. Gas in the disc
6. Disc space narrowing
7. Facet degeneration
8. Malalignment of the spinous processes at the affected level
9. A rotational deformity of the pedicles

Flexion and extension of the lumbar spine are the most common radiologic diagnostic tools. Anteroposterior projection during maximal sidebending is an uncommon technique for the diagnosis of instability. More than two thirds (79%) of the instability cases are revealed by flexion-extension films, but only half (47%) by sidebending films.

During sidebending, normal axial rotation can cause the spinous processes to move from the central line toward the concavity of the curve because of a coupling movement. Pathologic axial rotation can be detected if the spinous processes move to the convex side, producing an asynchronous spinous process line. Pathologic rotation can also manifest as a lateral translation of one vertebra on another during sidebending. Additional signs of abnormal movement include asymmetric closure of the disc space on bending, and paradoxic opening of the disc space on the bending side.

Sidebending films should not be routinely combined with flexion-extension films in the radiologic diagnosis of segmental lumbar instability. Sidebending films complement flexion-extension films, and they should be taken if sidebending instability is clinically suspected, especially when flexion-extension films are normal (293).

Disc Degeneration Causes Increased Joint Laxity

In all three loading directions (flexion-extension, axial rotation, lateral bending), greater joint laxity was found with disc degeneration. This measure of joint laxity has significant promise in detecting disc degeneration (294).

End Plate and Bone Marrow Changes

End plate and adjacent bone marrow change on MRI show two abnormalities in degenerative lumbar disc disease:

1. Type A: with decreased signal intensities
2. Type B: with increased signal intensities

Type A changes correlated with segmental hypermobility and low back pain, whereas type B changes were more common in patients with stable degenerative disc disease (295).

DISC CHANGES WITH DEGENERATION AND OSTEOARTHRITIS

Nutritional Factors

Declining nutrition is the most critical event responsible for the changes in central disc cells and their matrices. Lactate concentration rises as a result of low oxygen tension and decreased rate of lactate removal. Decrease in pH level, which compromises cell metabolism and biosynthetic functions, can cause cell death (296).

An autoimmune basis for the coexisting cervical and lumbar spondylitic disease, namely the demonstration of antigenic properties in the nucleus pulposus and high serum immuoglobulins, is suggested as the reason for the dual cervical and lumbar disc surgeries seen in 31% of 200 patients (297).

Regeneration of Disc Tissue

Regeneration of disc tissue may be possible. Enzymatic removal of degenerated disc tissue, possibly combined with other strategies, including implantation of artificial matrices, growth factors, and mesenchymal or chondrocytic cells, has the potential to restore viable disc tissue. Critical factors in this regeneration include decrease in central disc nutrition caused by increasing the volume of avascular tissue with growth, loss of peripheral blood supply, and alterations in the matrix. The possibilities of slowing the rate of disc degeneration and regenerating central disc tissue require further study (296).

Proteoglycan Loss

Early stages of articular cartilage osteoarthritis, studied experimentally in the mature beagle dogs, showed an increased release of the proteoglycan aggregates with glycosaminoglycan of the articular cartilage (298).

Normal and degenerated discs show clear proteoglycan difference and the cartilaginous end plate participates in the process of aging and degeneration in the disc (299). Age decreases the signal intensity of normal lumbar intervertebral discs concomitant with decreases in water and glycosaminoglycans and increases in collagen in the disc. The proteoglycan content of the nucleus is about five times that of the anulus in young discs and decreases with age as the disc becomes more fibrotic. Figure 2.134 contrasts the signal intensity of normal versus degenerative discs (300).

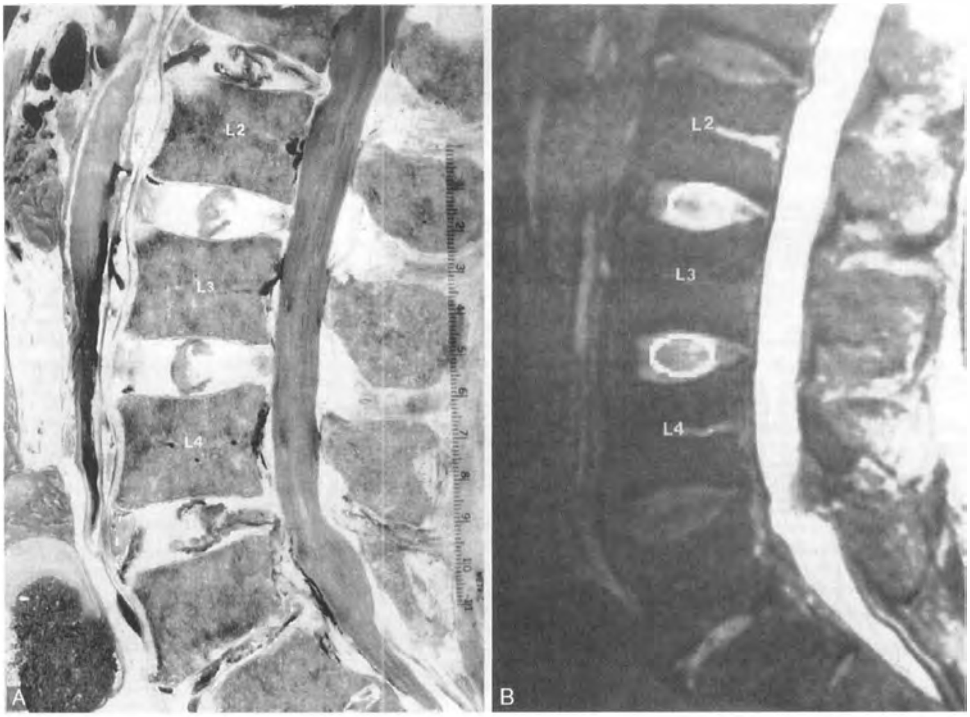

Figure 2.134. Sagittal anatomic section (**A**) and corresponding magnetic resonance (MR) image (**B**) of an adult lumbar spine. Normal adult discs are evident at L2–L3 and L3–L4, early degenerating discs at L1–L2 and L4–L5, and advanced degenerating discs at L5–S1. The MR image illustrates placement of cursors to measure signal intensity in the normal discs. Note the diminished signal intensity of L1–L2 and L4–L5 compared with that of discs at L3–L4 and L4–L5. Severely degenerated discs may have a low signal intensity, less commonly, of a moderately high signal intensity (e.g., L5–S1). (Reprinted with permission from Sether LA, Yu S, Haughton VM, et al. Intervertebral disk: normal age related changes in MR signal intensity. Neuroradiology 1990;177;385–388. Copyright 1990, Radiological Society of North America.)

Lactate Level Changes

The high lactate levels found in the central part of degenerated discs must be associated with the lowering of the pH, which in turn triggers and activates the proteolytic enzyme system. The intervertebral disc contains many different proteases; collagenase and elastase have recently been found. Also, protease inhibitors have been extracted from the human intervertebral disc. From the degradation of newly formed proteoglycans, however, it is evident that some degrading enzyme systems are present in the intervertebral disc matrix. In the healthy disc a delicate balance likely exists between the active and latent forms of enzymes and the potency of the inhibitors present; this balance could be pH sensitive. Hence, a change in the disc metabolism that decreases pH and could lead to a rapidly accelerating matrix break down (301).

Lactate Buildup

Disc metabolism is mainly anaerobic, and lactate concentrations in the center of the disc may be 8 to 10 times as high as in plasma; the pH in the disc center is thus acidic. Because low values of pH are known to affect proteoglycan synthesis in other cartilages, the effect of lactate levels and pH on S-sulfate and H-proline incorporation rates in the nucleus of bovine coccygeal discs and in human discs obtained during percutaneous nucleotomy showed the maximal incorporation rate occurred at pH 7.2 to 6.9 (302). Here the rate was 40 to 50% greater than at pH 7.4. Below pH 6.8 the rate fell steeply, more so for sulfate than for proline. At pH 6.3 the sulfate incorporation rate was approximately 20% less than at pH 7.4. Results indicate that proteoglycan synthesis rates, in particular, are sensitive to extracellular pH, with peak rates occurring at approximately the level of pH seen in vivo. Factors that cause lactate levels to rise, such as a fall in oxygen levels as the result of smoking or vibration, could lead to a fall in proteoglycan synthesis rates and ultimately to a fall in proteoglycan content and disc degeneration.

Loss of proteoglycan is the most marked change in disc degeneration. It would thus appear that a pH less than 6.8 would contribute to this change and cause disc degeneration partly because of the resulting change in cellular activity. It therefore is of interest that factors shown to increase intradiscal lactate levels experimentally (e.g., smoking) are also strongly associated with the development of disc degeneration (302).

High Levels of Interleukin-1

Interleukin-1's (IL-1) influence on degrading enzyme activity in normal and arthritic cartilage shows a strong trend to higher levels of IL-1 activity in degenerative and herniated disc tissue when compared with normal disc. These high levels of IL-1 could stimulate metalloproteases in the disc tissue, which, in turn, could cause disc degeneration (303).

Lipofuscin and Amyloid Buildup

When organ aging or atrophy is prominent, manifestation of age pigment (lipofuscin) occurs in the cardiac muscle and liver (304). Recently, amyloids were found to be present in aged individuals. Lipofuscin and amyloids build up in the degenerated lumbar intervertebral disc of surgical specimens and in individuals aged more than 50 years.

Biochemical Changes

The nucleus pulposus of injured sheep discs showed a significant loss of proteoglycans and collagen 8 months after a surgical incision was made in the anulus fibrosus, but recovered to within control values 6 to 8 months postoperative. The nucleus pulposus of discs adjacent to the incised disc also showed loss of collagen and proteoglycan; however, the anulus fibrosus matrix remained essentially unaffected. Loss of disc height and marked nucleus pulposus degeneration occurred within a few months (305). A loss of proteoglycans and water from the nucleus pulposus in the surgically damaged discs within 6 months of surgery is consistent with these histologic observations. Biochemical analysis would thus appear to represent a more sensitive means of assessing early disc degeneration. It is significant in this regard that low proteoglycan disc concentrations have been shown to precede morphologically assessed degeneration in the human spine (305).

Disc Degeneration in Adolescents with Low Back Pain

Frequency of disc degeneration was greater in 40 adolescents with low back pain (increased from 42 to 58%) than among 40 asymptomatic subjects (from 19 to 26%) (306).

End Plate and Vertebral Body As Sources of Pain

Intervertebral discs, with their sensory nerves and calcitonin gene-related peptide and substance P neuropeptides, are pain producing entities. These neuropeptides have potent vasodilatory effects in addition to their role as pain transmitters, which indicates an increased blood flow is probably the final neurovascular reparative attempt to increase the disc nutritional status. Such a changed profile and increase of sensory nerve fibers indicates that the end plate and vertebral body are sources of pain production. Nociceptor chemical sensitization and pressure changes caused by motility may partly explain the extreme pain experienced by patients with degenerative disc diseases (307).

Disc Morphologic Change

Disc incision of the anterior part of the anulus fibrosus of the L4–L5 disc in five domestic pigs (Fig. 2.135, *top*) had altered morphology at 3 months. The nucleus pulposus was small, fibrous, and yellowish (Fig. 2.135, *bottom*). The anular lamellar structure was partially destroyed and had been replaced by granulation tissue in the region of the injury. Large osteophytes had formed at the ventral edges of the vertebral bodies. In the nucleus pulposus, the total collagen concentration and the activities of enzymes active in collagen synthesis were significantly increased, whereas the water content had decreased (308).

In a sheep model, the creation of anular lesions caused im-

Figure 2.135. The morphology of the normal porcine disc is shown (*top*) at the time of introduction of a scalpel tear in the anterior anulus fibrosus (*arrow*) and the same disc 3 months after scalpel injury is shown (*bottom*). In the injured disc, the nucleus is fibrous and small; the anular lesion healed by formation of granulation tissue, but the lamellar structure has been partially destroyed. (Reprinted with permission from Kaapa E, Holm S, Han X, et al. Collagens in the injured porcine intervertebral disc. J Orthop Res 1994;12:93–102.)

mediate changes to the mechanics of the disc. A clear reduction was seen in torsion stiffness compared with controls, and clear evidence was found of a progressive degenerative response in the nucleus (309).

Common Types of Anular Defects

Three common types of anular defects have been described:

1. Rim lesions, which are defined as discrete defects of the outer anulus fibrosus.
2. Circumferential tears, more frequently seen in the lateral and in the posterior layers.
3. Radiating clefts, which are commonly seen in degenerating discs extending from the nucleus pulposus parallel or oblique to the plane of the end plates (310).

Peripheral Anular Tears

Peripheral tears are more frequent in the anterior anulus, except in the L5–S1 disc. Circumferential tears are equally distributed between the anterior and the posterior anulus. Radiating tears are in the posterior anulus, and they are closely related to severe nuclear degeneration.

Defects of the peripheral anulus fibrosus, which precede the dehydration and fraying of the nucleus pulposus, are likely to be caused by mechanical stress. These outer anular tears may influence and accelerate the degeneration of the intervertebral disc, and play a part in producing discogenic pain (311).

Incision of the outer anulus of a sheep disc resulted in lumbar disc degeneration within a short time. Complete healing of the incised outer anulus fibers was seen approximately 4 months after surgery, whereas the inner two thirds of the anulus showed no evidence of healing. Eventually the original incision extended inward, concentric clefts formed, and radiating tears occurred in the anulus with a degree of nuclear protrusion. The end plates of the sheep lumbar discs underwent extensive architectural changes shortly after outer anular injury, and these changes persisted for up to 2 years. The architectural changes observed resemble the sclerosis of subchondral trabecular bone seen in proximal femur osteoarthritis (312).

OSTEOPHYTE ROLE IN DISC DEGENERATION

Vascular Changes

Vascular changes occur before disc degeneration at every lumbar level, suggesting that disturbances in the nutritional supply may precede degeneration. Arteriolar vessels decrease in the posterior longitudinal ligament, whereas vascularity increases in the anterolateral vertebral space. The aging degenerative anulus shows increased vascularity with small thin-walled arteries that form clefts between layers of the anulus. Gradually, fibrous connective tissue with enlarged blood vessels form, which produces tears in the outer aspect of the anulus. Subsequently, osteophytes, consisting of cancellous bone rich in marrow cavities, replace the arteries. In the final stage, an osseous ankylosis between the adjacent vertebrae allows free communication of blood through the marrow cavities. It is reasonable to assume that vascular ingrowth is related to osteophyte formation near the periosteum beneath the cartilaginous end plate of the vertebral bone (313).

The anterior longitudinal ligament has a rich nerve plexus supply. Perivascular nerves supply the arteries, thus confirming that increased nerve supply caused increased vascularity. Coppes et al. (89) found nerve endings in abnormal discs that penetrated the anulus to reach the nucleus pulposus.

Disc Herniation Tissue Is Hypervascular

Herniated disc material is hypervascular with fibroblast growth factor promoting granulation tissue formation when stimulated chemically or secondary to an autoimmune response to the nucleus pulposus. Prolapsed disc tissue may disappear with time as vascularization of the herniated disc brings degrading agents to the disc (314).

Anular Damage Leads to Disc Degeneration

Traumatomechanical damage to the anulus fibrosus seems to alter the biomechanics and nutritional status of the whole disc,

leading to nuclear degeneration and aberrant collagen deposition. Anulus fibrosus healing is most active during the first month after injury and, although the nucleus pulposus may not be directly affected by the injury, its cell types change and start to synthesize, thereby increasing amounts of aberrant collagen types. An anular injury causes secondary cellular reactions in the nucleus pulposus (315).

MRI Studies

In a patient with no history of back pain, an MRI was performed on the day of an accident showing no abnormalities and indicating no disc pathology prior to the accident. A T2-weighted MRI 2 months after the accident showed a decline in disc signal intensity; extrusion became clearly visible on MRI 11 months after the accident. It is thought that the direct impact of an external force created a rupture (or incomplete rupture) in the anulus fibrosus, which represented a weak spot mechanically to release stress on the disc, leading to gradual extrusion of the nucleus pulposus. MRI showed that this was not a transient injury inflicted at the time of the accident, but rather one that developed over many months (316).

Subdiscal Bone Changes

Subdiscal bone has shown that deterioration accompanies disc degeneration. This bone change is in response to stress-adaptive properties of bone (Wolff's Law) as the well-hydrated nucleus loses proteoglycan macromolecules and develops a heterogeneous distribution of physical and mechanical properties in cancellous bone tissue. Intervertebral disc and vertebral bone properties are interdependent, which is an important implication in the degenerative processes in the spine and in the cause of low-spine pain (317).

End Plate Failure Starts Degeneration

The end plate is the weakest structure of the spinal segment (body-disc-body), and spinal failure always starts in the end plate and not in the anulus. The compressive load required to initiate failure in the anulus is approximately double that required to initiate a fracture in the end plate. The location of the initial failure is not affected by any initial tears in the anulus, and it always occurs in the end plate. Discrete peripheral tears in the anulus fibrosus may have a role in the formation of concentric anular clefts and in accelerating the degenerating process of the disc (318).

Nerve Innervation of the End Plate

Medullary cavities of vertebral bodies are innervated by both autonomic and sensory nerve fibers, with substance P and calcitonin gene-related peptide seen in perivascular nerve fiber plexuses of the vertebral bodies through the nutrient foramina into the disc. It is assumed that the end plate microcirculation supplies most of the nutrition to the nucleus pulposus and failure of nutritional supply leads to disc cell death and degeneration. Disc and end plate breakdown is accompanied by increased sensory nerve supply, which strongly suggests that the end plate and vertebral body are a source of pain. This may explain the severe pain on movement experienced by some patients with degenerative disc disease (319).

Sequestered Disc Material

Extruded disc material invariably is nucleus pulposus (54%) or end plate material (44%) in surgical specimens. Multiple and recurrent sequestered fragments almost always consist of end plate material. These findings may reflect the result of metabolic alterations in the course of disc degeneration (320).

Fragments of cartilaginous end plate are anulus fibrosus more often than nucleus pulposus in patients older than 30 years of age, especially in those more than 60 years of age (321). Avulsed end plate with anular anchoring occurs more often than herniation of the nucleus pulposus in the elderly. (322).

Schmorl's Nodes with Posterior Disc Herniations

Schmorl's nodes occur more frequently in patients with low back pain, and they are associated with posterior disc herniations most often at the L4–5 level. Schmorl's nodes are associated with increased disc herniation at the same level with increased age. They appear, therefore, to be a type of vertical disc herniation, an important pathognomonic condition, especially in young people (323).

Vibration Effects

Vibration affects the cancellous bone adjacent to the nucleus space with fatigue fracture of bone as a cumulative trauma and subsequent loss of nucleus content, which initiates the degenerative processes. Anulus fibers do not appear to be vulnerable to rupture when the segment is subjected to pure axial vibration (324). Whole body vibration causes increased height loss (325). A strong correlation exists between vibration and back pain as body vibration alters the normal neuropeptide profile seen in dorsal root ganglion neurons.

Nuclear clefting with mitochondria and rough endoplasmic reticulum free the ribosomes and lysosomal volumes crowded in the cleft spaces of vibrated cells (326).

FACET JOINT IN CAUSE OF LOW BACK PAIN

Fibrous Capsule, Ligamentum Flavum, and Multifidees Muscle

The outermost fibers of the facet fibrous capsule are intimately interwoven with the multifidi muscle's insertion on the lamellar process of the vertebrae (327). The outer layer of the fibrous capsule is dense regular parallel bundles of collagenous fibers and the inner layer consists of bundles of elastic fibers, similar to the ligamentum flavum (328) (Figs. 2.136 and 2.137). In the superior and middle capsule, the fibers run in the medial to lateral direction, crossing over the joint gap. In the inferior capsule, the fibers are relatively thicker and longer, and they run in a superior-medial to inferior-lateral direction, covering the inferior articular recess.

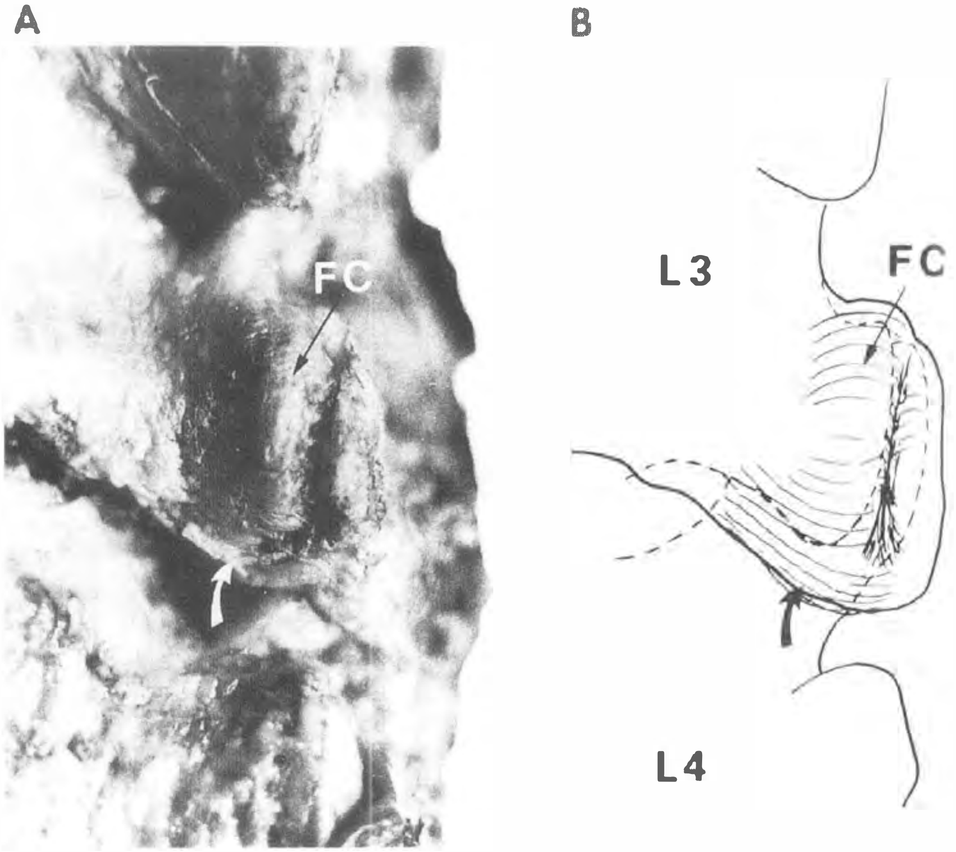

Figure 2.136. **A.** Posterior view of the right L3–L4 facet joint. The outer layers of the capsule were removed. **B.** Diagram showing the inner layer of the fibrous capsule of the facet joint. The bundles of fibers of the facet joint capsule (*FC*) run in the medial to lateral direction, crossing over the joint gap, and attach close to the joint margins. In the inferior part of the joint (*arrow*), the fiber bundles are longer and composed of thicker layers than those in the superior and middle parts of the joint. (Reprinted with permission from Yamashita T, Minaki Y, Ozaktay AC, et al. A morphological study of the fibrous capsule of the human facet joint. Spine 1996;21(5):538–543.)

The facet joint capsule is well innervated by fine nerve fibers, which may conduct nociceptive and proprioceptive sensations. Most nerve fibers and endings are located in the middle-lateral and inferior part of the capsule (328). Between the capsular ligament and the ligamentum flavum are elastic fibers from the ligamentum flavum, which are particularly abundant near the superior and inferior ends of the joint (328).

The L5–L6 rat facet joints are innervated by dorsal root ganglia and paravertebral sympathetic ganglia. L1 and L2 dorsal root ganglia receive sensory fibers from the facet joint via the sympathetic chain, and this pathway can explain anterior thigh and inguinal pain from facet irritation. Dorsal rami also innervate the facet capsule with each one supplying at least two facet joints; for example, L4–L5 facet joints would be innervated by the medial dorsal ramus branches from L3 and L4 spinal nerves in humans (329).

Pathologic findings in paraspinal muscles such as the multifidi are seen in electromyographic studies, and such findings can be caused by facet degeneration, which places compression on the dorsal ramus and innervates the laminar periosteum. Bending type injuries or hypermobility of the vertebral structures abnormally stress the dorsal ramus to result in pain (330).

Joint Capsule Mechanoreceptors

All the synovial joints of the body (including the apophyseal joints of the vertebral column) are provided with four varieties of receptor nerve endings (331).

Type I mechanoreceptors consist of clusters of thinly encapsulated globular corpuscles that are embedded (as three-dimensional bunches of grapes) in the outer layers of the fibrous joint capsule. They have a low threshold, so that they respond to small increments of tension in the part of the joint capsule in which they lie; some in each joint have such low threshold that they fire continuously even when the joints are immobile. Their response to sustained changes of tension in the joint capsule is one of slow adaption. When the joint capsule tension is increased by stretching (e.g., active movement or passive manipulation, or with traction), their frequency of resting discharge rises in proportion to the degree of change in joint capsule tension. Type I receptors, therefore, function as static and dynamic articular mechanoreceptors.

Type II mechanoreceptors are thickly encapsulated conical corpuscles embedded in the deeper layers of the fibrous joint capsule that abut the subsynovial tissue. They are inactive in immobile joints and emit only brief bursts of impulses (lasting less than 0.5 second) at the moment when joint capsule tension is augmented. Therefore, they behave exclusively as dynamic (or acceleration) mechanoreceptors.

Type III mechanoreceptors are much larger, thinly encapsulated corpuscles on the surfaces of joint ligaments, but they are absent from the ligaments of the vertebral column. They respond only when high tensions are generated in joint ligaments. Their discharge frequency is a continuous function of the magnitude of that tension no matter how it is generated (which is usually by powerful joint manipulation or the application of high traction forces).

Type IV receptor system is responsible for evoking joint pain when irritated. This unmyelinated nerve nociceptive plexus is contained in the entire thickness of the fibrous capsule, but it is absent from synovial tissue, intra-articular menisci, and articular cartilage. In collateral and intrinsic joint ligaments (and in the spinal ligaments), on the other hand, this nociceptive receptor system is represented by individual free unmyelinated nerve endings that weave between the fibers of the ligament. This receptor system remains entirely inactive in normal circumstances, and only becomes active when it is irritated by abnormal mechanical or chemical (as in joint inflammation) changes in the tissue in which it lies (331).

The facet capsule, but not the ligamentum flavum, is substantially innervated by sensory and autonomic nerve fibers, and it has a structural basis for pain perception (332).

Mechanoreceptor Location and Irritative Factors

Articular nerves contain myelinated and unmyelinated sensory afferent fibers and unmyelinated efferent sympathetic postganglionic fibers. Group II afferents are located in the fibrous capsule, articular ligaments, menisci, and adjacent periosteum, but not in the synovial tissue and the cartilage. Group III and IV fibers terminate as noncorpuscular or "free nerve endings" in the joint tissue. Noncorpuscular endings have been located in the fibrous capsule, adipose tissue, ligaments, menisci, and periosteum. Whether noncorpuscular sensory nerve endings are found in the synovial layer is disputed (333).

Chemical or Physical Stress of Joint Tissues Affects Receptors

Several inflammatory mediators (prostaglandins, thromboxanes, leukotrienes, kinins, and others) have been identified in synovial fluid. They are either produced by tissues in the joints

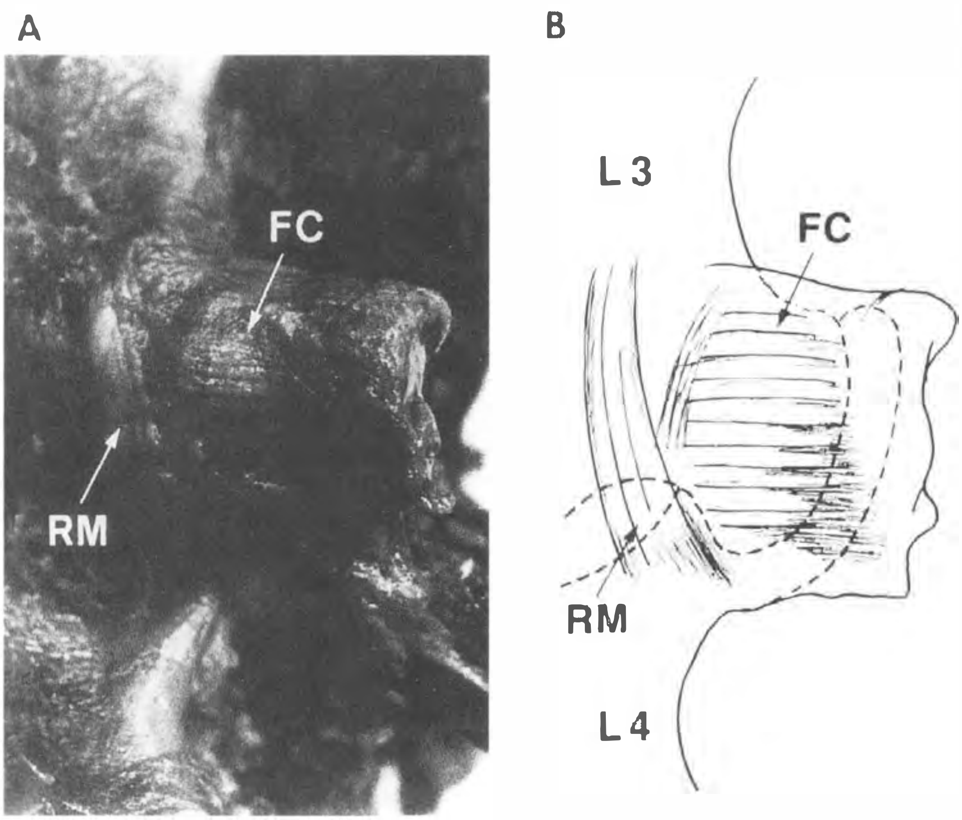

Figure 2.137. **A.** Posterior view of the right L3–L4 facet joint. **B.** Diagram showing the outer surface of the facet joint capsule. The ligamentous fibers of the facet joint capsule (*FC*) run in the medial to lateral direction. The tendinous band of the rotatores muscle (*RM*) (i.e., the deepest layer of the multifidus muscle) lies on the medial part of the capsule. (Reprinted with permission from Yamashita T, Minaki Y, Ozaktay AC, et al. A morphological study of the fibrous capsule of the human facet joint. Spine 1996;21(5):538–543.)

or are released during joint inflammation. Substance P causes articular plasma extravasation and cell inflammatory response (333, 334). Substance P nerve fibers within subchondral bone of degenerative lumbar facet joints implicates this type of joint in the cause of low back pain (335, 336, 336A).

Type II Mechanoreceptors

Chiropractic adjustment greatly affects type II mechanoreceptors. Type II mechanoreceptors are the most abundant type found in cervical facet capsules, proving that these tissues are monitored by the central nervous system. This implies that neural input from the facets is important to proprioception and pain sensation in the cervical spine. Previous studies have suggested that protective muscular reflexes modulated by these types of mechanoreceptors are important in preventing joint instability and degeneration (337).

Mechanical loading of the lumbar spine, which results in posterolateral bending, activates low- and high-threshold sensory fibers of the facet capsule, which may play a role in initiating facet joint syndrome (338). A "degenerative cascade" concept suggests that facet osteoarthritis may follow disc degeneration (339).

Facet Joints in Low Back Pain

Although lumbar zygapophysial joint pain admittedly exists, it cannot be clinically identified without diagnostic block (340). Although the lumbar facet joints are important biomechanically, the facet is not a common or clear source of significant pain. The facet syndrome is not a reliable clinical diagnosis. Intra-articular saline injection into the facets in control cases is as effective as local anesthetic and steroids in temporarily relieving the patient's pain (341). However, facet syndrome low back pain is frequently referred into the groin, hip, or thigh. It occasionally radiates below the knee but not into the foot. Pain is generally a deep, dull ache that is difficult to localize (341).

Pain Provocation

Single, uncontrolled, diagnostic blocks are unreliable as a diagnosis criterion because they carry a 32% placebo rate and a 38% false–positive rate. Therefore, correlating provocation to single blocks is, at best, capricious and, at worst, meaningless. Pain provocation does not identify those joints that respond to double blocks. Pain provocation as practiced in the context of lumbar zygapophysial joint pain does not control for false–positive response and, for that reason, cannot be used as a diagnostic criterion (342).

CT Value As a Diagnostic Test for Facet Joint Pain

No demonstrable relationship is seen on CT between the degree of osteoarthritic change and zygapophysial joint pain. Whatever the cause or mechanism of pain, it is not evident on CT, and it is not expressed in terms of the established, radiographic features of osteoarthritis. It must be concluded that CT has no value as a diagnostic test for lumbar zygapophysial joint pain (343).

Decreased Nuclear Pressure

A general, three-dimensional static nonlinear finite-element model has been used to analyze the effect of change in nucleus fluid content on the mechanics of a lumbar segment subjected to various combined loads. The results show:

1. Intradiscal pressure rises with a gain and diminishes with a loss in fluid content.
2. Fluid content loss markedly increases the facet contact forces and reduces the tensile force in the anulus layers, especially those situated in the inner layers. Reverse trends are predicted when the fluid volume is increased.
3. Except under combined extension and compression loading, segmental overall rigidity increases with fluid gain and lessens with fluid loss.
4. Nucleus fluid loss tends to cause inward bulge at the inner anulus layers.
5. Nucleus fluid plays a major role in segmental mechanics by carrying a portion of the applied compression, stressing the anulus layers, and supporting the surrounding anulus layers from bulging inward.
6. Disc fluid loss disrupts the normal function of the disc nucleus and predisposes the facets to additional loads, the anulus to possible instability and disintegration, and the vertebral body to bone remodeling (344).

Abnormal loading of the facets, either primarily or as a consequence of disc degeneration, may accelerate their degeneration and cause low back pain (345).

OTHER FACTORS IN BIOMECHANICALLY INDUCED LOW BACK PAIN

Sex

Overall, men have a mean total of seven osteophytes (range, 0 to 49) and women a mean total of three (range, 0 to 43) (346).

Diurnal Loss of Height Loads the Facet Joints

Simulated diurnal volume mean decrease in the lower three lumbar discs is 16.2% (1% of standing body height). Most of the diurnal loss in disc height is caused by volume loss (347).

Intervertebral distances between L1 and L4 are significantly greater in the morning than in the evening. Average diurnal change in the total intervertebral distance L1–L4 is 5.3 mm (348). Intervertebral disc height decreased significantly between 8 am and 1 pm, with little change from 1 to 6 pm. The greatest change is noted at the L4–L5 level (349). On MRI patients showed decreased signal:noise ratio from morning to evening because of dehydration (350).

High Heels Reduce Lumbar Lordosis in Men

A significant trend toward decreasing rather than increasing lumbar lordosis with progressively higher heels was found only among male subjects. No trend in either direction was found among female subjects. These and other results suggest that the greatest compensation for heel height occurs distally (351).

Immobilization

After immobilization, an increase (extension 62%; flexion 85%; left bend 30%; right bend 26%) in motion at the adjacent segment was found for all motions. For all configurations, the facet contact site impinged in extension, remained unchanged in left bending, and moved superiorly in right bending (352).

Surgical immobilization of long segments of the spine increases the load and motion both at the immediate adjacent segment and at the distal segments (353).

Chondromalacia Facetae

Even in young adults, many facet joints show ulceration or severe fibrillation, and this seems to remain constant throughout adult life (Fig. 2.138). Disc degeneration is an age-related finding, usually occurring with greatest frequency in older age. Thus, it is plausible that facet cartilage degenerates early in life, which leads to back pain unrelated to the age-related changes that occur in the disc (373).

Single Photon Emission Computed Tomography (SPECT)

Facet joint disease is often seen on SPECT as in the following case of a woman scanned to evaluate possible metastatic disease from breast carcinoma. Following the administration of

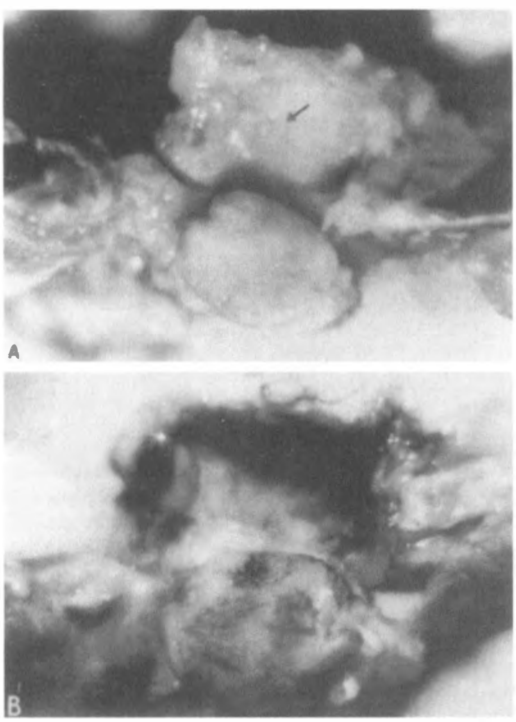

Figure 2.138. Examples of the surface appearance of facet cartilage after staining with India ink. **A.** Intact and superficially fibrillated facet. **B.** Deep fibrillation and ulcer. (Reprinted with permission from Ziv I, Marouda C, Robin G, et al. Human facet cartilage: swelling and some physicochemical characteristics as a function of age. Part 2: age changes in some biophysical parameters of human facet joint cartilage. Spine 1993;18(1):136–146.)

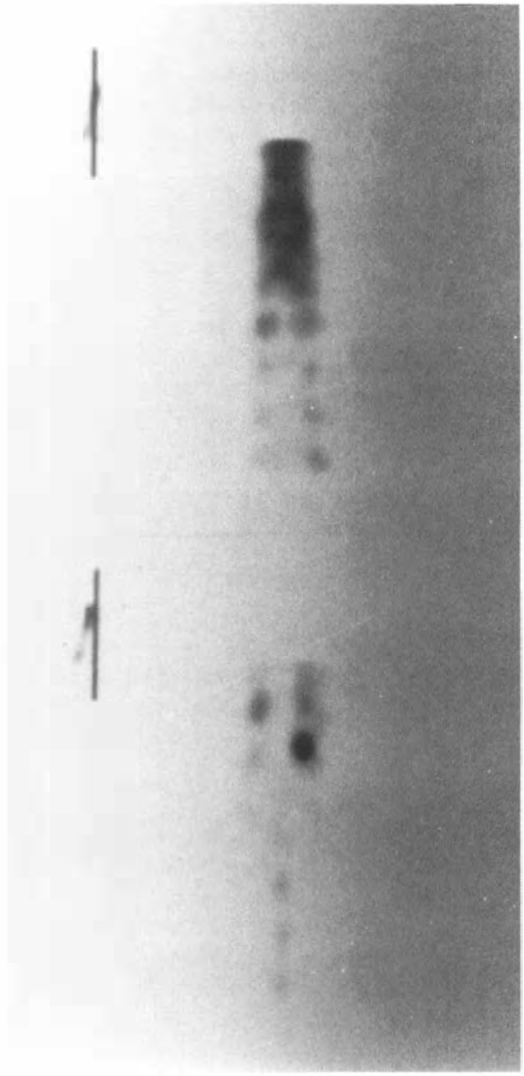

Figure 2.139. Planar radionuclide bone scan shows nonspecific uptake of the radioisotope within the T9–T10 vertebra.

22.0 mCi of ^{99M}Tc, multiple camera images of the body were performed.

Low-grade increased accumulation of radioisotope was seen within the lower thoracic region at approximately the T9–10 level on the left side (Figs. 2.139 and 2.140). SPECT imaging was obtained for further evaluation. The increased accumulation of radioisotope at the T9–10 level on the left side is posterior (Fig. 2.141), and it is likely within the facet joint. Therefore, the increased accumulation of radioisotope is most likely secondary to degenerative facet joint disease.

Single photon emission computed tomography is superior to planar radionuclide bone imaging in selecting patients for facet injection intervention for pain relief. Forty-three patients with the appearance of potentially symptomatic facet joints on planar and high-resolution SPECT radionuclide bone imaging were studied to relate the relative sensitivity of the two techniques and assess the predictive value in a clinical setting. Findings were high sensitivity (100% SPECT, 71% planar), but

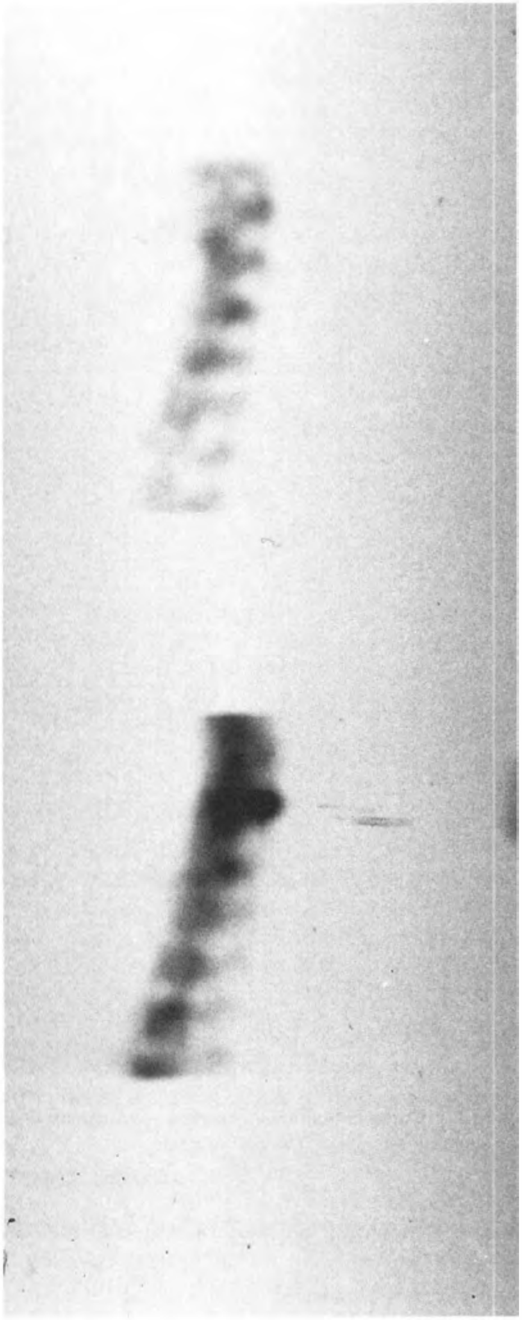

Figure 2.140. As in Figure 2.139, the planar bone scan shows nonspecific uptake of the radionuclide.

somewhat lower specificity (71% SPECT, 76% planar). The negative predictive value was high (100% SPECT, 93% planar). Radionuclide bone imaging additionally discovered nonfacet joint cause for patient symptoms in 16 of the 43 patients. Higher spatial resolution SPECT images are better accepted by referring physicians who correlated them with CT scan or MRI. The high negative predictive value allows radionuclide bone imaging to be used to select appropriate patients for the invasive facet injection procedure (354).

Diagnosis of Facet Fracture Following Hyperextension Rotation Injury

A common cause of back pain in athletes and dancers is stress injury to the posterior vertebral elements of the lumbar spine. A 36-year-old ballerina injured herself in a dance involving repetitive hyperextension and spine rotation. Nonprescription analgesics, massage, and chiropractic manipulation did not improve her condition.

A SPECT scan demonstrated increased tracer uptake in the L4 posterior elements. A CT scan showed normal pedicles, pars interarticularis sclerosis, and irregularity in the dorsal contour of the left L4 inferior facet. Stress fracture of the pars interarticularis became the working diagnosis.

Bracing in a 15° Boston Overlapping Brace gave relief; after 8 weeks, however, pain continued on extension or dancing. Repeat SPECT at 1 year showed the left L4 abnormality and CT showed facet degeneration. Anesthetic block provided temporary relief, but 16 months after presentation pain continued. Surgical exploration showed a left L4 inferior nonunion articular facet oblique fracture through the facet. Two months after surgery, the patient began dancing without restrictions, and she was asymptomatic 2 years after surgery. SPECT scanning provides high specificity and sensitivity in diagnosing stress injuries to the posterior vertebral elements (355).

PIRIFORMIS SYNDROME

Piriformis syndrome is a little known entity in which injury to the piriformis muscle results in buttock pain, often associated with leg pain. It is probably more common than has been recognized. Higher resolution MRI may visualize local areas of scarring or edema within the piriformis muscle, and it offers some hope for objectively documenting severe cases (356).

Figure 2.141. Here, the uptake within the facet articulation is demonstrated on SPECT, and it suggests facet degenerative change as opposed to metastatic disease.

Symptoms

The primary symptom of piriformis syndrome is buttock pain, with or without posterior thigh pain, that is aggravated by sitting or activity. Associated low back pain suggests involvement of other structures (e.g., facet joints or iliopsoas muscles). In an isolated piriformis syndrome, the major findings included buttock tenderness from the sacrum to the greater trochanter, piriformis tenderness on rectal or vaginal examination and reproduction of buttock pain on prolonged hip flexion, adduction, and internal rotation. Because of the location of the piriformis muscle deep in the pelvic floor, a female patient may also present to her gynecologist with synpareunia or to a gastroenterologist with rectal pain exacerbated by bowel movements. It can also be a complication of pelvic, hip, or other surgery caused by rough handling during anesthesia, extreme or unusual positioning of the hips, or prolonged weightbearing on the buttocks during the surgical procedure. Minor findings include leg length discrepancy, weak hip abductors (possibly, a positive Trendelenburg sign), and painful hip abduction against resistance while sitting. External rotation of the hip on lying supine has also been noted (356).

Sciatic Nerve Relationship with the Piriformis Muscle

The origin of the piriformis muscle is the anterior sacrum and the gluteal surface of the ilium near the posterior inferior iliac spine and the capsule of the sacroiliac joint; it inserts the greater trochanter upper medial border. This muscle acts as an abductor and external rotator of the hip joint. Double insertion of the piriformis muscle is seen in 10 to 15% of persons and the sciatic nerve or its peroneal division passes through the split piriformis muscle.

Bipartite Piriformis Muscle Compression of the Sciatic Nerve

Case 3

A 28-year-old housewife had chronic aching pain in her left buttock radiating to the posterior thigh for 3 years. Progressive ankle and toe extensor weakness, and intermittent claudication developed in 3 months. She had been treated with nonsteroidal anti-inflammatory drugs, ultrasound, diathermy, and physiotherapy without improvement (357). She held her left lower extremity externally rotated and her left gluteal muscles were slightly atrophic. Tenderness at the left notch was elicited by external palpation and by rectal examination. Straight leg-raising and Laseque tests were both positive. Weakness of the ankle and toe extensors was evidenced by a drop foot. Both knee and ankle jerks were normal. Hypesthesia and numbness were noted in the distribution of the peroneal nerve. Computerized tomography of the pelvis showed a hypertrophic piriformis muscle on the left side (Figs. 2.142–2.144)

On surgical exploration, the piriformis muscle was found to be bipartite with a larger upper two thirds and a smaller lower one third; both parts blended as a conjoint tendon inserting into the piriformis fossa at the medial aspect of the upper border of the greater trochanter. The sciatic nerve, inferior gluteal nerve,

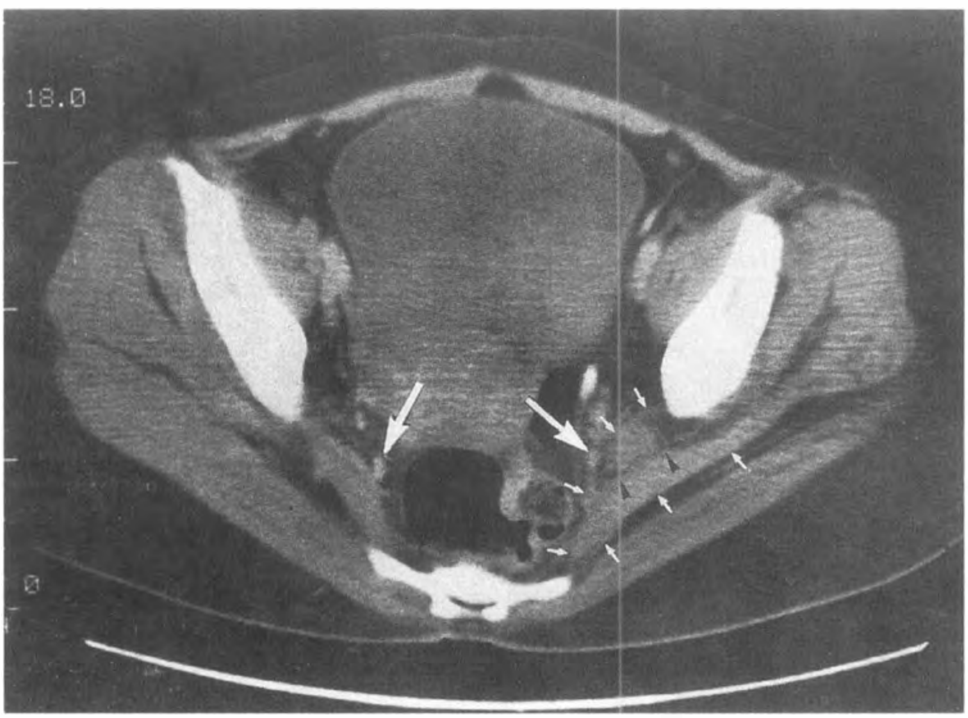

Figure 2.142. The computed tomography scan of the pelvis showing the hypertrophic bipartite piriformis muscle. The piriformis is outlined by *small white arrows*. *Black arrows* identify the sciatic nerve. *Large white arrows* show the intramuscular septum. (Reprinted with permission from Chen WS. Bipartite piriformis muscle: an unusual cause of sciatic nerve entrapment. Pain 1994;58:269–272.)

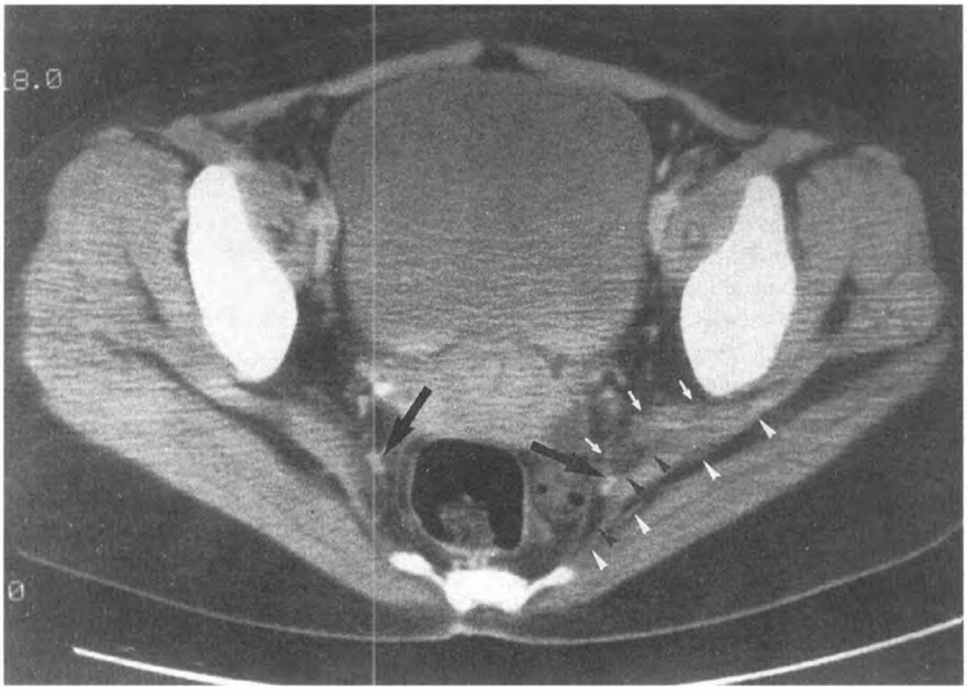

Figure 2.143. The left sciatic nerve (*black arrows*) traversing the intramuscular septum (*black arrowheads*) within the piriformis muscle (outlined by *white arrows* and *arrowheads*) is shown. The nerve is anterior to the upper portion of the muscle (*white arrowheads*) and posterior to the lower portion of the muscle. (Reprinted with permission from Chen WS. Bipartite piriformis muscle: an unusual cause of sciatic nerve entrapment. Pain 1994;58:269–272.)

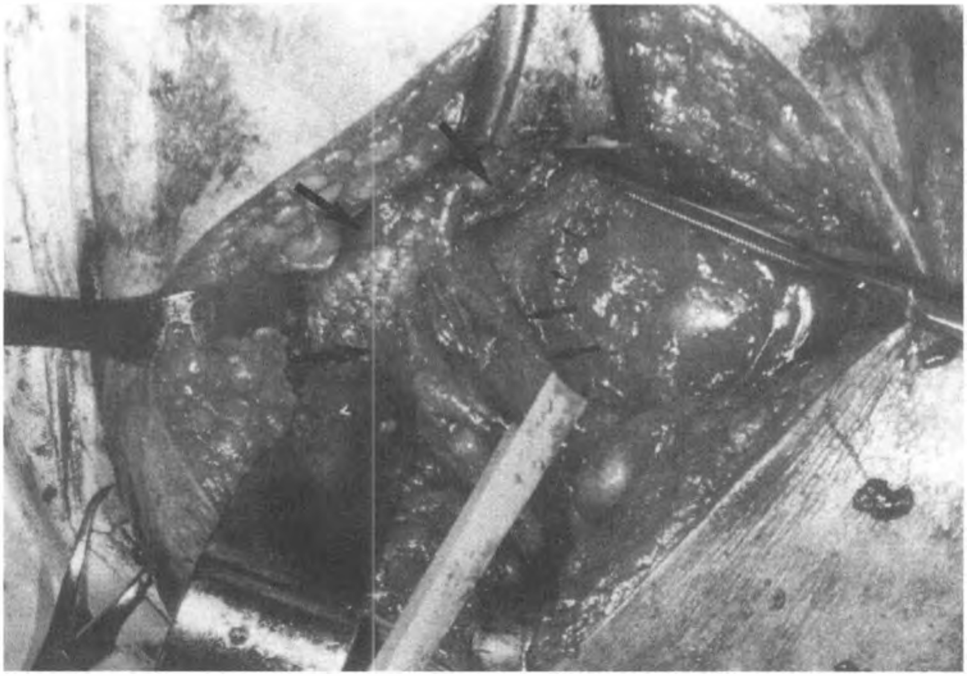

Figure 2.144. The intraoperative photograph showing the sciatic nerve and its accompanying vessels (*arrows*) traversing the piriformis muscle, which was bipartite with a larger upper portion (*large arrows*) and a smaller lower portion (*small arrows*). (Reprinted with permission from Chen WS. Bipartite piriformis muscle: an unusual cause of sciatic nerve entrapment. Pain 1994;58:269–272.)

and inferior gluteal vessels traversed the septum between the two parts of the muscle and were entrapped. The lower part of the piriformis muscle was dissected and brought posterior to the sciatic nerve and sutured to the upper portion. Sciatic pain was completely relieved postoperatively. Weakness of the ankle and toe extensors resolved in 2 years (358).

In another surgical exploration of the sciatic nerve, a fibrous constricting band around the nerve and a piriformis muscle lying anterior to the nerve was documented. Subsequent sectioning of the anomalous muscle and the constricting band yielded complete resolution of the patient's symptoms (358).

Superior Gluteal Nerve (SGN) Entrapment Syndrome

The SGN, which is derived from the posterior branches of the fourth and fifth lumbar and the first sacral nerves, lies between the gluteus minimus and gluteus medius muscle fibers. It can become entrapped as a result of trauma or abnormal posture. Increased lumbar lordosis with internal rotation of the hip can press the piriformis muscle against the ilium and the inferior fibers of the gluteus minimus, entrapping the nerve. A clinical triad of presentation is aching gluteal pain, a profound weakness of the hip abductor muscles, and tenderness to deep palpation in the region just lateral to the greater sciatic notch (359).

Piriformis Bursitis-Induced Sciatica

Case 4

A 73-year-old woman was admitted to the hospital with a history of several weeks of increasing pain in her right knee and right hip. She was being considered for total hip and knee replacements. The femoral head was grossly deformed. The acetabulum was mildly deformed with hypertrophic bone production and sclerosis. A single contrast arthrogram of the right hip showed an enlarged and irregular joint space.

Contrast medium filled a large, irregular, saclike space within the pelvic cavity that communicated with the medial aspect of the hip joint. As this cavity filled, the patient complained of increasing pain in her right knee identical to that which she had been having. CT scan confirmed the presence of contrast medium in an enlarged right piriformis bursa, which compressed the right sciatic nerve and caused it to deviate from its normal course.

Diagnosis was piriformis bursitis causing sciatic neuropathy. The patient was managed conservatively with some improvement, and she was discharged 3 weeks after admission. Enforced bed rest during her hospital stay probably allowed spontaneous resorption of joint and bursal fluid to occur (360).

BIOMECHANICAL FACTORS IN CHIROPRACTIC MANIPULATION

Stenosis Reversal

Reversal of spinal stenosis was tested by distraction of ten cadaveric motion segments, and stenotic narrowing of the intervertebral disc and facet subluxation were reversed. Decompression of the foraminal space was statistically significant in 7 of 10 cadaveric specimens after 5 mm of distraction, and in 9 of 10 specimens after 10 mm of distraction. Minimal yet insignificant improvement in stenotic canal area was evident with distraction (361).

The intervertebral foraminal shape is oval when the intervertebral disc is normal, and auricular shaped when abnormal. Foraminal size varied from 40 to 160 mm^2 with great variation even at individual levels (362).

Manipulation Side Posture Adjustment Effect on Myofascial Point Relief

Thirty subjects aged 18 to 50 years with chronic mechanical low back pain were randomized into two groups. One group received manipulation and the other received mobilization. Manipulation was performed in the side-lying position. The mobilization procedure consisted of an assisted supine knee-to-chest maneuver.

Pain-pressure threshold of selected myofascial points were measured before, immediately after, and 15 and 30 minutes postintervention. Three myofascial points selected for measurements were:

1. Over the erector spinae muscle at the L5 level, located 4 cm lateral to the ipsilateral L5 spinous process.
2. Over the posterior sacroiliac ligament, located 1 cm medial to the most prominent part of the ipsilateral posterior superior iliac spine.
3. Over the gluteus muscle group, located by the intersection of a line joining the ipsilateral posterior and anterior iliac spines and a perpendicular line originating from the most lateral aspect of the ipsilateral ischial tuberosity.

Repeated measured analysis of variance for all locations failed to show clinical or statistical significance. The overall effect between treatments and the interaction between treatment and time was not significant (363).

Chemical Inflammation of the Nerve Root

One final method of aggravation of the nerve roots in the lumbar spine that can result in sciatic pain is chemical radiculitis. Regardless the form of manipulation given, the chemical irritation of the nucleus pulposus to the nerve root is important. Degenerative disc disease may produce an autoimmune mechanism as a prolonged cause of pain. Marshall and Trethewie (364) consider the acute disc pain to be caused by local irritation of the nerve root by edema and release of protein and H substance at the site of disc injury. Auto-antibodies to autogenous nucleus pulposus have been experimentally produced in rabbits.

Manipulation and Flexion-Extension Exercises

The relative effectiveness of an extension program and a manipulation program with flexion and extension was examined

in 49 patients with low back syndrome seen at physical therapy clinics. The rate of positive response was greater in the manipulation/hand-heel rock group than in the extension group (365).

SHORT-LEG BIOMECHANICS AND BASIC CORRECTION

The possible association between pelvic obliquity and low back pain was investigated in low back pain patients and a control population (366).

The clinical importance of leg length inequality depends on the degree of the inequality and its relationship with a number of conditions and problems:

1. A possible correlation between the resultant pelvic obliquity and any degenerative changes in the lumbar spine (e.g., arthrosis, spondylosis).
2. A possible association with low back pain.
3. A correlation with hip joint degenerative changes.
4. A correlation with knee joint degenerative changes—"long leg arthropathy."
5. Psychological difficulties associated with the esthetic consequences of the postural deformity.

However, most patients with leg length inequality of 1 cm or more have no known cause for this inequality, which arises during normal growth without any apparent pathology.

Radiographic asymmetric structural changes in the lumbar spine, which appear to be correlated with pelvic obliquity and the consequent postural lumbar scoliosis, were described in two groups of nonacute low back pain patients: those with a leg length difference of greater than 9 mm, and those with no leg length difference (0 to 3 mm). With leg length inequality, concavities in the end plates of lumbar vertebral bodies, wedging of the fifth lumbar vertebra, and traction spurs appear (367).

Disc Protrusion on Long-Leg Side

In 700 patients, aged 14 to 89 years, with chronic low back pain, the incidence of leg length inequality (LLI) was two to five times that observed in the symptom-free control group. In a series of 228 cases of sciatica, the pain radiated to the longer leg in 78%. In 241 cases, the chronic unilateral hip pain symptoms and arthrotic changes were located on the long-leg side. In 73% of 180 cases with chronic unilateral knee symptoms and arthroses, the symptoms were found on the short-leg side (368).

These observations can logically be interpreted by the biomechanical effects of LLI on the musculoskeletal system. Pelvic tilt caused by LLI is generally compensated with a functional scoliosis convex to the short-leg side. During bending of lumbar motion segments, the discs are compressed on the concave side of the curve, and they put a tensile load on the opposite side. On the compression side, the disc bulges. In the case of LLI, the disc bulges in the posterolateral direction toward the spinal nerve root on the long-leg side. Lateral bending of the lumbar motion segment is always coupled with an axial rotation, so that the posterior elements tend to rotate toward the concavity of the curve. These complicated bending and torsional loads on lower intervertebral joints, ligaments, and, especially, on discs may be causative factors for low back symptoms associated with LLI (368).

Hip Osteodegenerative Arthritis on Long-Leg Side

Present knowledge of hip biomechanics supports the contention that the stresses imposed on the hip on the side of the longer leg are greater than normal; those on the short side are comparably reduced. Indirect measurements have demonstrated greater stress on the hip if the pelvis is adducted, a persistent and chronic condition of the hip joint on the side of a long leg. Furthermore, the pressure on the acetabulum will be displaced laterally in those circumstances. The consistent pattern of degeneration in unilateral superolateral osteoarthritis of the hip is what would be expected if the consequences of leg length disparity were as described. Leg length inequality may be a major contributing factor in the development of this type of unilateral degenerative hip disease (369).

Gauging Leg Length Inequality

The postural considerations outlined in the discussion of x-ray methodology are useful in clinically estimating leg length disparity. The examiner sits behind the patient who stands with the feet parallel and about 7 inches apart. The patient should stand erect and look forward, not downward. The knees must be straight and the pelvis centered over the feet. If significant leg length disparity exists, three observations will be made: (*a*) the upper lateral thigh on the long side will protrude; (*b*) scoliosis will be apparent; (*c*) the examiner's hands placed on top of the iliac crests will rest at different heights. All three of these findings should be present to accurately estimate disparity.

Next, place under the foot of the presumed short side, a

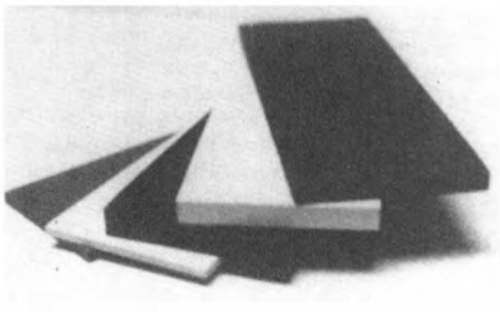

Figure 2.145. Lifts used in corrective procedures. (Reprinted with permission from Aspergren DD, Cox JM, Trier KK. Short leg correction: a clinical trial of radiographic vs. non-radiographic procedures. J Manipulative Physiol Ther 1987;10(5):233–237. Copyright by the National College of Chiropractic, 1987.)

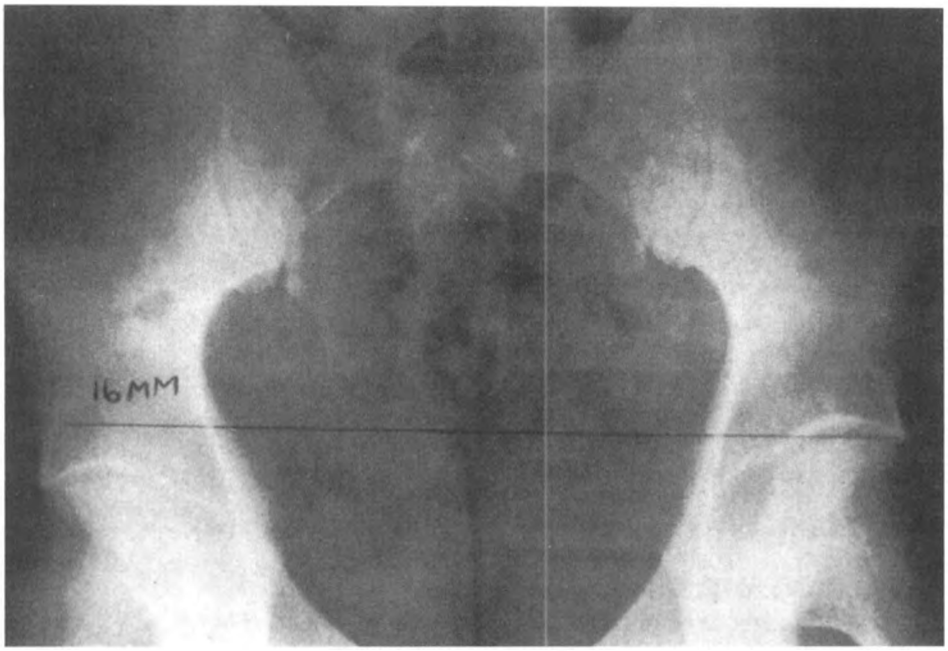

Figure 2.146. Radiograph demonstrating 16-mm difference in femoral head height. (Reprinted with permission from Aspergren DD, Cox JM, Trier KK. Short leg correction: a clinical trial of radiographic vs. non-radiographic procedures. J Manipulative Physiol Ther 1987;10(5):233–237. Copyright by the National College of Chiropractic, 1987.)

block of an appropriate thickness (e.g., 1/4 inch, 3/8 inch, 1/2 inch) and repeat the observations. The thighs should now be symmetric, the spine straight, and the hands level.

Finally, place the same block under the presumed longer leg. The three observations originally made should now be exaggerated. Unless these simple checks confirm the initial observations, the estimate of leg length disparity is in doubt. The size of the block necessary to bring the pelvis to an appropriate level is an indication of the amount of disparity (369).

Short-Leg Incidence and Correction

In 576 low back pain cases, I found 11% had a leg length discrepancy of more than 6 mm after maximal correction of the patient's mechanical faults and maximal improvement attained (370). In that study, a minimal shortness of 6 mm of one femoral head was corrected with heel or heel and sole lift; with up to 9 mm difference, a heel lift was inserted; and with more than 10 mm difference, an entire lift was placed on the heel and 5 mm less under the sole of the shoe. Also, for reasons of vanity, up to 9 mm could be placed inside the shoe with any lift over 9 mm placed under the heel and sole.

Correction of Leg Length Disparity

To outline the treatment protocol used in our clinic to correct leg length disparities, a study will be summarized in which visual leg length insufficiency detection and correction were compared with established radiographic procedures on 41 consecutive patients.

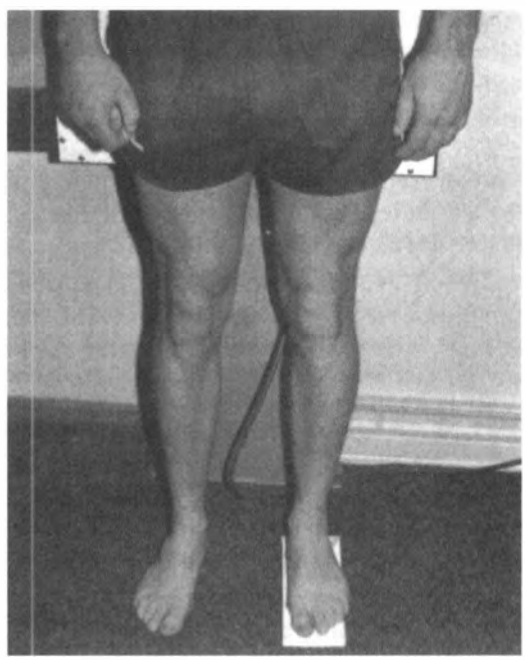

Figure 2.147. Photograph of patient being radiographed with short leg buildup in place.

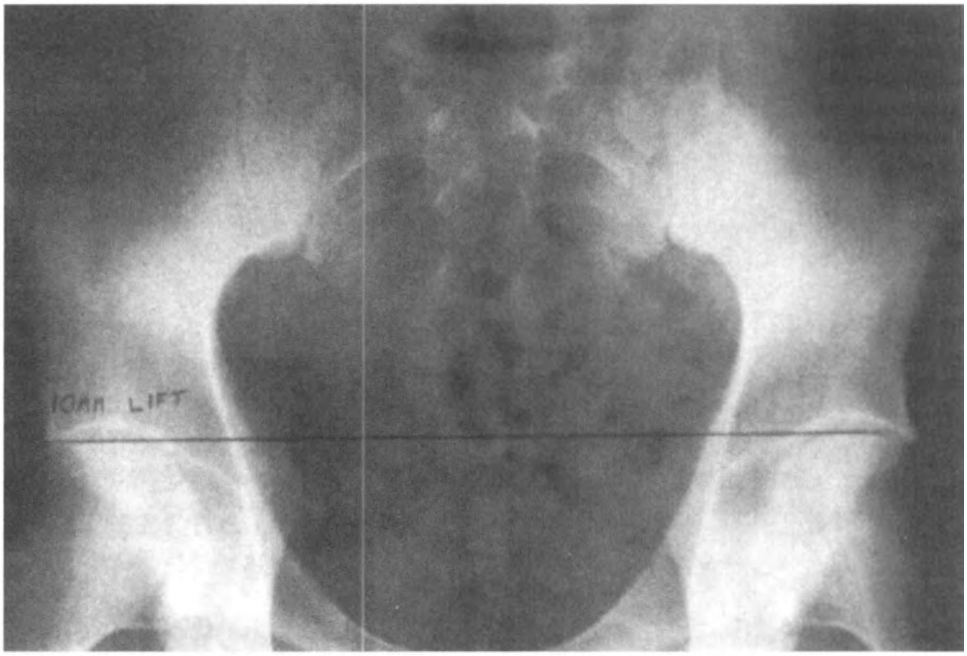

Figure 2.148. Same patient as in Figure 2.146, only requiring 10 mm to level the femoral heads. (Reprinted with permission from Aspergren DD, Cox JM, Frier KK. Short leg correction: a clinical trial of radiographic vs. non-radiographic procedures. J Manipulative Physiol Ther 1987;10(5):233–237. Copyright by the National College of Chiropractic, 1987.)

Cailliet (136) used visual correction with three points of reference to determine short leg and its correction: (*a*) iliac crest levelness; (*b*) vertical appraisal of the spine from the sacral base (the spine should be perpendicular to the sacral base); and (*c*) levelness of the posterosuperior iliac spine dimples. Lifts of varying thickness are placed under the foot of the short leg in leg length corrective procedures.

Cailliet's study (136) found that visual measurement did not differ significantly from x-ray measurement of leg length insufficiency. This allowed us to level the iliac crests on the short-leg side and low hemipelvis in comparison to the opposite side by placing lifts under the short extremity. We then took a radiograph through the femoral heads with the patient standing to confirm that the heads were now indeed level. In 75% of the cases of short leg, the buildup seen visually to level the pelvic iliac crests was the amount shown on radiograph to correct to the insufficiency. This allowed us to minimize radiation to the patient by having to take only one radiograph to confirm levelness of the femoral heads. In the other 25% of cases, we had to add to or subtract from the buildup until we found correction.

Procedure to Level the Iliac Crests for Short-Leg Correction

The patient is examined standing barefoot with both legs fully extended the same distance apart as the femoral heads. First, at arms length, the doctor places one index finger on each iliac crest and evaluates the horizontal level of the pelvis. It is desirable for the crests to be level. If they are not, a short leg may be present, and correction may be needed. Second, the "dimples" noted in most people in the region of the sacroiliac joints can be "lined up" by eye to furnish another estimate of pelvic level. It is also desirable to have the dimples horizontal. Except in obese or thin patients, we found the dimples were easily visualized. The third phase of visual evaluation involves observation of the lumbar spine in its vertical position related to the base of the sacrum. The spinous processes of the vertebrae are usually prominent, and they can be seen in the groove created by the erector spinae muscle groups. The desired position of the spine is at a right angle to the sacral base. If an oblique vertical position is noted, either the spine is curved or the sacral base is not level. A short leg can curve the spine or tilt the sacral base. These observations were used as indicators of discrepancy.

If these three clinical methods indicated any leg length discrepancy, correction was performed. Boards of predetermined and marked thickness were placed under the foot of the short leg until the pelvis became horizontal as gauged by the three methods described above. The board thickness required to achieve a level pelvis is equal to the shortness of the leg corrected. Board thickness was either 3, 6, 12, 18, or 25 mm (Fig. 2.145).

Following visual determination and correction of the pelvic discrepancy, radiographic short-leg study was performed with the patient barefoot in an upright position. Focal film distance was 40 inches, with the central ray centered anterior to posterior at the height of the femoral heads. In accordance with Chamber-

Table 2.7

Description of Visual and X-ray Measurements

Concept	Mean[a]	Range[a]	SD[b]	η
Visual	4.88	0–19	5.28	41
X-ray	4.73	0–19	5.03	41

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[a]Measurements are all reported in millimeters.

Table 2.8

Is There a Difference Between Visual and X-Ray Measurements?

Concept	Mean	SD	Z[a]
Visual	4.88	5.28	0.128
X-ray	4.73	5.03	

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[a]Note: The Z score is not significant at the $P = 0.05$ level.

Table 2.9

Is There a Difference Between Those Visual Scores That Are Less Than and Those That Are Greater Than X-ray Measurement?

Visual scores equal to x-ray = 15
Visual scores less than x-ray = 14
Visual scores greater than x-ray = 12

ANOVA Analysis

Dependent variable:	X-ray measurement			
Independent variables:	**Sum SQ**	**DF**	**F**	**SIG**
Visual	797.55	4	54.9	0
Groups	325.70	2	44.8	0
Between groups (explained)	893.80	8	30.8	0
Within groups (error)	116.25	32		
Total	1010.05	40		

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[b]To determine strength of relationship: Eta (η^2) = 0.885.

lain's view (371), the patient's feet must be directly under the femoral heads to prevent distortion (370). Giles and Taylor (366) note that the x-ray tube must be at the height of the femoral heads to avoid artificially inducing differences caused by the divergent ray (372). The radiologic technician was not informed of the visual correction previously recorded. When the film was viewed, a horizontal line was drawn perpendicular to the vertical side of the film across the top of the highest femoral head (Fig. 2.146). The same boards used to level the pelvis previously were used to build up the short leg and level the femoral heads (Fig. 2.147). The board was placed under the entire sole on the side of the low femoral head. A second radiograph was taken to confirm the proper height required for correction (Fig. 2.148).

Results

Overall results of this study show that, in 13 of 41 cases, visual determination was as accurate as radiographic appraisal. A second group of 13 cases were correct within 3 mm when compared to the radiographic standard. Six varied by 6 mm, and eight by 9 mm. One patient who was corrected was found to be off by 12 mm when compared on standing x-ray films.

In reviewing the results, it was generally found that the difference between visual and radiographic measurements for leg length insufficiency and musculoskeletal disorders is minimal. Table 2.7 demonstrates that the mean radiographic measurement is 4.73 mm. Ranges and standard deviations are also equal. Furthermore, in testing for a significant difference between visual and radiographic measures (Table 2.8), it is found that the z score is 0.128, which is not significant at the $P = 0.05$ level. Therefore, the null hypothesis stands: no difference is found between the two types of measurements.

Technically, radiographic measurement is considered to be most accurate. Table 2.9 shows that 15 of the visual measurements equal the x-ray measurement, whereas 14 are less than and 12 are greater than the x-ray measurement. A significant relationship is seen between visual and x-ray measures. The n^2 further shows that the relationship is very strong (0.885).

This study found that visual measurement did not differ significantly from radiographic measurement for leg length insufficiency. Furthermore, it was found that, when comparing those cases in which the visual measurement was less or greater than the x-ray measurement, a significant relationship was seen between visual and x-ray measurements. The n^2 demonstrates a very strong relationship between visual and x-ray methods of measurement.

The purpose of this chapter was to lay a foundation for understanding the diagnosis and treatment of low back and sciatic pain.

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Neurophysiology and Pathology of the Nerve Root and Dorsal Root Ganglion

James M. Cox, DC, DACBR

chapter 3

The credit belongs to the man who is actually in the arena, whose face is marred by dust and sweat and blood; who strives valiantly; who errs and comes short again and again, who knows the great enthusiasms, the great devotions, and spends himself in a worthy cause; who at the best, knows the triumph of high achievement; and who, at the worst, if he fails at least fails while daring greatly, so that his place shall never be with those cold and timid souls who know neither victory nor defeat.

—Theodore Roosevelt

DORSAL ROOT GANGLION ANATOMY AND PHYSIOLOGY

The dorsal root ganglion (DRG) is vulnerable to compression by degenerative structural changes of the disc, facet, pedicle, lamina, and ligamentum flavum, and it modulates pain from the motion segments of the spine by several intrinsic neuropeptides.

Anatomic and Radiographic Location of the Dorsal Root Ganglia (1)

The dorsal root ganglion and ventral root can be bifurcated or nonbifurcated. Figure 3.1 shows the connecting patterns of the DRG and ventral root, and Figure 3.2 shows the types of bifurcations seen, consisting of one DRG and one ventral root, one DRG and two ventral roots, or two DRGs and two ventral roots. L4 and L5 nerve roots bifurcate and S1 is nonbifurcated.

The positions of the dorsal root ganglion are classified into three types: intraspinal, intraforaminal, and extraforaminal (Figs. 3.3–3.5). At L4 and L5 nerve roots, they are mostly intraforaminal, whereas at S1 they are mostly intraspinal. Proximally placed ganglia have a high frequency of ganglionic indentation. The DRG is clinically important, and its location may correspond to clinical symptoms. Proximally positioned DRGs have been associated with radicular symptoms (1).

The main cause of ganglionic indentation is compression by the superior facet at the intervertebral foramen, which is found in 24 of 34 roots (70.6%) (Fig. 3.6) Other causes were bulging disc in three roots and bulging disc and facet in seven roots (1).

Analysis of Dorsal Root Ganglion Positions

Of 442 DRGs analyzed, 100% of L2, 48% of L3, 27% of L4, and 12% of L5 were located extraforaminally; 52% of L3, 72% of L4, and 75% of L5 were located intraforaminally; and 13% of L5 and 65% of S1 were located intraspinally (2). See Figures 3.7 to 3.9 for neuroanatomic location and appearance of the normal and pathologic DRG.

Magnetic Resonance Imaging (MRI) Study of DRG Size and Location

The size and location of the lumbar and S1 DRG are:

Level	Size	Location
L1	3.7 × 4.3 mm	92% in the lumbar intervertebral foramen
L2	4.6 × 5.7 mm	98% in the lumbar intervertebral foramen
L3	5.7 × 7.1 mm	100% in the lumbar intervertebral foramen

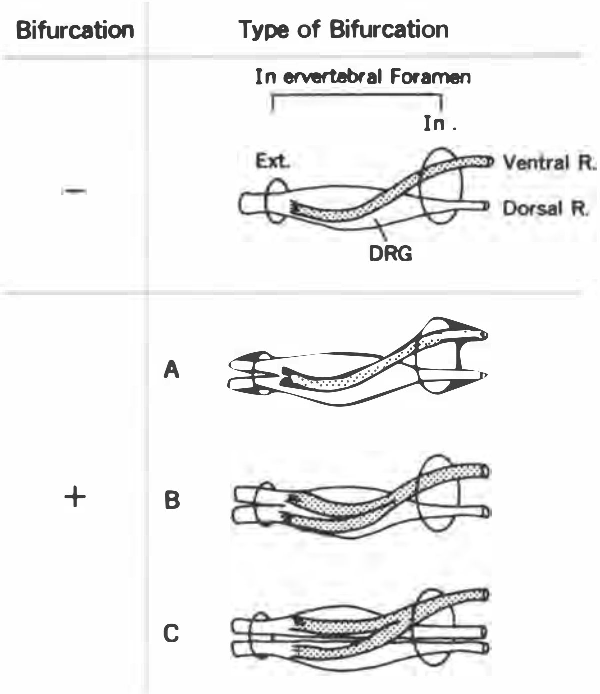

Figure 3.1. Classification by connecting patterns of dorsal root ganglion and ventral roots at the intervertebral foramen. *Ext*, extraspinal; *Int*, intraspinal; *R*, root. (Reprinted with permission from Kikuchi S, Katsuhiko K, Konno S, et al. Anatomic and radiographic study of the dorsal root ganglion. Spine 1994;19(1):6–11. Copyright 1994, Lippincott-Raven.)

L4	6.2 × 8.4 mm	100% in the lumbar intervertebral foramen
L5	5.9 × 9.4 mm	95% in the lumbar intervertebral foramen
S1	6.2 × 11.2 mm	79% in the intraspinal region

The nerve roots occupy 23 to 30% of the area of the intervertebral foramen. The relatively larger DRG in the lower lumbar region may be more susceptible to compression than the upper DRG, particularly with the higher propensity to disc degeneration and intervertebral narrowing in the lower lumbar region. The S1 nerve root and DRG may both be involved as a result of disc herniation or degenerative changes of the L5-S1 facet because it is the most intraspinally located DRG of all lumbar nerve root complexes (3).

DRG CHANGES PRODUCE RADICULOPATHY AND THERMAL HYPERALGESIA

Lindblom and Rexed first alluded to the dorsal root ganglion as a source of lumbar pain in 1948 in a cadaveric study. They reported that the DRGs were compressed and deformed by dorsolateral protrusions of lumbar discs or enlarged facet joints, and underwent microscopically gross alterations of the internal structure. These authors concluded that mechanical compression of the DRG was a causative factor in radicular pain. Some clinical investigations have reported that decompression of the DRG was necessary in some patients with sciatica or leg paresthesia. Thus, the DRG likely plays an important role in the underlying pathomechanisms of spinal pain. DRG mechanical irritation caused endoneurial edema with the production of pain-producing neuropeptides (substance P, calcitonin gene-related peptide, and vasoactive intestinal peptide [VIP]), which produced thermal hyperalgesia (4).

DRG Circulation and Protein Synthesis

The dorsal root ganglion has a very rich microvascular network. The dorsal root ganglia, where the sensory nerve cell bodies are located, comprise the site of synthesis of several essential substances (e.g., proteins, which are transported down the axons through the axonal transport and are needed to maintain the structural and functional integrity of the entire sensory neuron) (5).

Arterial occlusion occurs at a pressure close to the mean arterial blood pressure. For instance, 10 mm Hg was sufficient to induce a 20 to 30% reduction of methyl-glucose transport to the nerve roots as compared with controls (5).

Trophic Function of Nerves

In recent years, scientists have ceased to be self-conscious and apologetic about the use of the word *trophic* in connection with nerves. Neural phenomena have always been explained in terms of impulses, electrical potentials, and frequencies; and it was unsettling to the scientific world to discuss factors other than impulses and reflexes as influences on target tissues supplied by nerve fibers.

In Korr's (6) inquiry about the simple muscle atrophy following a severed nerve supply, he states that as long as protoplasmic continuity is maintained in the axon from perikaryon to motor end plate, even if it is nonconducting, the neuronal trophic influence continues to be exerted. The longer the nerves are attached to the muscle, the longer the time before postdenervation changes appear. This would indicate that the amount of nerve substance still available to the muscle is what is important, and that when it has been exhausted, trophic support ends. Thus, the crucial factor is not that the nerve has been severed, stopping its impulses, but rather, the length of time the trophic support is available to the muscle.

Among the components axonally transported to muscle by nerves are proteins, phospholipids, enzymes, glycoproteins, neurotransmitters and their precursors, mitochondria, and other organelles. Although rates of approximately 1 mm/day have been found to be common to many mammalian nerves, it is now known that there are several rates of transport, up to several hundred millimeters per day, and different cargoes are being carried at different rates.

Axoplasmic Transport

The demonstration of multiple delivery waves raised a new set of questions. Are different proteins axonally transported and delivered in each of the periods? Can specific proteins be traced

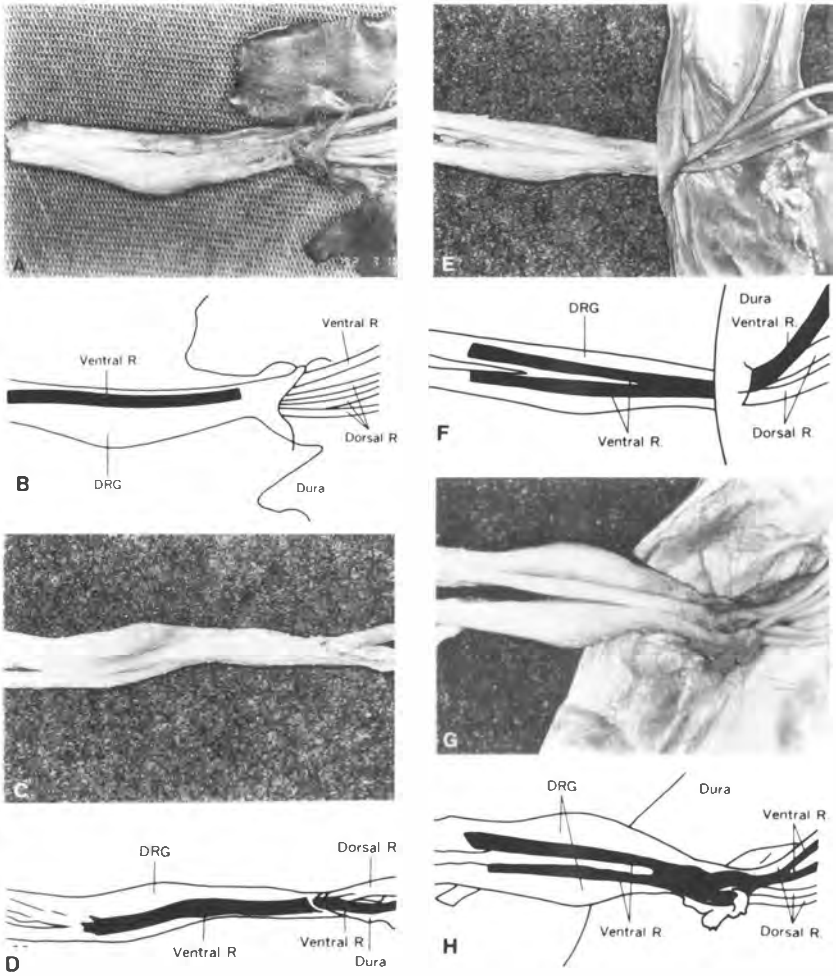

Figure 3.2. Connecting patterns of dorsal root ganglion and ventral roots. **A** and **B.** Non-bifurcated type. **C** and **D.** Bifurcated type A. **E** and **F.** Bifurcated type B. **G** and **H.** Bifurcated type C. (Reprinted with permission from Kikuchi S, Katsuhiko K, Konno S, et al. Anatomic and radiographic study of the dorsal root ganglion. Spine 1994;19(1):6–11. Copyright 1994, Lippincott-Raven.)

from the medulla, through the nerve, to the tongue muscle? Do all of the protein fractions carried in the axon reach the muscle or is there some selection process? Are the proteins delivered to the muscle different from those synthesized by the muscle itself (6)? Research shows:

1. Rabbits that had been prepared as in previous investigations were sacrificed for tissue specimens at peak times in each "wave" (delivery of proteins via the axon), namely days 1, 12, 22, and 34, to maximize the yields of radioactive protein.
2. The proteins extracted from the tissues were first divided by centrifugation into soluble and insoluble portions (the insoluble being those associated with particular cellular elements), each of which was assayed for radioactivity.

Observations were as follows:

1. Of the 12 conspicuous "spikes" of soluble radioactive proteins evident in the medulla (hypoglossal nerve cells) on day 1, only two to three were evident in the nerve, the rest not appearing until day 12.
2. The proteins reaching the muscle in the first wave, on day 1, were almost exclusively insoluble. Those studying axonal transport had also generally agreed that insoluble or structural proteins are carried in the rapid transport system.
3. With certain exceptions, electrophoretic fractions were traceable from hypoglossal neurons through nerve to muscle.
4. Each wave carried a different mixture of proteins, as observed in nerve and muscle, although there were fractions common to consecutive waves because of the overlap previously mentioned.
5. Proteins synthesized by the muscle were different from those delivered by the nerve.

From these observations and from earlier studies, the following conclusions are made:

1. Some proteins synthesized in the hypoglossal nerve cells are held for up to 12 days before being dispatched into axons.

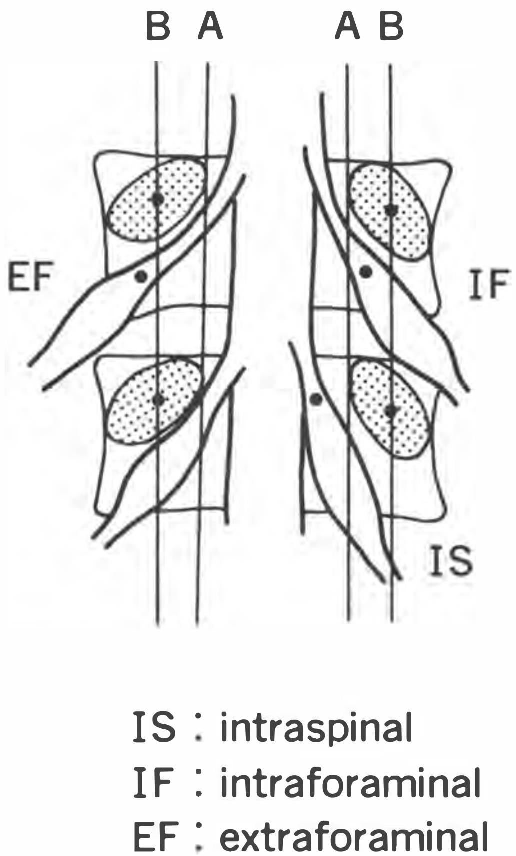

Figure 3.3. Classification by positions of dorsal root ganglion. (Reprinted with permission from Kikuchi S, Katsuhiko K, Konno S, et al. Anatomic and radiographic study of the dorsal root ganglion. Spine 1994;19(1):6–11. Copyright 1994, Lippincott-Raven.)

2. Each of the four waves carries a different complement of protein synthesized in the perikaryon, with some admixture due to overlap of the waves.
3. Although continuity of transport exists from one part of the nerve to the next, there is discontinuity of transfer from nerve to muscle.
4. Transfer of proteins from nerve to muscle is a different, apparently slower, process than transport along the nerve.
5. The neuron supplies proteins that are not manufactured by the muscle.

With increased support and elaboration, a hypothesis is presented: trophic influences of nerves on target organs depend, at least in substantial part, on the delivery of specific neuronal proteins by axonal transport and junctional transfer.

Hence, in considering the neurologic impact on human health of postural and biomechanical defects in the body framework that are amenable to manipulative therapy, we can no longer limit ourselves to disturbances in impulse traffic. Conspicuous and distressing as are the resultant pain and the motor, sensory, and autonomic dysfunctions, the more subtle and insidious trophic consequences of disturbances in axoplasmic composition and transport are no less important (6).

Rydevik et al. (7) discusses the biomechanical aspects of nerve root deformation induced by compression. The functional changes induced by compression can be caused by mechanical nerve fiber deformation, but they can also be a consequence of nerve root microcirculation, leading to ischemia and intraneural edema. Intraneural edema and demyelinization seem to be critical factors for the production of pain in association with nerve root compression.

Inside the dural root sheath, the dorsal and ventral nerve

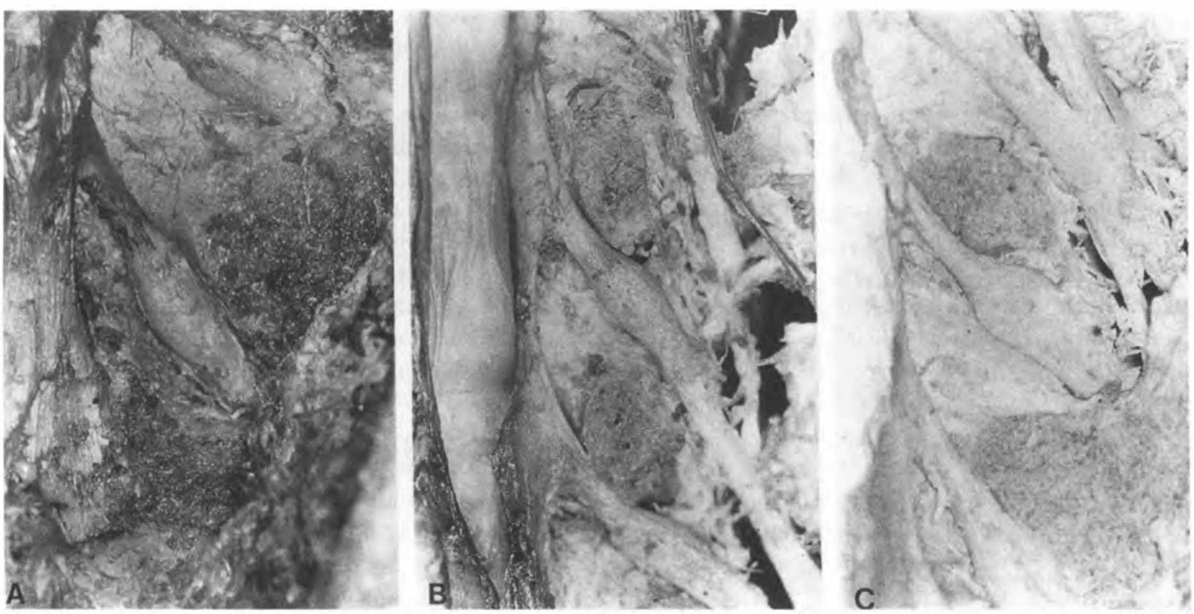

Figure 3.4. Types of dorsal root ganglion. **A.** Intraspinal type (IS type). **B.** Intraforaminal type (IF type). **C.** Extraforaminal type (EF type). (Reprinted with permission from Kikuchi S, Katsuhiko K, Konno S, et al. Anatomic and radiographic study of the dorsal root ganglion. Spine 1994;19(1):6–11. Copyright 1994, Lippincott-Raven.)

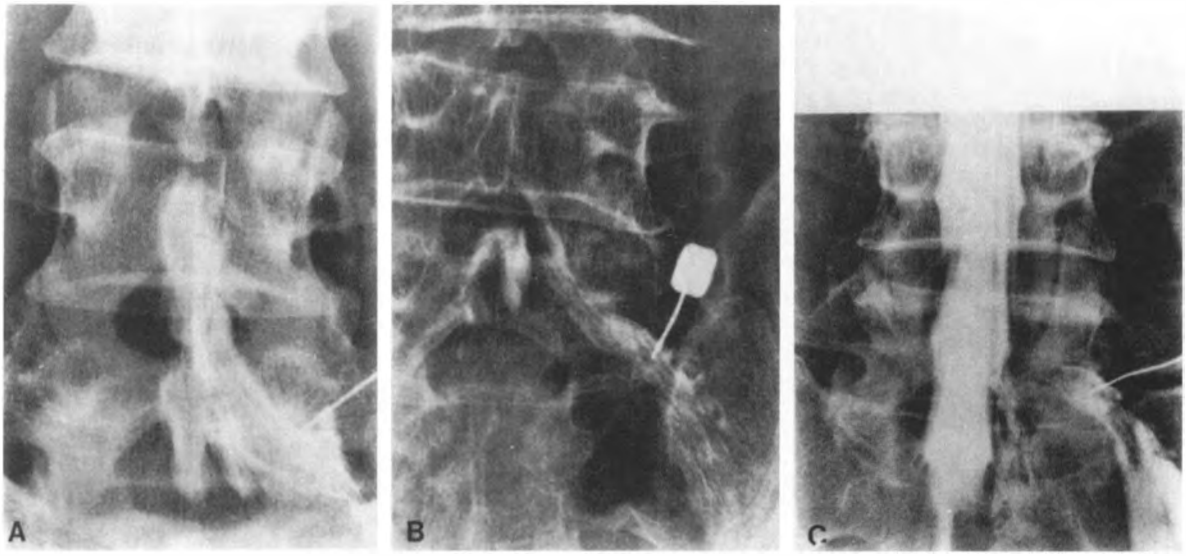

Figure 3.5. Radiographs of L5 nerve roots showing three types of dorsal root ganglion. **A.** Intraspinal type. **B.** Intraforaminal type. **C.** Extraforaminal type. (Reprinted with permission from Kikuchi S, Katsuhiko K, Konno S, et al. Anatomic and radiographic study of the dorsal root ganglion. Spine

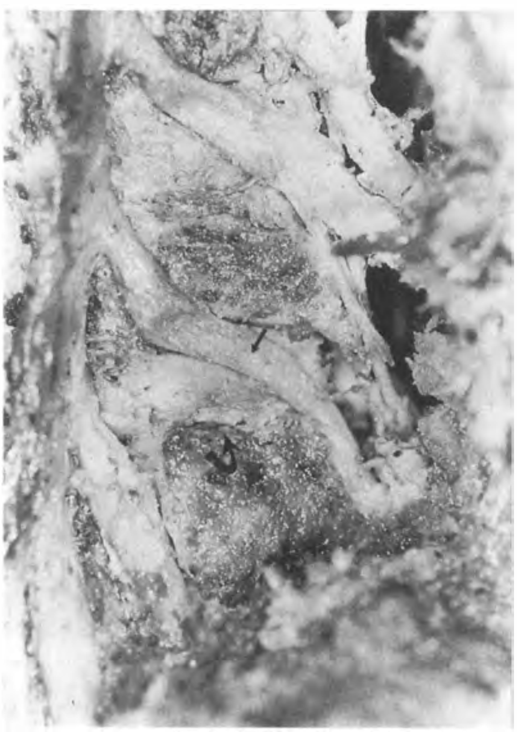

Figure 3.6. Ganglionic indentation of the L5 nerve root (*straight arrow*) by the superior facet of sacrum (*curved arrow*). (Reprinted with permission from Kikuchi S, Katsuhiko K, Konno S, et al. Anatomic and radiographic study of the dorsal root ganglion. Spine 1994;19(1):6–11. Copyright 1994, Lippincott-Raven.)

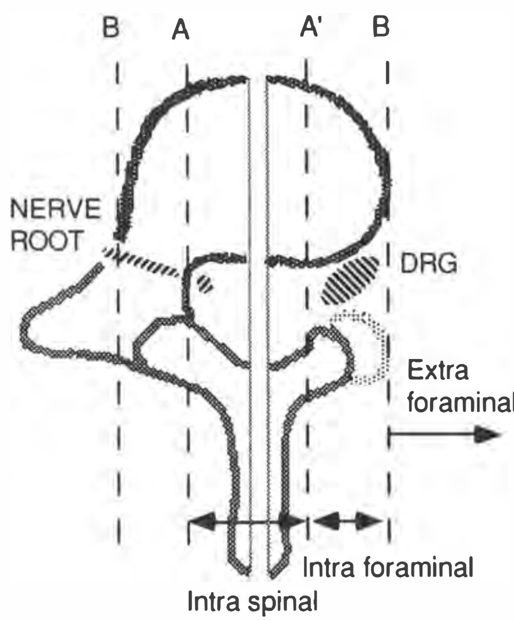

Figure 3.7. Foraminal space on the axillar views of magnetic resonance imaging. *Line A:* inner edge of the lower facet or pedicle. *Line B:* lateral edge of the vertebral body or superior facet. (Reprinted with permission from Haminishi C, Tanaka S. Dorsal root ganglia in the lumbosacral region observed from the axial views of MRI. Spine 1993; 18(13):1753–1756. Copyright 1993, Lippincott-Raven.)

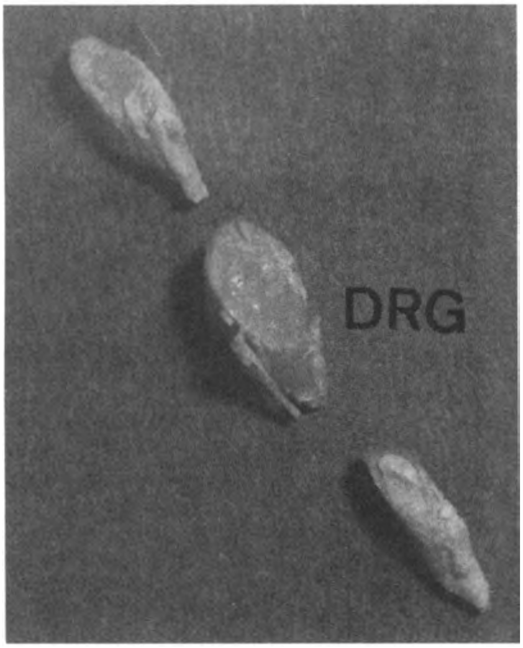

Figure 3.8. The axial view of the cadaveric L5 nerve root and dorsal root ganglion (DRG), which was cut obliquely by 45° with slice thickness of 5 mm. The diameter of the proximal portion of the root, the widest portion of DRG, and distal root are 3.2, 5.4, and 2.7 mm, respectively. (Reprinted with permission from Haminishi C, Tanaka S. Dorsal root ganglia in the lumbosacral region observed from the axial views of MRI. Spine 1993;18(13):1753–1756. Copyright 1993, Lippincott-Raven.)

roots approach the intervertebral foramen. The dorsal root continues into the dorsal root ganglion, which usually is located within the central portion of the intervertebral foramen. More distally, the roots join to form the spinal nerve, which continues into the peripheral nerve (Fig. 3.10).

Nerve Root Compared with Peripheral Nerve

The nerve roots lack a perineurium, whereas peripheral nerves have a well-developed epineurium wherever they are subjected to mechanical forces such as compression and tension. Nerve roots, having no such well-developed epineural connective tissue, are more susceptible to mechanical deformation than are peripheral nerves. To some extent, nerve roots are protected by the cerebrospinal fluid, which acts with the dura and arachnoid membrane to mechanically protect them (8).

Nerve Root Blood Supply

An adequate supply of oxygen to nerve fibers via intraneural microcirculation is necessary for nerve function. The dorsal root ganglion receives its blood supply from spinal branches from each segmental artery (9). Figure 3.11 shows the nerve roots within the cauda equina, the motor and sensory components of the spinal nerve, and the DRG lying within the intervertebral foramen. Interference with the blood supply of the nerve root or the DRG can lead to disturbed nerve root function. Nerve root compression may interfere with the blood supply to the nerve root (10, 11). (We will discuss the vulnerability of the dorsal root ganglion to compressive reaction later in this chapter.)

Rydevik et al. (12) studied the intraneural microcirculation under graded compression of a rabbit tibial nerve. It was found that the first sign of intraneural blood flow impairment was epineural vessel stasis, appearing at pressures as low as 20 to 30 mm Hg. Such compression at higher pressures or for prolonged periods of time can damage the endoneural blood vessels, resulting in an increased permeability or a breakdown of the

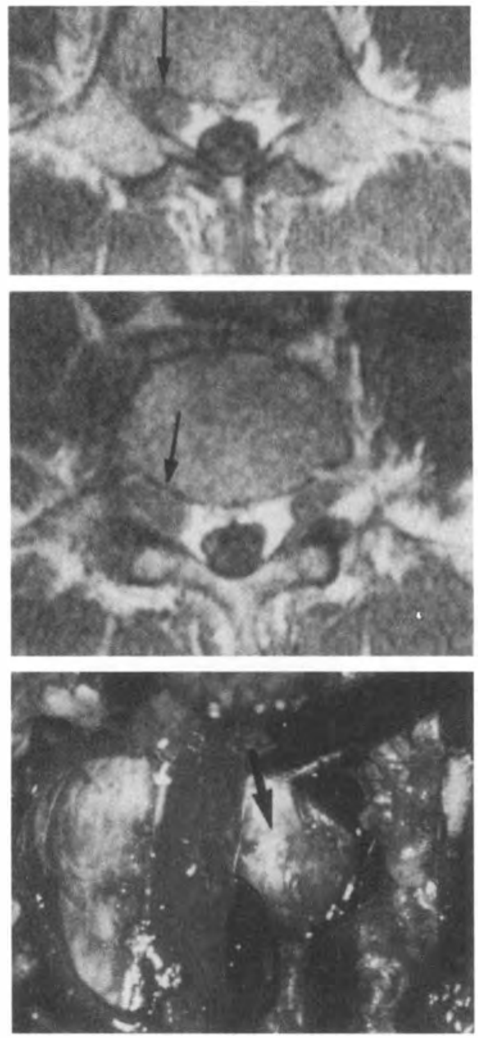

Figure 3.9. Asymmetric dorsal root ganglion (DRG). A 42-year-old man with intermittent claudication due to the radicular pain on the right leg. **Top:** the right L5 DRG (*arrow*) locates intraspinally. **Middle:** the next slice shows that the right DRG is far larger than the intraforaminally located left DRG, and it extends into the foraminal space (*arrow*). **Bottom:** the intraspinal portion of the right DRG was swollen markedly (*arrow*), and it was pushed down and kinked by the pedicle, which had been excised. (Reprinted with permission from Haminishi C, Tanaka S. Dorsal root ganglia in the lumbosacral region observed from the axial views of MRI. Spine 1993;18(13):1753–1756. Copyright 1993, Lippincott-Raven.)

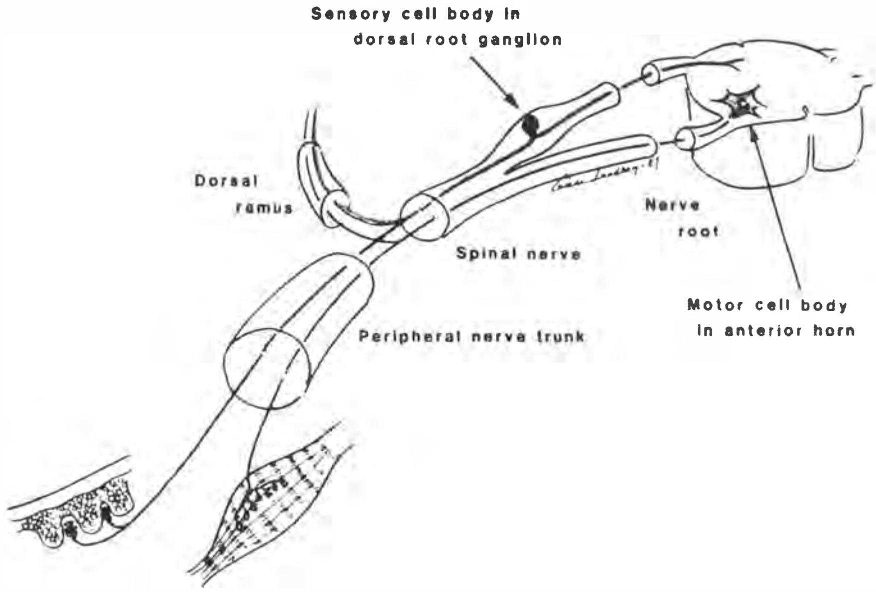

Figure 3.10. Schematic drawing of the arrangement of nerve roots, spinal nerve, and peripheral nerve, including the target organs of the neurons. The axons are long cellular extensions from the nerve cell bodies, located in the anterior horn of the spinal cord or in the dorsal root ganglia. (Reprinted with permission from Rydevik B, Brown MD, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984;9(1):8.)

blood-nerve barrier and, consequently, formation of an endoneurial edema (13). This has been compared to a "closed compartment syndrome" in which the microcirculation of the nerve fascicles is jeopardized and a posttraumatic ischemia of the injured nerve established. A corresponding mechanism may operate in the case of nerve root compression at the level of the intervertebral foramen, or in that of a tight sheath surrounding the nerve roots and spinal nerve where these nerve components are enclosed in a rigid bony canal (Fig. 3.11). The dorsal root ganglion contains more permeable microcirculation than peripheral nerves, and may be easily subjected to endoneurial edema by compression. The ganglion has a tight capsule, and therefore any such edema could increase the pressure on it easily (15).

Basic Anatomy and Pathophysiology of Lumbar Nerve Injury

The motor (i.e., ventral) nerve root and sensory (i.e., dorsal) nerve root pass dorsal and lateral to the intervertebral disc.

The dorsal nerve roots have a larger diameter than the ventral nerve root and therefore a greater susceptibility of the sensory axons to compressive forces. The S1 nerve roots are approximately 170 mm long, whereas the L1 nerve roots are 60 mm long. The nerve roots as well as the spinal nerves are composed of axons that have arisen within the substance of the spinal cord and course to their final destination in the periphery. These axons may exceed 100 cm in length (16).

Spinal nerve roots lack the connective tissue protection that sheaths peripheral nerves. This sheathing has considerable mechanical strength and possesses properties to form a barrier to diffusion of certain molecules. The spinal nerve roots, therefore, are at a disadvantage mechanically and, possibly, biochemically. The nerve roots, however, are surrounded by cerebrospinal fluid, which, acting with the dura, gives the spinal nerve roots an element of mechanical protection. The dura of a spinal nerve root appears to be continuous with the epineurium of the peripheral nerve. It must be kept in mind that the nerve root complex must be extraordinarily mobile.

Nerve roots must change length depending on the degrees of flexion, extension, lateral bending, and rotation of the lumber spine. Lumbar nerve roots limited in motion by either intraspinal or extraspinal fibrosis will create traction on the nerve root complex, causing ischemia and secondary neural dysfunction. This fact must also be kept in mind during the rehabilitation process. Flexibility exercises must be designed to maintain nerve root mobility.

Intraneural blood flow is markedly affected when the nerve is stretched about 8% beyond its original length. Complete cessation of all intraneural blood flow is seen at 15% elongation.

The dorsal root ganglion, because of its fibrous capsule and its rich vascular supply, may be more susceptible to changes in intraneural blood flow and to the development of secondary intraneural edema with consequent fibrotic change. This may explain sensory loss on gross neurologic examination (16).

Sunderland (11) has also stated that spinal nerves will tolerate remarkable degrees of deformation provided the deformation occurs slowly and does not alter the blood supply. Normal spinal nerves appear to have a high tolerance to mechanical deformation. Damaged nerve fibers are more susceptible to

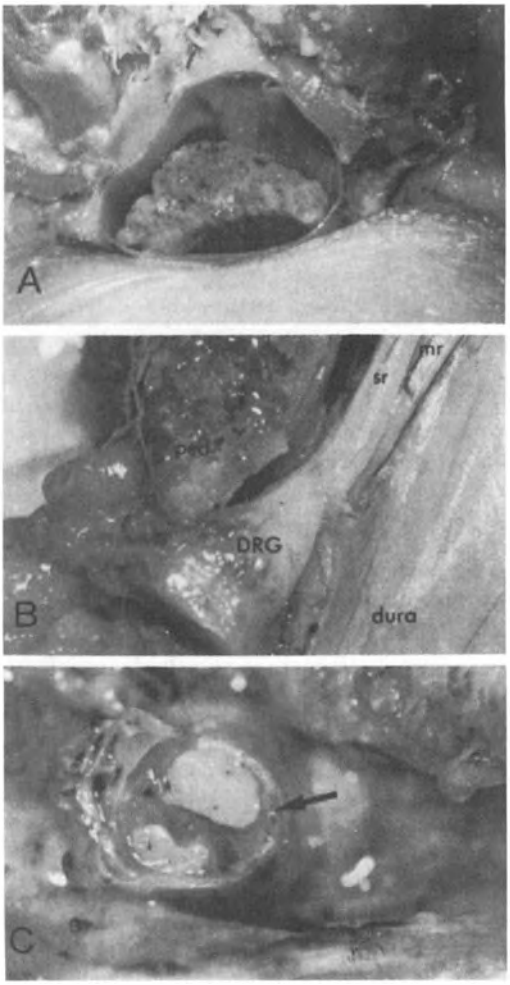

Figure 3.11. **A.** Cross-section, demonstrating the cauda equina in the spinal canal. The intervertebral disc is seen at bottom. **B.** Dorsal view of the nerve roots inside the dural sheath, following removal of the lamina and opening of the dura. The nerve root complex, composed of motor root (*mr*), sensory root (*sr*), and dorsal root ganglion (*DRG*), is located beneath the pedicle (*ped*), which has been divided. **C.** Cross-section through the root sheath (*arrow*) just central to the ganglion. The two roots are located within the tight sheath, which in turn is running in a rigid bony canal. (Reprinted with permission from Rydevik B, Brown MD, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984;9(1):8.)

deformation and ischemia. Therefore, a patient with long-standing radiculopathy will tolerate less instability and mechanical stress than will the patient with a healthier nerve root.

Normal nerve root compression usually induces a sensation of numbness but not one of pain. However, mechanical deformation of a previously compressed nerve does cause pain. The dorsal root ganglion appears to be the most sensitive to mechanical deformation (11).

DRG Mediates Pain

The dorsal root ganglion seems to play a crucial role as a mediator of pain in the lumbar spine. Experimental whole body vibration, a risk factor for low back pain, has been shown to induce significant changes in the synthesis of various neuropeptides, such as substance P (SP) and vasoactive intestinal peptide in the ganglion. The DRG also seems to be mechanosensitive, and compression may induce both a pressure increase in the ganglion and radiating nerve root pain (5).

DRG As an Origin of Pain-Producing Impulses

Nerve impulses were recorded in dorsal roots or in the sciatic nerve of anesthetized rats. It was shown by sectioning, stimulation, and collision that some ongoing nerve impulses were originating from the dorsal root ganglia and not from the central peripheral ends of the axons. In a sample of 2731 intact or acutely sectioned myelinated sensory fibers, 4.75% + 3.7% contained impulses generated within the dorsal root ganglia. Slight mechanical pressure on the DRG increased the frequency of impulses (17). Unmyelinated fibers were also found to contain impulses originating in the dorsal root ganglion.

Fine filament dissection of dorsal roots and of peripheral nerves, as well as collision experiments, showed that impulses originating in the DRG were propagated both orthodromically into the root and antidromically into the peripheral nerve. It was also shown that the same axon could contain two different alternating sites of origin of nerve impulses: one in the sensory ending and one in the ganglion. These observations suggest that the DRG, with its ongoing activity and mechanical sensitivity, could be a source of pain-producing impulses. Furthermore, it could particularly contribute to pain in those conditions of peripheral nerve damage where pain persists after peripheral anesthesia or where vertebral manipulation is painful.

DRG: An Active Pain Generator

Sensory nerve fascicles central to amputation neuromas in two patients were found to produce considerable ongoing activity that was not silenced by local anesthesia of the neuroma. Reasons are found to suspect that the dorsal root ganglia might have contributed to this ectopic barrage, and Wall and Devor (17) state that De Santis and Duckworth identified dorsal root ganglia as a source of discharge in rat muscle nerves damaged by freeze lesions. Further, they state that Kirk had previously shown in cat and rabbit that transection of the spinal nerve immediately peripheral to the DRG produces firing in dorsal root filaments. In contrast to axons (excluding sensory endings), which are highly resistant to impulse generation following mechanical impact and even after having been cut across normally produce only a brief injury discharge, DRG cells produce a prolonged discharge with relatively gentle mechanical compression (17).

It is generally presumed that afferent signals received by the spinal cord in normal animals arise exclusively in sensory nerve endings. The results described here show that, at least under experimental conditions, the dorsal root ganglion constitutes a second source of afferent impulses. Specifically, DRG contribute a tonic, low-level (about four impulses per second in 5% of sciatic nerve afferent fibers), spontaneous background discharge.

From these results it is probable that afferent impulses originate from dorsal root ganglion cells. In fact, the possibility that DRG cells might be a significant source of afferent barrage under certain circumstances has already been shown by Howe et al. (18) with their demonstration of the relatively low mechanical threshold of normal dorsal root ganglia. They did not, however, discuss the possibility of spontaneous discharge. The finding that chronic peripheral nerve section exaggerates the tendency of axotomized DRG cells to fire spontaneously also confirms the conclusions of Kirk, De Santis, and Duckworth as cited by Wall and Devor (17), based on results from quite different preparations.

The high degree of excitability of normal dorsal root ganglion cells and its enhancement by chronic nerve injury may have important clinical significance. The Lasègue sign—pain in the leg on straight leg raising—could be the consequence of shifting tension on the dorsal root ganglia, which are mechanically stressed by the maneuver. It is conceivable that the afferent barrage is being affected by manipulation of the dorsal root ganglia. The increase in ganglion discharge in cases of chronic nerve injury could partly account for prolonged intractable pain and paresthesia that may follow nerve damage, including phantom limb sensation and pain.

The mechanism and exact site of ectopic spontaneous impulse generation in dorsal root ganglia is not known for certain. Circumstantial evidence, however, places it in the axon hillock region.

The radicular pain of sciatica has been ascribed to be compression of the spinal root by a herniated intervertebral disc (18). It was assumed that root compression produced prolonged firing in the injured sensory fibers and led to pain perceived in the peripheral distribution of those fibers. This concept has been challenged on the basis that acute peripheral nerve compression neuropathies are usually painless. Furthermore, animal experiments have rarely shown more than several seconds of repetitive firing in acutely compressed nerves or nerve roots. It has been suggested that "radicular pain" is actually pain referred to the extremity through activation of deep spinal and paraspinal nociceptors (18).

Chronically Irritated Nerve Roots Are Most Sensitive

Experiments on cat lumbar dorsal roots and rabbit sural nerves confirm that acute compression of the root or nerve does not produce more than several seconds of repetitive firing. However, long periods of repetitive firing (5 to 25 minutes) follow minimal acute compression of the normal dorsal root ganglion. Chronic injury of dorsal roots or sural nerve produces a marked increase in mechanical sensitivity; several minutes of repetitive firing may follow acute compression of such chronically injured sites. Such prolonged responses could be evoked repeatedly in a population of both rapidly and slowly conducting fibers. Because mechanical compression of either the DRG or of chronically injured roots can induce prolonged repetitive firing in sensory axons, it is concluded that radicular pain is caused by activity in the fibers appropriate to the area of perceived pain. Although repeated or maintained compression of the roots did not produce prolonged activity, a minor chronic injury altered the response to a subsequent acute compression (18).

Repetitive Discharge

Data show that minor compression of the dorsal root ganglion invariably produces repetitive firing lasting several minutes. Occasionally a discharge lasting as long as 25 minutes can be detected in small multifiber filaments dissected from dorsal rootlets. Similar forces can produce several seconds or, rarely, a few minutes of repetitive firing when chronically damaged dorsal roots are compressed at the site of prior trauma, but not at sites along the root or nerve other than the chronically injured region. The forces sufficient to excite these responses are similar in both situations. They are of small magnitude and can be slowly applied. This abnormal response can be triggered repetitively without changing stimulus parameters. Such forces are insufficient to excite normal dorsal roots unless applied rapidly. In the normal dorsal root, it is more difficult to repeatedly elicit the same response from the same region. Furthermore, an adequate initial force, when repeated, usually results in irreversible damage to the axons.

Injured nerves (end-bulb neuromas and incontinuity regenerating nerves) have a markedly increased sensitivity to mechanical stimulation. Minor movements can result in 15 to 30 seconds of repetitive firing. Evidence for slightly longer periods (2 to 3 minutes) of activation in response to acute compression of chronically injured roots has been seen. The usual response to compression of the chronically injured region is 15 to 30 seconds of repetitive firing. This mechanical sensitivity may represent the physiologic equivalent of Tinel's sign (18).

It seems likely that compression of the dorsal root ganglion is important in the generation of the radicular pain of an acute herniated intervertebral disc. Typically, the patient with this syndrome describes the sudden onset of pain in the back and leg that radiates into the foot. The dermatome in which the pain is perceived usually predicts the compressed spinal root. Neurologic deficit, if present, usually occurs in the same dermatome. The pain persists much longer than the momentary response seen in the acute compression of a normal nerve root, and it is more consistent with the slowly adapting response seen following DRG compression. This radicular pain can often be relieved by immobilization or complete bed rest; minor movements or coughing reactivate the pain.

Anatomic studies have shown that the lumbar dorsal root ganglion can be trapped easily between a herniated disc and the facet. Small and repeated movements of the joint could intermittently traumatize the DRG. The DRG in the lumbar region lies directly over the lateral portion of the disc. In an autopsy study, in all cases of herniated lumbar discs, the dorsal root ganglion was compressed and distorted, and it manifested various degrees of degeneration (18).

Reproduction of radicular pain was described in patients who had undergone laminectomies for the removal of a herniated disc. In these patients at the time of surgery, a nylon suture was looped around the root and the ends were brought out through the skin. Postoperatively, the preoperative radicular

pain was precisely reproduced by gentle traction on the suture. It was noted that the injured or involved root was much more sensitive to this manipulation than an adjacent normal or uninvolved root. Other similar findings were reported (18).

The ease of activation and prolonged response of A a and C fibers in response to dorsal root ganglion compression implies that the radicular pain associated with a herniated intervertebral disc, and perhaps with other intraspinal masses, is due initially to compression of the dorsal root ganglion. Subsequent development of mechanical sensitivity in the chronically injured nerve roots may also contribute to the production of continuing radicular pain. Studies demonstrate that radicular pain can be due to activity in the fibers appropriate to the region of pain and need not be a referred phenomenon (18).

DRG Produces Repetitive Firing of Impulses

The middle of an axon is not usually a site of impulse generation; it is a region of impulse replication, where impulses originating elsewhere are faithfully reproduced in one-for-one fashion. Nonetheless, it is not so specialized that it cannot generate impulses on compression. For example, because of rudimentary mechanosensitivity of axons, compression of the ulnar nerve at the elbow produces paresthesia. This ectopic generation of nerve impulses appears to operate in the manner of most mechanoreceptors: a generator potential is developed, and the repetitive firing patterns that result are those expected from the pacemaker-like rhythmic firing mode in which depolarization is converted into firing rate. Although repetitive firing in normal peripheral nerves and dorsal roots is usually transient even with sustained compression, DRG cells and chronically injured axons are capable of producing sustained repetitive firing on sustained compression, as has been described elsewhere (19).

The dorsal root is extremely sensitive to pressure. In peripheral nerves, the Aa fibers that mediate impulses from the muscle spindle and the Golgi apparatus, as well as efferent motor impulses, are the most pressure-sensitive structures. According to this hypothesis, slight pressure on these fibers in the dorsal roots and ganglia results initially in reduced inflow of afferent impulses from muscle and tendon receptors. This is in agreement with the anatomic arrangement in the intervertebral foramina. As a result, the central nervous system responds with an increase in outflow of efferent impulses. This in turn gives rise to sustained increase in muscle tone, leading to myalgia and tendinitis (20).

Sciatica, Claudication, and Groin Pain Due to DRG Irritation

A series of patients with leg pain, whose dorsal root ganglia were located more proximal than usual and lay within the nerve root canal, was reported (21). These proximal ganglia became entrapped in a space that, although slightly narrowed, would have accommodated a normal nerve root without causing pain.

Of the 11 patients, two were men and nine were women whose ages ranged from 28 to 60; more than 50% were in their 40s or 50s. Most of these patients presented with an exceptionally long history of symptoms prior to diagnosis (average 7 years).

Pain was aggravated by the standing position in seven patients. Seven complained of intermittent claudication. The pain radiated down the leg in a sciatic distribution in all patients and extended to the ankle or foot in eight. Radiation to the groin was reported in three patients, two with compression of the L5 ganglion and one with compression of both the L5 and S1 ganglia.

Physical examination was entirely normal in three patients. Straight leg raising was limited to 70° in the affected lower limb in four patients, significantly reduced to 60° in one, and reduced to 30° in another. Muscle weakness or wasting was found in four patients, and sensory abnormalities were present in three. The ankle reflex was depressed in three patients and absent in one.

All 11 patients underwent surgical decompression. Subarticular entrapment of the L5 dorsal root ganglion was found in six patients, both L5 and S1 DRG entrapment in three, and S1 DRG entrapment alone in two (21).

Substance "P" Produced In DRG

The neuropeptide, "substance P," is known to be synthesized in cell bodies of the dorsal root ganglia. This neuropeptide is also known to modulate sensory, nociceptive transmission postsynaptically in the dorsal root ganglia, nerve roots, and substantia gelatinosa of the spinal dorsal horn that the cell bodies innervate. These results were determined by using both immunohistochemistry and radioimmunoassay. This study suggests that SP may modulate nociception when lumbar nerve roots are stimulated mechanically (22).

The dorsal root ganglion normally lies within the lateral portion of the intervertebral foramen, and it is not directly compressed by a bulging disc prolapse or a bony spur that may compromise the nerve root (23). This ganglion contains cell bodies of first-order sensory neurons. The chemical response to mechanical deformation of the DRG may be significant for some of the unknown causes of low back pain.

Stimulation and release of the neuropeptide substance P, or similar agents, by pathophysiologic mechanisms has been postulated to explain the pain of spinal nerve root pathology. Proximal flow of SP from the dorsal root ganglion to the spinal cord certainly occurs (23). Substance P is one of the neurotransmitters produced in the cell bodies of the DRG. This neuropeptide probably acts as a neuromodulator of pain signals at synapses in the region of the substantia gelatinosa where pain perception is first integrated in the spinal cord. The appearance of SP in this area may be the first chemical signal of exteroceptive pain in the spinal cord.

The abundance of substance P immunoreactive nerve terminals in the substantia gelatinosa of the dorsal horn of the spinal cord suggests that SP is contained in primary afferent fibers that penetrate this region—as well as the dorsolateral funiculus radially—and terminate in the dorsal horn.

The increased amounts of substance P produced after mechanical stimulation provide a possible neurophysiologic explanation for the nociceptive effects produced by mechanical

compromise of the spinal nerve roots. Dorsal root ganglion irritation associated with various syndromes of mechanical compromise seem able to produce increased amounts of substance P, which is known to have central effects modulating nociceptive afferent transmission. A delay between mechanical stimulation and the appearance of SP centrally, where it may modulate neurotransmission, may also be important. It is interesting to speculate whether this may be an explanation for the well-known clinical observation that after a disc prolapse a period of several days may pass before sciatic pain is experienced, although back pain may be immediately apparent following the disc protrusion (22).

Sympathetic Trunk Compressed by Osteophytosis of Thoracic Spine

Osteophytes were found to compress the sympathetic structures in the thorax in 655 (65.5%) of 1000 cadavers. In 60.4% of the affected cases, the compression was on the right side, and in 36.9% it was bilateral, although the right side was more severely affected. In 2%, the compression was on the left side only. The highest frequency of compression was at the T8–T10 level. The sympathetic trunk itself (ganglia and cord) was affected only by vertebral osteophytes at the lowest thoracic levels; however, bony excrescences due to costovertebral joint arthritis were frequently found impinging on the sympathetic trunk and its rami communicantes, with similar frequencies on both sides (24).

We assume that symptoms result either from irritation (stimulation) of the sympathetic structures or from inhibition (paralysis), according to the degree of compression. In our opinion, many pathologic conditions might be explained by compression of osteophytes on sympathetic structures (24).

Effects of Nerve Fiber Compression

Some degree of nerve fiber deformation is probably created by 30 to 50 mm Hg (25, 26). According to Hahnenberger (25) and Ochoa (26), these changes are probably reversible if the compression is released after a single trauma. Sustained compression at these pressure levels or repeated compression is likely to create disturbances in nerve structure and function (26). Higher pressures may lead to different kinds of nerve lesions, either segmental demyelination or, in cases of severe trauma, loss of axonal continuity, leading to Wallerian degeneration. A nerve root lesion associated with a herniated nucleus pulposus probably creates demyelination and Wallerian degeneration.

Rydevik et al. (12) placed a small inflatable cuff around peripheral nerves in animals and demonstrated that compression at 30 mm Hg and higher could block axonal transport. Chronic nerve entrapments with long-standing blockage of axonal transport can lead to Wallerian degeneration of the axons distal to the lesions. These axons, however, can regenerate, a process that in humans may take place at a speed of about 1 mm/day in optimal conditions.

Critical Pressure Levels

A pressure of 30 to 50 mm Hg applied to a peripheral nerve results in changes in intraneural blood flow, vascular permeability, and axonal transport. No measurements have been performed in vivo on the pressure levels acting on a nerve root due to a herniated disc, for example. Some data can be extrapolated, however, from existing knowledge on the pressures generated by swelling of the nucleus pulposus. It has been demonstrated in vitro that specimens of nucleus pulposus can generate pressures of several hundred millimeters of mercury if exposed to free fluid within the confined space (27–29). If a sequestered fragment of nucleus pulposus is displaced into the foramen, one can speculate that the nearby nerve root could be compressed at high pressure levels by the swelling disc fragment. The validity of this hypothesis remains to be proved experimentally, however (7). The "edge effect" (Fig. 3.12) in neural damage by compression refers to the injuries seen in nerve fibers and intraneural blood vessels at the edges of the compressed segment, with sparing in the center (14, 30). Compression of a nerve at high pressure can induce intraneural damage, leading to functional deterioration at the compressed segment, but with preserved axonal continuity and nerve function proximal and distal to the compressed segment.

Compression of normal peripheral nerve or nerve root may induce numbness, but it usually does not cause pain. Experimental investigations on human peripheral nerves in vivo have indicated that the numbness induced is a result of ischemia, not mechanical nerve fiber deformation, of the compressed segment. If a nerve root—or a peripheral nerve—is the site of chronic irritation, however, even minor mechanical deformation can induce radiating pain. This has been demonstrated by placing sutures or inflatable catheters around nerve roots at the time of surgery for herniated discs and postoperatively inducing stretching or compression of the nerve root. Lindblom and Rexed (31) investigated a large number of postmortem specimens of the lumbar spine with special reference to the relations between disc herniations and nerve root compression. They found that the dorsal root ganglion often was deformed by the pressure from the intervertebral disc. The DRG thus is frequently compressed by herniated intervertebral discs, and experimental data indicate that compression of this nerve structure can induce radiating pain.

Obviously, mechanical factors are involved in nerve root injury in connection with intervertebral disc herniations. It also has been speculated that breakdown products from the degenerating nucleus pulposus may leak out to the root and induce a "chemical radiculitis. Nachemson (32) has measured pH in intervertebral discs intraoperatively and found high hydrogen ion concentration in some patients who had extensive adhesion formation around the nerve root. Such changes in the tissue electrolyte balance can lead to pain in various ways. Autoimmune mechanisms also have been proposed to be involved in the inflammatory tissue reactions seen around degenerating discs.

The nerve fibers react to trauma with demyelination or axonal degeneration, leading to changes in nerve function.

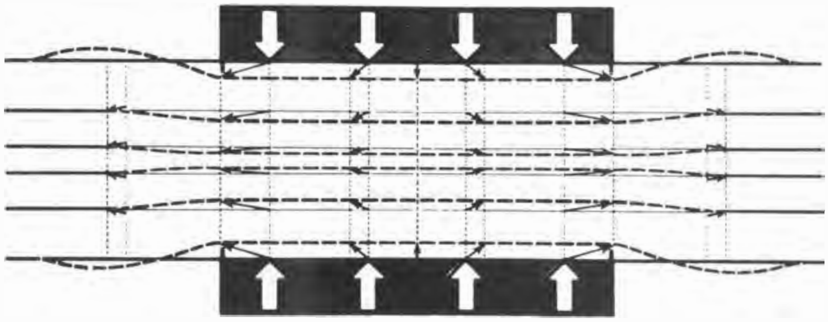

Figure 3.12. Schematic drawing, demonstrating the displacement of nerve tissue, which can be induced by circumferentially applied pressure. The pressure application leads to a bidirectional displacement of nerve tissue from the compressed nerve segment toward the noncompressed parts of the nerve. The *interrupted lines* show the positions of different tissue layers during compression. The *arrows* are vectors that indicate the displacement of nerve tissue components as a result of the applied pressure. Note that the displacement is maximal at the edges of the compressed segment. The diagram is based on computations performed by Professor Richard Skalak, Columbia University, New York, NY. (Reprinted with permission from Lundborg G, Rydevik B. Lakartidn [Sweden] 1982; 79:4035. In Rydevik B, Brown MD, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984;9(1):21. Copyright 1984, Lippincott-Raven.)

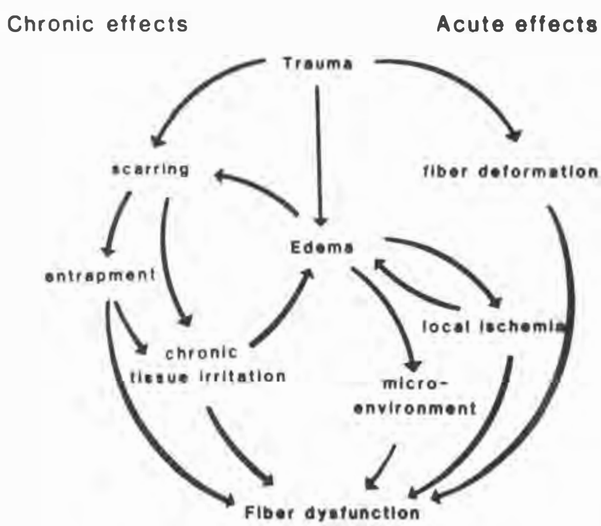

Figure 3.13. Proposed sequence of events leading to changes in nerve root function as a cause of acute and chronic compression. The dysfunction of the nerve fibers can be either loss of function or increased sensitivity to further mechanical stimulus. (Reprinted with permission from Rydevik B, Brown MD, Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 1984;9(1):8.)

Another important causative factor for the functional deterioration is the impairment of intraneural microcirculation and the formation of intraneural edema. In cases of chronic compression, intra and extraneural fibrosis can develop, which leads to further tissue irritation and establishes a chronic inflammatory process (Fig. 3.13).

The functional changes seen may be either loss of nerve function, seen as muscle weakness or sensory deficit, or a state of hyperexcitability of the nerve tissue. These two conditions can be present at the same time, which means that nerve fibers may have a decreased conduction velocity at the site of injury and be hypersensitive to further mechanical stimulus at the injured segment. The hyperexcitability can give rise to positive symptoms from the respective nerves (i.e., pain, paresthesia, and possibly muscle fasciculations).

Conclusion

The anatomic complex of nerve root, ganglion, and spinal nerve may be involved in pathologic processes in association with disc herniation and spinal stenosis. Compression of the nerve tissue may induce structural damage to the nerve fibers, impair intraneural blood flow, and form intraneural edema as well as axonal transport block (7).

The following points regarding the effects of compression on spinal nerve roots is based on the work of Sharpless (8):

1. Dorsal roots are far more susceptible to compression block than is the peripheral (sciatic) nerve. When pressure is applied for 3 minutes followed by 3-minute recovery periods, 100 mm Hg must be applied to the sciatic nerve to achieve the same conduction block that can be produced in spinal roots by 20 mm Hg.
2. Pressure as slight as 10 mm Hg, maintained for 15 to 30 minutes, reduces the compound action potentials of dorsal roots to about half of their initial values. With such small pressures, nearly complete recovery occurs in about 30 minutes.
3. It is probable that the compression block produced even by such small pressures is due to mechanical deformation rather than ischemia, because the larger fibers are blocked first, whereas anoxia is believed to affect small fibers first.
4. It has been shown elsewhere (8) that a pressure vessel model of a nerve predicts that large fibers would be most compressed, which may account for their susceptibility to blockage. The pressure vessel model might also account for the

progressive character of compression block, assuming a viscous flow of the fiber contents.

5. The slow onset of compression block would have adaptive value, because transient increments of pressure that occur in confined spaces during extremes of motion would have little effect.
6. Spinal nerves acquire a structural feature that protects them from compression block before they enter the intervertebral foramina. The sheath does not appear to play an important role. The nature of this protective feature is still unknown.

Comparison of Normal and Chronic Nerve Root Irritability

Compression of a normal nerve root can be associated with numbness and motor weakness, but it does not usually cause pain. However, if the nerve tissue is chronically irritated, mechanical deformation can induce radiating pain. Thus, intraneural inflammation seems to be a factor of importance in the pathogenesis of pain production in nerve root compression syndromes. It is debated whether such intraneural inflammation is the result of an inflammatogenic effect of nucleus pulposus on nerve tissue ("chemical radiculitis") or whether it is an effect of mechanical nerve root deformation by the herniated disc (33).

Injection under pressure of normal saline into a degenerated lumbar disc elicits pain that the patient experiences as identical to lumbago. The moment the pressure on the syringe is released, the pain disappears. It returns each time the pressure is increased. Identical experiments on a normal disc produce no pain. The amount of liquid that can be introduced into a normal nucleus pulposus is very small (34).

CHEMICAL RADICULITIS

Chemical Irritation of the DRG Results In an Autoimmune Response

Nuclear material leaking into the epidural space is considered "foreign" and an autoimmune response develops, which can lead to a chronic inflammatory response. Mononuclear cells infiltrating along the margins of the extruded discs expressed inflammatory mediators, and they might induce neovascularization and persistent inflammation (35).

Marshall et al. (36) describe a new pathologic concept termed *chemical radiculitis*. This lesion occurs when the anulus fibrosus has been weakened by intervertebral disc degeneration and finally ruptures under the stress of some traumatic episode. At that time nuclear fluid is of a waterlike consistency. It is ejected into the peridiscal tissues and tracks down to the nerve root. Because of its glycoprotein component, nuclear fluid is highly irritating to nerve tissue, and sudden severe sciatic pain results.

Liberation of nuclear fluid from the sealing anulus fibrosus capsule converts the role of its glycoprotein into one of an antigen, which results in antibodies detectable in high titer in the bloodstream after a 3-week interval. This is an autoimmune reaction. Chemical radiculitis can explain some or possibly all cases of acute or chronic inflammatory change around the nerve root.

Walk (36) suggested that two neurologic syndromes were caused by the lumbar intervertebral disc: (*a*) compression of the nerve by the disc; and (*b*) irritation of the nerve by the perineural spread of the contents of the nucleus pulposus occurring through a disc rupture.

If chemical radiculitis is to be accepted as a viable theory, several important factors need to be verified, such as: (*a*) Can nuclear fluid reach the nerve root? Lindblom and Rexed (31) dissected 160 cadavers and demonstrated a connecting pathway from the nucleus via the anular rupture to the root. (*b*) Is nuclear fluid in a liquid form at some stage of the degenerative process? These findings have been noted by many individual observers at operation and verified by Armstrong and Walk. Armstrong (37) has reported incising the anulus on occasions in which fluid has squirted out of the wound.

If the immunologic research proves to be correct, a rupture of the anulus with consequent liberation of nuclear fluid into the tissues will be followed by a high serum titer to glycoprotein. Thus, for the first time, a serum test will be available to detect a valid disc lesion 3 weeks after the initial pain. Armed with this knowledge, the correct immediate treatment could be administered.

Chemical radiculitis is an inflammatory condition of the nerve root caused by the rupture of the anulus fibrosus and dissemination of disc fluid along the nerve root sheath. The inflammatory component of disc fluid is glycoprotein. The inflammation is a reaction to repeated injuries of the spinal column (e.g., in occupational lifting of heavy loads). Rupture of the anulus fibrosus and liberation of disc fluid into the tissues also evoke circulating antibody response and autoimmune reaction. A high titer to glycoprotein at 3 weeks after an acute attack of back pain is evidence of the presence of a significant disc lesion. In selected cases, immediate relief from pain has occurred after administration of cortisone or a suitable cortisone derivative. Prolonged rest may be contraindicated because of the risk of formation of radicular adhesions (36, 38).

Although an inflammatory component in degenerative disc disease is known to occur, the chronicity of this process requires further evaluation. An autoimmune mechanism as a possible cause of prolonged signs and symptoms once a herniated nucleus pulposus has been considered. Clinical evidence for this is found in patients who develop recurrent signs and symptoms at the same level following surgery. Factors important in the autoimmunity theory are:

1. Degenerative disc disease of the lumbar spine is mediated in some patients by an inflammatory component.
2. The chronicity of the inflammation may have an autoimmune basis.
3. The leukocyte migration-inhibition test has demonstrated the presence of a cellular immune response in patients

whose discs were found to be sequestrated at the time of surgery.
4. No human humoral antibody could be demonstrated (39).

Degenerative changes in cartilage set off a series of well-defined pathoanatomic reactions in surrounding structures, which applies to the synovial joints as well as to the intervertebral discs. The structures adjoining the cartilaginous area become the focus of vascularization, which at times results in the formation of granulationlike tissue. In discs, this vascular reaction does not occur until the degenerative process has reached the outer layers of the anulus, where a capillary network is present. The disc itself is avascular. An extensive capillary network can also be seen as an ingrowth into a degenerated disc.

The chemical components of the material in discs are not constant. It seems likely that morphologic changes in the disc may release polysaccharide-bound proteins into surrounding structures outside the anulus, where these substances act as foreign elements, because the lack of vascular communication with the disc normally keeps them enclosed inside the intervertebral space. It is possible that the response could be interpreted as autoimmunization (i.e., the production of a reactive inflammation). Animal experiments with transplantation of disc material to other areas have produced results that are not incompatible with this theory. Therefore, it is felt that we should pay attention to the chemical and immunologic aspects of disc reactions on surrounding structures if this novel concept of low back pain is to achieve increased biologic relevance.

If we continue to believe that the disc is the origin of symptoms, this must be ascribed to certain properties of the disc that are active during a certain period of life are lost at a later stage. It is not inconceivable that the physical properties of the disc produce more severe mechanical disorders at the beginning of the degenerative process than during its late stages, when the disc has collapsed and lost most of its function. This theory is supported by numerous morphologic and mechanical data. If in some way we could affect the degenerative process by speeding up the conversion of the intervertebral disc into a fibrous structure with different behavior, we might have succeeded in finding a solution to the problem (34).

The chemical irritation of the nerve root in association with disc prolapse is the likely cause of the acute pain following injury. This view has arisen from the frequent operative finding of a swollen, inflamed nerve root without bone pressure. The chemical content of the nerve root includes glycoprotein. Previously, it was shown that the carbohydrate capsule of the pneumococcus liberates histamine and other H substances from perfused organs in much the same way as venom. Direct pharmacologic tests of nucleus pulposus show the presence of 1 to 4 μg of histamine per gram, but no tryptamine, slow-reacting substance, or kinin. Extract of the glycoprotein from human nucleus pulposus released considerable quantities of histamine, edema fluid, protein, and another amine with four times the mobility of histamine from the isolated perfused lung of the guinea pig. Acute pain in disc injury is caused by local irritation of the nerve root that produces edema and releases protein and H substances at the site of injury. Relief of pain by cortisone accords with these findings, because cortisone inhibits the peripheral response to H substances (41).

We have seen that mechanical compression of the nerve root is a source of low back and sciatic radiculopathy, and that chemical radiculitis is a new and scientifically equally exciting cause of nerve root irritation. What effects such mechanical and chemical irritants will have on trophic function of nerves is yet to be learned. Certainly, chiropractors can be excited about the interest in an area of the human anatomy—the spinal nerve root—that has been the primary focus of their contribution to the healing arts for almost 100 years.

Piriformis Muscle Syndrome Caused by Chemical Irritant

Sciatic neuritis is now believed to result from irritation of the sciatic nerve sheath, which is caused by biochemical agents released from an inflamed piriformis muscle where the two structures meet at the greater sciatic foramen. The symptoms of piriformis syndrome present almost identically to those of lumbar disc syndrome, except for the consistent absence of true neurologic findings. Diagnosis is accomplished by palpation of myofascial trigger points within the piriformis muscle. Treatment, which consists of a conservative approach employing local anesthetics and osteopathic manipulation, is without significant risk. Reducing muscle spasm, restoring joint motion, and keeping the patient ambulatory and in motion are keys to successful treatment (40).

Pain Receptors In Low Back Pain

According to Livermore (42) there are two basic types of sensory nerve fibers: (*a*) type A, which are large, myelinated fibers that conduct impulses quickly and tend to conduct modalities of touch and pressure; and (*b*) type C, which are fine, unmyelinated fibers that conduct impulses slowly and transmit pain and temperature perception. Peripheral sensory fibers carry both A and C fibers and run with the motor portion of the nerve.

The course of a pain impulse is as follows: peripheral sensory ending→common root at intervertebral level via ventral ramus→dorsal root→dorsal root ganglion cell bodies→spinothalamic tracts (lateral carries type C fibers for pain and temperature, anterior carries A fibers for touch and pressure)→thalamus (for organization and modification) (42).

Two types of pain sensation are found: (*a*) deep pain (splanchnic pain—associated with C fiber irritation—dull, deep, aching, diffuse pain that follows myotomes and sclerotomes. Referred pain is close to the site of pathologic condition. Pain is of later onset—more disabling and difficult to localize. (*b*) Superficial pain—called *somatic* pain—sharp, localized, and carried by type A fibers. Pain follows the dermatome (42).

Chemical Radiculitis Occurs Via Epidural Transport to the Nerve Roots

A rapid transport route between the epidural space and the intraneural capillaries exists; therefore, nucleus pulposus material, as well as epidurally applied substances (e.g., local anesthetic drugs or epidurally injected corticosteroids), may have a rapid, direct transport route to the spinal nerve root axons (43). Nociceptors respond to chemical, mechanical, and thermal stimuli; damage to a peripheral nerve results in physiologic, morphologic, and biochemical changes that act as a focus of pain. Reduced food supply to myelinated fibers results in demyelination. Inflammatory response to irritation results in a combination of inflammatory mediators such as potassium, serotonin, bradykinin, substance P, histamine, and products of arachidonic acid metabolism (44).

Substance P Is Found in Nerve Fibers of the Intervertebral Anulus Fibrosus

The intervertebral disc is innervated by the sinuvertebral nerve and by branches of the sympathetic trunk. Nerve fibers extend into the anulus fibrosus either as free fibers or in association with blood vessels. Some of these nerve fibers in the outer anulus in humans are immunoreactive for SP. Substance P is a neuropeptide that, when released from the central terminals of primary afferent neurons in the dorsal horn of the spinal cord, acts as a neurotransmitter or neuromodulator. Substance P is transported antidromically to peripheral nerve endings where it contributes to inflammatory processes such as mast cell degranulation, vasodilation, and increased capillary permeability in skin and joints.

Substance P is a member of the tachykinin group of peptides. The biologic effects of the tachykinins are mediated by their interaction with specific high-affinity receptors on cell surfaces. It stimulates endothelial cell proliferation and migration, essential features in the formation of a new vascular network, suggesting that SP could itself be a contributory factor in neovascularization in human intervertebral disc. The effect of increased blood flow and angiogenesis caused by SP stimulus may promote tissue repair and improved nutrition; however, sustained high levels could contribute to inflammation (45).

Phospholipase A_2 (PLA_2) Inflammatory Enzyme

Herniated and degenerated lumbar discs show high levels of this inflammatory enzyme, which may result in the biochemical rather than mechanical cause of pain (46). The liberation of phospholipase A_2 from a herniated disk could also cause direct inflammation in the surrounding region. Nerve endings have also been described in the granulation tissue of degenerated discs, which had previously been without innervation (47). PLA_2 has also been seen elevated in patients with malaria, endotoxic shock, peritonitis, psoriasis, and pancreatitis, and in the synovial fluid, serum, and bronchial lavage fluid of patients with arthritis. PLA_2 released from nuclear material might bind to the nerve sleeve and other tissue of the neural canal to act as a proinflammatory agent (48). Gronblad et al. (49), however, report the phospholipase A_2 levels no higher in herniated or degenerated disc tissue than in normal discs.

Phospholipase A_2 was found in the facet synovial fluid of 28 low back pain patients with associated sciatic or femoral neuropathy. At surgery lateral recess stenosis was found as a result of facet joint hypertrophy; decompression surgery with excision of the facet joints and release of nerve root adhesions had gratifying results on these patients. Inhibition of PLA_2 may prove beneficial in future treatment of arthritis and low back pain (50).

Cytokines

Lumbar disc herniations show inflammatory cytokines such as interleukin-1, which increases prostaglandin E_2 production. Further studies are required to elucidate the role of inflammatory cytokines in causing sciatic pain (51).

IgG and IgM Elevation

Spiliopoulou et al. suggest that IgG and IgM trigger a local inflammatory process at the nerve root exposed to nuclear material, which may be the causative agent of the low back pain associated with sciatica (52). Chemical irritants stimulate the dorsal root ganglion, resulting in referred pain (dysesthesias) without root tension signs or neurologic deficit. The sinuvertebral nerve is a sensory afferent nerve that relays pain stimuli from nociceptive free nerve endings (52). Chronic inflammation can occur as the nucleus pulposus becomes accessible to the vascular system and an autoimmune response occurs (47).

In 29 of 52 disc herniations (56%), immunoglobulin M deposits were observed, and in 18 of 52 disc herniations (35%) immunoglobulin G could be demonstrated. The results lend support to prior suggestions of inflammation and immune reaction in disc herniations, including previous biochemical studies suggesting immunoglobulin deposition. The exact role of the demonstrated immunoglobulins in disc tissue pathophysiology is not clear (53).

Prostaglandins

Prostaglandin (PG) and leukotriene (LT) released from human disc and lumbar facet joint tissue has been documented with high levels of PLA_2 in the human disc. PLA_2 releases fatty acids from lipid membranes. These can be converted to PG and LT, which are potent inflammatory mediators and purported to be involved in lumbar diseases (54).

Three recent studies have shown elevated levels of prostaglandin E_2 in intervertebral disc herniations. Sequestrated discs tend to be associated with a higher prostaglandin E_2 content than extruded discs, which in turn tend to be associated with higher prostaglandin E_2 content than protruded discs. A positive straight leg raising test appeared to be associated with a higher prostaglandin E_2 content than a negative test (55).

Substance P and Calcitonin Gene-Related Peptide Released In Joints and DRG

Substance P (SP) and calcitonin gene-related peptide (CGRP) cause vasodilation and increase plasma protein extravasation in-

duced by tachykinin. Severely affected arthritic joints have the highest concentrations of SP. Thus, neuropeptides may play a significant role in degenerative spine disease.

The dorsal root ganglion is sometimes referred to as the "brain" of the spinal motion segment or functional spinal unit. Within it, SP and CGRP are co-localized in small ganglion cells of rats and cats, in the sensory parts of the nervous system.

Because of the dorsal root ganglion's vascular supply and tight capsule, it is suggested that mechanical compression of the ganglion may result in intraneural edema and subsequent decrease in cell body blood supply, accounting for abnormal DRG activity and pain. Anatomically, the DRG serves as a vital link between the internal and external environment and the spinal cord (56).

Chemical Radiculitis of the Nerve Root by Nuclear Material

Epidural application of autologous nucleus pulposus in pigs, without mechanical nerve root compression, induced a pronounced reduction in nerve root function after 1 to 7 days because of possible direct biochemical effects of nucleus pulposus components on nerve fiber structure and function and microvascular changes, including inflammatory reactions in the nerve roots (57).

Epidural application of autologous nucleus pulposus without any pressure may induce nerve function impairment and also axonal injury and significant primary Schwann cell damage with vesicular swelling of the Schmidt-Lanterman incisures (58). High-dose methylprednisolone administration within 24 to 48 hours after epidural application of autologous nucleus pulposus reduced the inflammatory changes (59).

Mechanism of Action of Steroid Injections Is Offered

Compression on a nerve root by disc, facet, pedicle, or ligaments impedes blood circulation within the nerve root, resulting in swelling and venous congestion. Inflammatory chemicals may leak from the degenerative disc into the nerve root, causing a chemical radiculitis. These changes can result in nerve malnutrition, causing cellular changes in the nerve fibers; and also electrophysiologic changes and abnormal nerve impulses.

The nerve root compression causes sensitization of nerves in both directions from the lesion, centrally and peripherally. The nerve endings thus stimulated, produce more and more aberrant impulses. The net result is a "vicious cycle" of pain-producing impulses and sometimes severe radicular pain. It may be possible to disrupt the "vicious cycle" through injections of steroids into the nerve root, even if the steroids do not actually reach the site of compression (60).

Nuclear Material Causes Inflammation and Fibrosus of Epidural Space

The nucleus pulposus (escaping from the intervertebral disc), lactic acid (from anaerobic glycolysis in the disc), chondroitin sulfate (a component of glycosaminoglycans in the disc), or synovial fluid (from degenerating facet joints) cause inflammation in the meninges if they contact the dura mater. Nucleus pulposus produced significant fibrosus in the arachnoid and epidural spaces; the other substances did not cause fibrosus or inflammation. This suggests that leakage of nucleus pulposus into the epidural space causes an inflammatory response in the arachnoid and epidural spaces (61).

Nucleus pulposus cells (chondrocytes, fibrocytes, notochordal cells, and secretory stellate cells) induce the pain response at the nerve root through immunologic, inflammatory, or other mechanisms; it is not the proteoglycan constituent of the nuclear material that causes the irritation (62).

Herniated Disc Material Contains Nerve Fibers That Produce Chemical Irritants

Nerve terminals were found in 83% of 35 herniated discs that produced substance P and sympathetic C-flanking peptide of neuropeptide Y, which could be involved in the mechanism of discogenic pain and inflammation (63).

Nervous and Immune Systems Interact

It has become increasingly apparent that the nervous and immune systems are not entirely independent of each other. For example, a variety of cytokines is released in the vicinity of the nerve root by phagocytic and antigen-presenting cells of the immune system. The cytokines play an important role in the orchestration of the immune response. The hyperalgesic effects of each type of cytokine differ with hyperalgesia resulting within hours after injection into the rat paw (64).

MECHANICAL AND CHEMICAL IRRITATION COMPARED AS CAUSE OF RADICULOPATHY

The neurochemical and neurophysiologic factors associated with lumbar nerve root irritation were studied via a rat study model using compression and chemical approaches to nerve root trauma (65). The L4–L6 nerve roots and dorsal root ganglia were surgically exposed, and silk ligature with and without chromic gut was placed around the nerve root or ganglion (Fig. 3.14) Motor function and the reflex responses to noxious thermal and mechanical stimuli were measured in the rats preoperatively and at 1 to 12 weeks postoperatively. Increased patterns of substance P, calcitonin gene-related peptide, and c-fos changes within the dorsal root ganglia reflect possible disturbance of axonal transport or an increased production in response to nerve irritation.

Mechanical constriction of lumbar spinal nerve roots, as evidenced by a loss of myelinated fibers, is not sufficient to produce the behavioral effects associated with lumbar radiculopathy. The chromic chemical irritation may play a role in the pathophysiology and development of the behavioral but not the histologic changes in this rat model of lumbar radiculopathy (65).

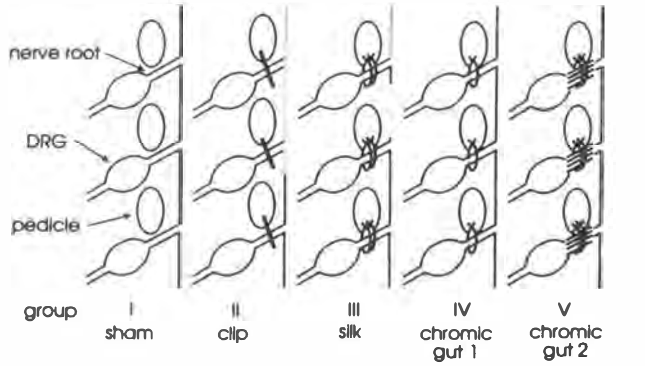

Figure 3.14. Schematic of surgical procedures: Sham operation to totally expose the L4, L5, and L6 nerve roots and dorsal root ganglia on the left side. (*I*) Nerve root clipping, where the nerve roots were clipped with a microhemoclip (*II*); 4–0 silk ligature, where two loose ligatures of 4–0 silk were placed around the nerve roots (*III*); 4–0 chromic gut 1, where one loose ligature of 4–0 chromic gut was placed around the nerve roots (*IV*); 4-0 chromic gut 2, where four 0.3 cm pieces of 4–0 chromic gut were laid adjacent to the nerve roots and secured by two loose ligatures of 4-0 chromic gut (*V*). (Reprinted with permission from Kawakami M, Weinstein JN, Spratt KF, et al. Experimental lumbar radiculopathy: immunohistochemical and quantitative demonstrations of pain induced by lumbar nerve root irritation of the rat. Spine 1994; 19(16):1780–1794. Copyright 1994, Lippincott-Raven.)

Mechanical Nerve Root Compression Alone May Not Cause Radiculopathy

Many patients who have no symptoms, in particular pain, show obvious compression of the nerve root or cauda equina in myelographic, computed associated tomographic, and magnetic resonance imaging studies, which suggests that mechanical compression of a nerve root is not necessarily associated with leg pain. An autoimmune reaction from exposure to disc tissues and/or an increased concentration of lactic acid and a lower pH in the region of the nerve roots correlate with the clinical signs of radiculopathy. Chemical inflammatory reaction within or around the nerve roots may be necessary to produce radiculopathy (66).

Herniated lumbar discs make spontaneously increased amounts of matrix metalloproteinases, nitric oxide, prostaglandin E_2, and interleukin-6, which may be involved intimately in the biochemistry of disc degeneration and the pathophysiology of radiculopathy implicating a biochemical processes in intervertebral disc degeneration (67). Arachnoid cells may initiate or sustain the intradural inflammatory reaction found in cervical myeloradiculopathy (68).

Inman and Saunders (69), commenting on the concept that sciatica is caused solely by pressure on the nerve root, stated that this is not borne out by the existing experimental evidence on the effects of pressure on nerves, and they found no experimental evidence to indicate that pressure alone on the nerve root initiates pain of this characteristic type.

From a series of 22 patients with sciatica caused by intervertebral disc pressure, eight were chosen. Each of these patients had classic sciatica with an unequivocal disc herniation pressing on the fifth lumbar and first sacral roots demonstrated at operation (69). The disc material was removed through a small aperture cut in the ligamentum flavum. An effort was made to center this aperture directly over the protruding disc. When the disc material was removed, a loop of nylon thread was passed around the involved root and its two ends brought to the surface. It was so placed that, when the slack was taken up, the loop pressed on the root at the same place as the disc had. It tended to maintain this relation to the root because of its passage through the small aperture in the ligamentum flavum directly above it. It was hoped that, by pulling on this nylon thread to bring it in contact with the root, the effects of disc pressure would be closely simulated.

The experiment was performed on the first postoperative day in three patients, on the 14th day in one, and on the 10th day in the remaining four. In all these eight patients, symptoms were completely relieved by removal of the disc.

In 11 patients, 10 of whom had herniated discs, the ligamentum flavum, interspinous ligament, and anulus fibrosus were tested instead of a nerve root or the dura mater. In eight patients, one nylon suture was passed through the ligamentum flavum and one through the interspinous ligament. In two patients, the ligamentum flavum was tested; in the last, the anulus fibrosus alone was tested.

It has been established that the nerve root need only be touched to cause sciatica. It would also appear that if touched repeatedly or continuously the root becomes hypersensitive (69).

MECHANICAL COMPRESSION OF THE DRG IN THE NEUROFORAMEN

The DRG was studied for compression by the superior articular facet and/or degenerative bulging discs in 35 cadavers. The incidence of indentation on the DRG was highest when located in the proximal foramen, whereas extraforaminally located ganglia had the lowest incidence of indentation. The incidence of indented DRG increased with age. The possible correlation between these observed anatomic abnormalities and clinical symptoms must be further elucidated (70).

Another Explanation of Pain Transmission and Locus

Pain is usually thought to occur when a receptor is stimulated, resulting in transmission of the impulse to the brain for interpretation; however, if a nerve is stimulated between the receptor and the brain, the pain may be recognized at the receptor site even though it is not the origin of stimulation. This explains the mode of pain felt in the sciatic nerve when the nerve root is compressed by a herniated disc, resulting in pain not being localized to the lumbar spine but rather to the receptor site of the compressed axon (71) (Fig. 3.15).

Pain Perception Neural Transmission

Pain perception begins with activation of peripheral nociceptors and conduction through myelinated A and unmyelinated C

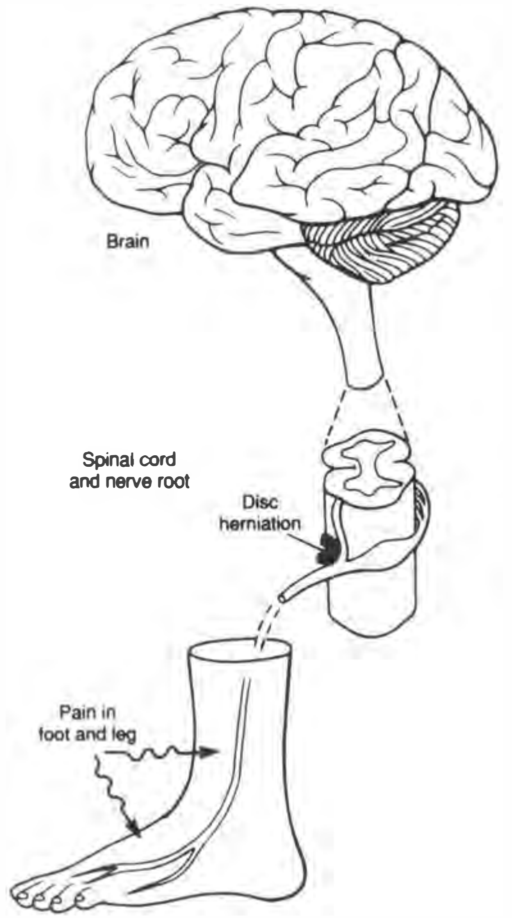

Figure 3.15. When a herniation of an intervertebral disc affects a nerve root in the lumbar spine, the brain may interpret the pain as coming from the end of the axons involved (i.e., from the foot and leg). (Reprinted with permission from Olemarker K, Hasue M. Classification and pathophysiology of spinal pain syndromes. In: Weinstein JN, Rydevik BL, Sonntag V, eds. Essentials of the Spine. New York: Raven Press, 1995:19. Copyright 1995.)

fibers to the DRG. From here, signals travel via the spinothalamic tract to the thalamus and the somatosensory cortex. Descending pathways from the hypothalamus, which have opioid-sensitive receptors and are stimulated by arousal and emotional stress, transmit signals to the dorsal horn that modulate ascending nociceptive transmissions.

Nociceptive receptors are free nerve endings found throughout the body in skin, viscera, blood vessels, muscle, fascia, and joint singular capsules. Nociceptors also have neuroeffector functions. They release neuropeptides from the cell bodies in the dorsal horn (e.g., SP, CGRP) that act on peripheral cells (72).

DRG Is Highly Sensitive to Irritation

Hypoxia Changes of DRG with Compression

Dorsal root ganglia are more highly sensitive than the dorsal root to mechanical compression and hypoxia, and they are closely related to abnormal sensations and radiculopathy. A hypoxic condition evokes spontaneous firing and increased sensitivity to mechanical stimuli in the DRG. Nerve conduction firing velocity caused by mechanical compression or hypoxia ranges between 20 to 35 m/sec (73). Inhibitory synaptic transmission is depressed preferentially in the early phase of hypoxia. Excitatory and inhibitory transmissions are suppressed in prolonged severe hypoxia. Glucose deficiency has aggravated hypoxic inhibition of synaptic transmissions (74).

Vasculature of the Spinal Nerve Roots and the Effect of Compression On Blood Flow to the Nerve Roots

Nerve Roots Are Susceptible to Ischemia

Nerve roots lack the continuous blood supply of regional arteries and veins. This results in a nutritional deficit if a nerve root is compressed at two locations: the area of nerve root between two points of compression is more aggravated than nerve root tissue compression at one point only, and the symptoms are more severe for the patient. Figure 3.16 shows the differing blood supply to nerve root and peripheral nerve (71).

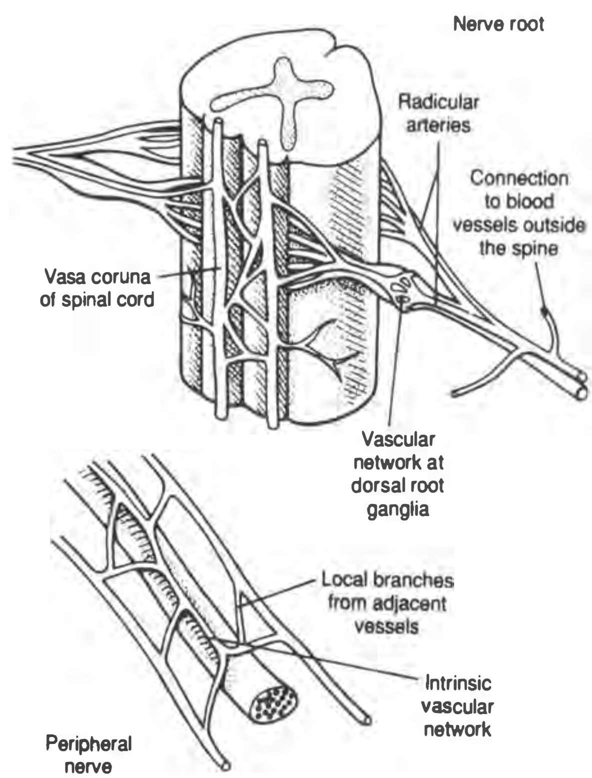

Figure 3.16. The vascular supply to the nerve root comes from the spinal cord and peripherally from blood vessels outside the spine. Radicular arteries are formed, and they supply the nerve root from both directions. The nerve root has no connections to surrounding vessels. In the peripheral nerve, however, the vascular system of the nerve has numerous connections to surrounding vessels along its course. (Reprinted with permission from Olemarker K, Hasue M. Classification and pathophysiology of spinal pain syndromes. In: Weinstein JN, Rydevik BL, Sonntag V, eds. Essentials of the Spine. New York: Raven Press, 1995;22. Copyright 1995.)

The segmental arteries generally divide into three branches when approaching the intervertebral foramen: (*a*) an anterior branch, which supplies the posterior abdominal wall and lumbar plexus; (*b*) a posterior branch, which supplies the paraspinal muscles and facet joints; and (*c*) an intermediate branch, which supplies the contents of the spinal canal (75).

Nerve Roots Depend On Nutrients from Cerebrospinal Fluid and Arterioles

Nutrients can be transported to the nerve roots both by the intrinsic blood vessels and via diffusion from the cerebrospinal fluid. Ten millimeters of mercury was sufficient to induce a reduction of methylglucose transport to the nerve roots by 20 to 30% as compared with control (76).

Reduced Blood Flow to the Nerve Root

The intraneural blood flow between two compression balloons on a pig nerve root showed that at 10 mm Hg compression, total blood flow decreased 64% in the uncompressed segment compared with precompression values. Total ischemia occurred at pressures 10 to 20 mm Hg below the mean arterial blood pressure. After two-level compression at 200 mm Hg for 10 minutes, intraneural blood flow gradually recovered toward the baseline. Recovery was less rapid and less complete after 2 hours of compression. Double-level compression of the cauda equina can thus induce impairment of blood flow, both at the compression sites and in the intermediate nerve segments located between two compression sites, even at very low pressures. These findings may have clinical importance in the understanding of the pathophysiology of multiple level cauda equina compression (76).

Compression compromised cerebrospinal fluid percolation through the cauda equina and spinal nerve roots with venular ischemia occurring at 30 mm Hg compression and arteriolar compromise at approximately 60 to 70 mm Hg on the nerve root. Diffusion of metabolites and nutritional support mechanisms are markedly reduced (77).

Extradural nerve root compression, often seen in degenerative conditions of the spine, disturbs the blood flow in the proximal part of the nerve root more than the distal part. Block of the cerebrospinal fluid flow around the nerve root on the distal side of the compression, however, provokes reduction of the blood flow to a certain extent not only in the distal part of the nerve root but also in the dorsal root ganglion, suggesting that clinical symptoms derived from the dorsal root ganglion may exist even when it is not compressed directly (78).

Lower Extremity Vascular Abnormalities Secondary to Sciatic Radiculopathy—Reflex Sympathetic Dystrophy

Abnormality in vascularization of the lower extremity was found in 24 (80%) of patients with sciatica and in 11 (68.7%) of the patients with low back pain. The median blood flow difference was −12.5% and +4%, respectively, in these two groups versus +2.9% in the control group. Vascular perfusion abnormalities observed in patients with sciatica secondary to disc herniation, which may be more important than previously considered, possibly result from alteration in sympathetic vascular autoregulation. Compression and inflammation of the root by the disc could distort the signals coming from the lower extremities and heading toward sympathetic centers responsible for blood flow to the extremities. This mechanism would be analogous to reflex sympathetic dystrophy (79).

Intraneural Edema with Compression of the Nerve Root

Permeability of the endoneurial capillaries of the nerve roots under compression is altered to cause edema when 2 minutes of 50 mm Hg compression is applied. Intraneural edema may increase the endoneurial fluid pressure. Such increased pressure may impair the endoneurial capillary blood flow and in this way impair the nutrition of the nerve roots. Nutritional impairment probably occurs within seconds to minutes after starting the compression (75).

Compression of the Herniated Disc Is Greater Than Spinal Stenosis

A herniated or protruded disc may induce much higher compression pressure levels than central spinal stenosis. Also, nerve roots compressed by disc material often show signs of chemical inflammation from nuclear leaking. The root sleeves are innervated by the recurrent nerve of Luschka that runs in a cranial direction from the caudal end of the nerve roots. Pain recorded by this nerve might be referred to the dermatome of that specific nerve root level. The pain induced by the straight leg-raising test simply might be an effect of the irritated meninges sliding over herniated disc tissue.

The most recently observed substances that might be injurious to the nerve tissue per se are cytokines, which can leak from, for instance, degenerated facet joints. In an experimental model, cytokines may have impaired nerve impulse conduction of rat sciatic nerves (75).

Chronically Compressed Nerve Roots Adapt to Compression Effects

Time and pressure controlled compression of dog cauda equina indicate that a metabolism or vascularity adaptation process occurs in compressed nerve tissue. It may be more difficult than previously assumed to study neuroischemic nerve changes in noncompressed nerves (80).

DRG Compression by Herniated Disc Causes Sciatica More Than Dorsal Horn Compression

Three types of mechanoreceptive neurons are seen in the dorsal horn of the spinal cord:

1. Nociceptive-specific neurons—respond to strong, overt noxious mechanical stimulation of the skin.
2. Wide-dynamic-range neurons—respond to general mechanical stimulation of the skin.
3. Low-threshold mechanoreceptive neurons—respond to noxious stimuli.

The DRG has a mechanically sensitive nervi-nervorum that can be activated by compression. The DRG blood supply and tight capsule may cause intraneural edema on compression that accounts for pain. The DRG is more susceptible to mechanical compression than the dorsal root. Radicular pain associated with a herniated intervertebral disc initially results from compression of the DRG. Mechanical compression of either the dorsal root ganglion or chronically injured roots can produce hyperpathic symptoms of paresthesia, hyperalgesia, and allodynia associated with herniated intervertebral disc (81).

Substance P Accumulates in the DRG at 4 Weeks of Chronic Compression

Substance P is mainly accumulated within the nerve root tissue with a smaller increase in the DRG after 1 week of chronic compression. At 4 weeks the DRG accumulates greater amounts of SP. This suggests that amounts of SP accumulation may be related to pain production occurring during controlled nerve root compression (82).

DRG Surgical Removal Relieves Sciatica

Dorsal root ganglionectomy in 61 patients gave 60% of them relief of intractable monoradicular sciatica. Dysesthesia, which continued in 60% of the patients, was relieved with systemic lidocaine (83).

SUMMARY OF CAUSES OF NERVE ROOT IRRITATION

A working hypothesis for the pathomechanism of radicular pain is proposed. When the nerve root is involved, mechanical and circulatory changes are produced. Inflammatogenic materials may leak from the degenerative disc and facet into the nerve root, causing chemical radiculitis. These changes can be followed by nerve fiber and cell changes including blockage of axonal flow and demyelination, causing ectopic discharges and cross talk. Disturbed or enhanced synthesis and transport of neuropeptides can also be elicited. These multifactorial changes may finally result in sensitization of both the central and peripheral nervous systems, causing radicular pain (84).

The dorsal root ganglion has a rich blood supply. Seventy percent of its nutrition is supplied by cerebrospinal fluid. Consequently, if the cerebrospinal fluid flow is blocked, as in the case of adhesive arachnoiditis, nerve root nutrition is markedly disturbed.

Movement of the nerve root by flexion of the whole spine is marked in the upper lumbar spine, whereas movement by straight leg raising maneuvers happens mostly in the L5 and S1 roots. The elastic limit of the nerve root is 15% of its length, and if stretched more than 21%, complete failure can occur.

The normal DRG can produce spontaneous ectopic discharges and reflected impulses as well as mechanically induced discharges.

Inflammatogenic materials, such as PLA_2 and synovial cytokines, may leak from the degenerative disc or facet joint into the nerve root, causing "chemical radiculitis" (84).

HOW MUCH DISC REDUCTION IS NECESSARY TO RELIEVE PAIN?

Next will be shown cases from my practice showing varying degrees of reduction in disc herniation size in patients receiving relief of their low back pain and sciatica. Based on the discussion in this chapter, it can be seen that much relief can be gained from dissipation of chemical irritants with or without reduced compression by disc herniation. Diagnostic imaging of both pre and post-treatment relief cases I have treated will be presented.

Case 1

This is a case showing the reduction of disc protrusion on pre- and post-treatment computed tomography (CT) scan. This patient was totally relieved of low back and leg pain, but you can see that there was far less than 100% reduction of the disc protrusion. Figure 3.17 reveals that the L5–S1 intervertebral disc protrusion occupied 38% of the sagittal diameter of the vertebral canal. Following complete relief of low back and leg pain, the percentage occupied by the intervertebral disc protrusion was still 38% (Fig. 3.18).

Figure 3.19 reveals that the L4–L5 disc protrusion occupied 50% of the sagittal diameter of the vertebral canal. Six months later, however, the intervertebral disc protrusion occupied 37.5% of the total canal; this is a 12.5% reduction in the size of the disc protrusion (Fig. 3.20).

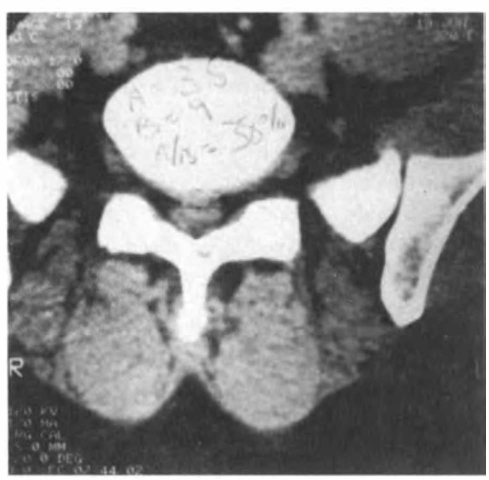

Figure 3.17. At L5–S1, 38% of the sagittal diameter of the vertebral canal is occupied by the disc protrusion when the patient's low back and leg pain symptoms are severe.

Figure 3.18. Six months later, following complete relief of the low back and leg pain symptoms, the patient shows the same 38% disc protrusion at the L5–S1 level.

Figure 3.19. L4–L5 shows 50% of the sagittal diameter of the vertebral canal to be occupied by the disc protrusion when the low back and leg pain symptoms are severe.

Figure 3.20. Following complete relief of low back and leg pain, the L4–L5 disc seen in Figure 3.19 is reduced to 37.5% of the canal diameter.

Figure 3.21. The L4–L5 disc protrusion occupies 50% of the vertebral canal diameter when this patient has low back and left sciatic pain.

Figure 3.22. The L5–S1 disc protrudes into the vertebral canal to occupy 30% of the canal when the patient has low back and sciatic pain.

Case 2

A 32-year-old white man was involved in an automobile accident. Prior to this he had never had low back or leg pain. Following the accident, he developed low back pain and eventual left lower extremity pain in the distribution of the L5 and S1 dermatomes, especially the S1 dermatome. The patient was seen 11 months following the automobile accident. During that time, he had been treated by his family doctor, who referred him to a neurosurgeon when he failed to respond to drug therapy. This was 10 months following the injury. The neurosurgeon recommended that surgery be performed due to the positive CT scans at the L4–L5 and L5–S1 disc spaces, both of which revealed intervertebral disc protrusions.

Ten months following the injury, and prior to surgery, the patient chose to have a chiropractic consultation for possible conservative treatment of his disc protrusions. Our examination revealed that the leg and back pain were aggravated by Déjérine's triad. The patient complained of night pain. The ranges of motion were limited only by 10° of extension and 10° of left lateral flexion. Straight leg raising produced no low back pain; however, Bechterew's sign or sitting straight leg raising sign did produce low back pain. There was no sign of motor weakness. The deep reflexes at the ankle and knee were +2 bilaterally, and no sensory deficit was seen on pinwheel examination. Circulation of the lower extremities appeared adequate. The original CT scans, revealing the bulging L4–L5 and L5–S1 discs, are shown in Figures 3.21 and 3.22.

Treatment was started 11 months following the initial onset of pain. It consisted of flexion-distraction manipulation of the L4–L5 and L5–S1 disc spaces. Physical therapy was given in the form of positive galvanism and alternating hot and cold applied to these disc areas and to the sciatic nerve distribution on the left lower

extremity. Tetanizing current to the paravertebral muscles was utilized. The patient was placed in a lumbar support and given home exercises consisting of knee-chest exercises and intra-abdominal increasing of pressure. He was told to avoid sitting as well as bending and twisting at the waist. He attended low back wellness school to learn how to perform the proper bending and lifting in daily life without aggravating his back.

After 3 weeks of this care, the patient was relieved of more than 50% of his low back and left leg pain. At that time, he was started on Nautilus exercise regimens, and he rapidly regained muscle strength to the lumbar and abdominal areas. At the beginning of his Nautilus work, he could perform lumbar extension at approximately 10 pounds of pressure, and he eventually was able to lift well in excess of 150 pounds at 15 repetitions, with three sets of repetitions at each session. These sessions were held three times weekly during the course of care. The patient continued the stretching of very tight hamstring muscles, which responded well even though they showed extreme shortness at the beginning of this care. This patient returned to work 5 weeks following the onset of treatment and has worked ever since. He was gradually weaned from his lumbar support and was told to wear it only in stressful situations that necessitated heavy lifting or repetitive bending or twisting. During the course of his healing, he had slight discomfort at the end of a work day, but this was relieved by rest and performing his exercises.

Figures 3.23 and 3.24 are repeat CT scans performed 4 months following the original CT scan and 3 months following the institution of our conservative flexion-distraction manipulation. By measuring the disc protrusion percentage occupying the vertebral canal, we determined that no change had occurred in the size of the disc bulge, although the patient's symptoms had been totally relieved. Therefore, we note that the patient does maintain disc herniations at the L4–L5 and L5–S1 levels; however, he is totally asymptomatic. As has been discussed elsewhere in this chapter, we know that fully one third of people who have never had low back or leg pain will reveal such disc protrusions.

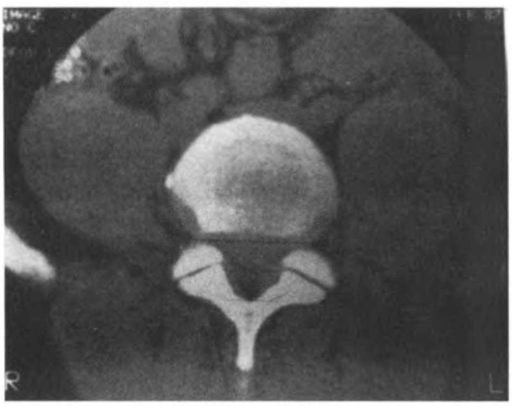

Figure 3.23. Following total relief of both low back and leg pain, computed tomography scan of this patient still shows 50% of the L4–L5 vertebral canal to be occupied by the disc protrusion.

Figure 3.24. Likewise, the L5–S1 disc still bulges into the canal to occupy the same 30% of its diameter when the patient is asymptomatic.

Discussion of Cases 1 and 2

Here we have seen two patients with large disc protrusions—true sciatic radiculopathy—who obtained complete relief of symptoms under treatment, although a large disc protrusion was still present. Certainly, some degree of tightness was created within the vertebral canal by the presence of this foreign element of disc material, but only when a certain degree of compression was reached were symptoms produced.

To further discuss the reduction of disc protrusion by conservative treatment, the cause of nerve root irritation, and its effective relief by conservative care, I published a paper in the *Journal of Manipulative and Physiological Therapeutics*. At this time, excerpts from that paper will be presented to enlighten the reader on the conservative reduction of disc protrusions and how disc protrusion can be determined from diagnostic imaging.

A HYPOTHESIS INTRODUCING A NEW CALCULATION FOR DISCAL REDUCTION: EMPHASIS ON STENOTIC FACTORS AND MANIPULATIVE TREATMENT[a]

Measurement of a Disc Protrusion by CT Scan

We offer a technique to measure the disc protrusion size and to evaluate change in size of the bulge. These measurements were then correlated to the patient's subjective and clinical objective findings.

The technique involves obtaining three consecutive, parallel 2-mm cuts through the disc with the gantry angulation set to obtain axial scans in the plane of the disc. Of course, perpendicular sections to the rostrocaudal axis may be best in some cases. In this particular case, the same three cuts were made on each of three dates: January 1985, when the pain led to hospitalization and CT; June 1985, when the pain worsened; and August 1985, when the pain was absent. The first measurement (*A*) is from the posterior vertebral body to the most posterior aspect of the disc bulge (Figs. 3.25 and 3.26). The second measurement (*B*) is from the posterior edge of the vertebral body to the posterior spinal canal where the laminae join with the spinous process (Figs. 3.25 and 3.26). These two measurements are used to form a percentage, $(A/B) \times 100$ (Table 3.1). The three disc-bulge-to-spinal-canal percentages obtained on

[a]Modified from JM Cox, DD Aspegren: A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987;10(6):287–294, copyright, National College of Chiropractic, 1987.

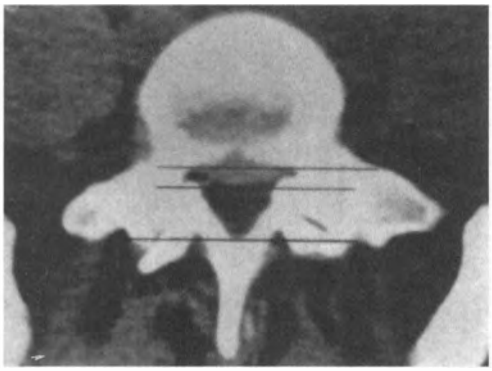

Figure 3.25. Computed tomography scan showing the line drawn along the posterior vertebral body, along the posterior disc bulge, and at the junction of the spinous process and laminae (posterior vertebral canal border). (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987;10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

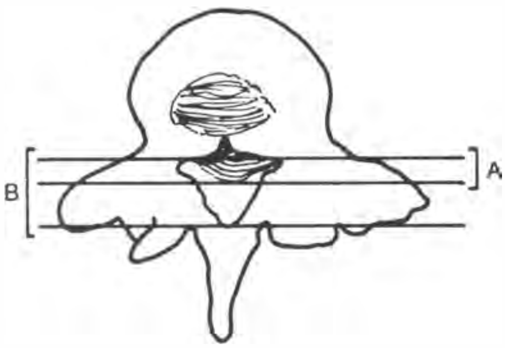

Figure 3.26. Schematic showing *A* as the disc bulge measured in millimeters and *B* as the sagittal vertebral canal diameter in millimeters. *A/B*, percentage of vertebral canal occupied by the disc protrusion. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987;10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

each of the three CTs (Figs. 3.27–3.38) were then averaged for each date (Table 3.1). The sagittal diameter of the spinal canal may be measured on CT with cursors placed on the posterior surface of the vertebral body and the anterior surface of the lamina.

In January 1985, the three percentages averaged 40.6%. The June 1985 slices, done prior to the initiation of therapy, had increased to 48.0%, whereas in August 1985, after therapy and relief of symptoms, a marked reduction in the average disc-canal percentage was demonstrated at 34%.

In our case presentation, the patient had a sagittal L5 vertebral canal of 18 mm with the L5 body being 38 mm. This was a 2:1 ratio (85, 86) of body to canal, although the canal was well above accepted stenotic levels of 12 mm (85, 87–91). Our patient did not have vertebral canal stenosis by Eisenstein's measurement (85, 91–96). Some facet arthrosis was present, but not hypertrophic lateral recess changes. We did have disc protrusion dimensions as reported in Figures 3.27–3.38. We saw that a 14% reduction in disc protrusion size gave complete relief.

We know that the disc protrusion was far from complete, and that the size of the canal and dural sac did not change nor did the facet hypertrophy reduce. A conclusion is that the disc reduction lowered the nerve root pressure below threshold pressure for pain production.

The disc tissue bulge is importantly measured by this disc-canal percentage, which can give an idea of disc increase, decrease, or maintenance of size. Other plain film measurements for stenosis of the bony canal are well documented for accuracy (85–90, 93, 97–100). No one has offered measurements of

Table 3.1

Disc Protrusion Measurements

Gantry Angulation	January 9	June 5	August 23
3rd angled gantry	$\frac{2.1}{5} = 42\%$	$\frac{5}{10} = 50\%$	$\frac{3.1}{9} = 34\%$
2nd angled gantry	$\frac{1.5}{5} = 30\%$	$\frac{4}{8} = 50\%$	$\frac{2.5}{7} = 36\%$
1st angled gantry	$\frac{2}{4} = 50\%$	$\frac{4}{9} = 44\%$	$\frac{2}{6} = 33\%$
Average	40.6%	48%	34%

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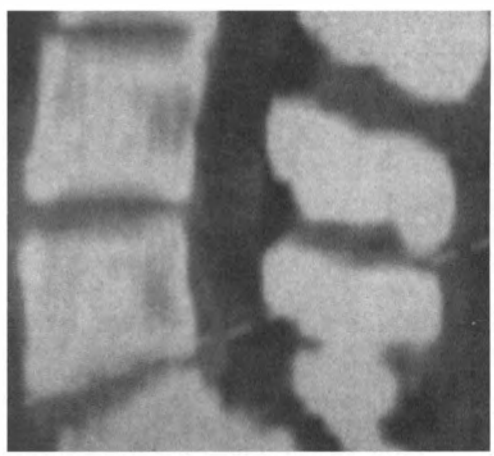

Figure 3.27. Third angled gantry parallel to the L5–S1 disc space. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987;10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

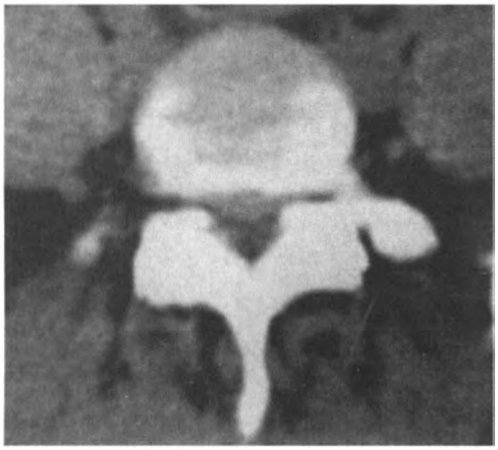

Figure 3.28. January 9 disc bulge at L5–S1 from Figure 3.27 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

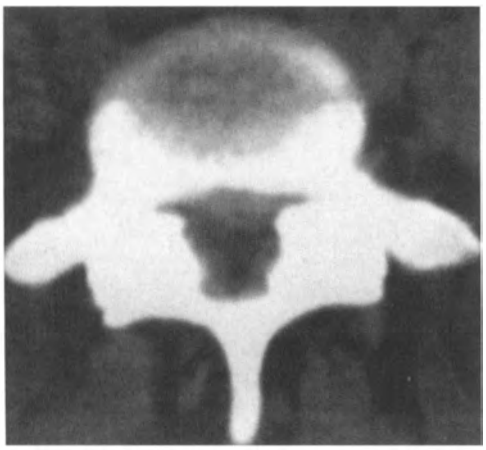

Figure 3.29. June 5 disc bulge at L5–S1 from Figure 3.27 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987;10(6): 287–294. Copyright, the National College of Chiropractic, 1987.)

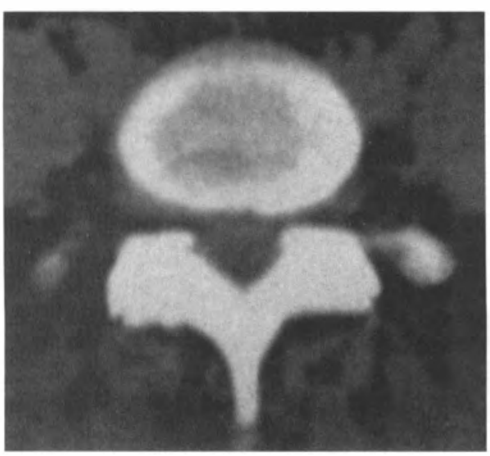

Figure 3.30. August 23 disc bulge at L5–S1 from Figure 3.27 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

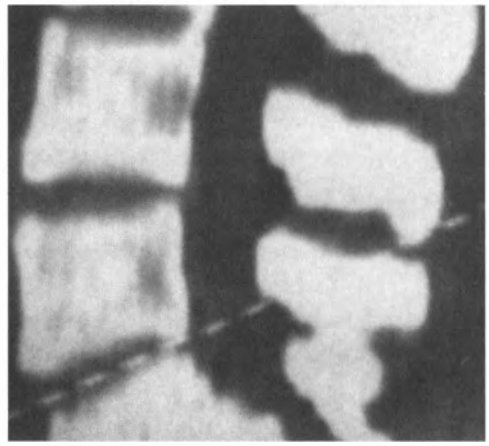

Figure 3.31. Second angled gantry parallel to the L5–S1 disc space. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

Figure 3.32. January 9 disc bulge at L5–S1 from Figure 3.31 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

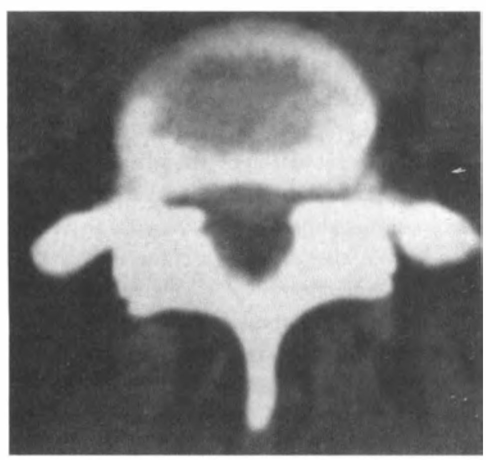

Figure 3.33. June 5 disc bulge at L5–S1 from Figure 3.31 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987;10(6): 287–294. Copyright, the National College of Chiropractic, 1987.)

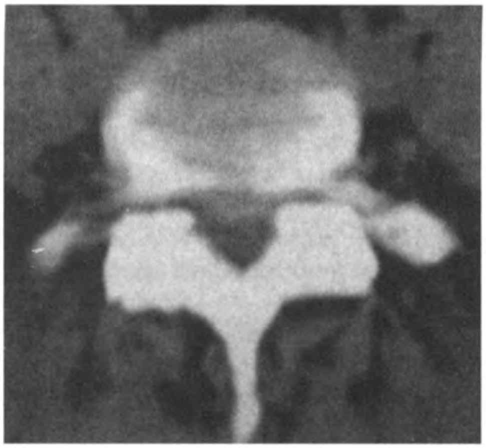

Figure 3.34. August 23 disc bulge at L5–S1 from Figure 3.31 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

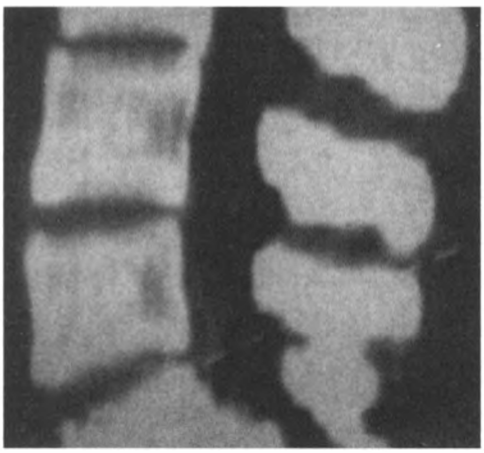

Figure 3.35. First angled gantry parallel to the L5–S1 disc space. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

disc bulge size to monitor patient treatment progress. With the acceptance of mensuration procedures as outlined for stenosis of the canal, our CT measuring system is an extension of such methods.

Need for measurement systems for stenosis has recently been shown by the work of Schonstrom et al. (101, 102) who introduced a new measurement for the transverse area of the dural sac on CT scan. They believed that bony measurements alone did not reliably identify patients with spinal stenosis, the dural sac transverse area being the most accurate method of identifying stenosis, with the critical size for the dural sac less than 100 mm. Further, they found the most common causes of spinal stenosis to be intervertebral disc and ligamentum flavum soft tissue encroachment as well as facet degeneration and hypertrophic changes (101). By careful manometric monitoring of highly pressure-sensitive catheters in the dural sac of seven spines removed at autopsy, Schonstrom et al. (102) found that circumferential restricting of the transverse area of the intact cauda equina at 60 to 80 mm caused a build-up of pressure in the dural sac. Once that critical size was reached, even a minimal further reduction of the area caused a distinct pressure increase among the nerve roots.

The dural sac can tolerate a degree of compression above which additional pressure increases symptoms. The compression of the cauda equina was most commonly due to intervertebral disc protrusion or ligamentum flavum hypertrophy (101). We can correlate our disc canal percentage reduction with relief of pain as possibly lowering the dural sac and nerve root pressure.

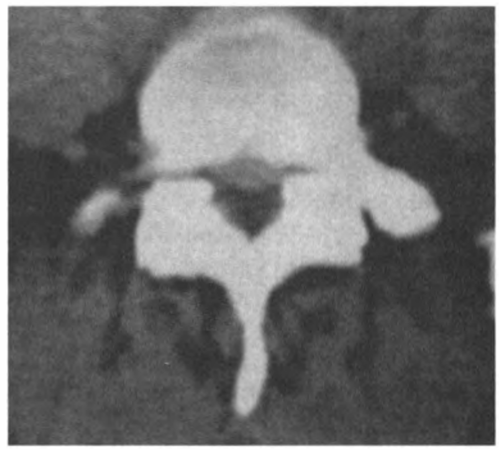

Figure 3.36. January 9 disc bulge at L5–S1 from Figure 3.35 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

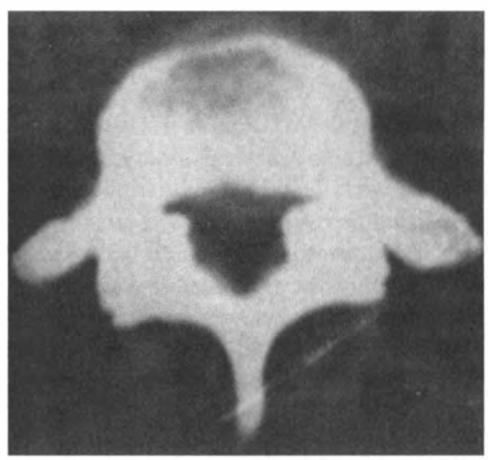

Figure 3.37. June 5 disc bulge at L5–S1 from Figure 3.35 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

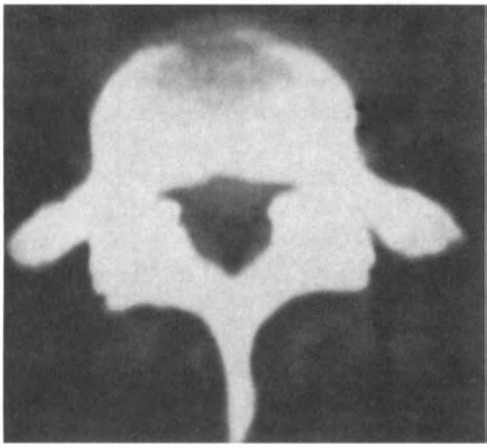

Figure 3.38. August 23 disc bulge at L5–S1 from Figure 3.35 gantry. (Reprinted with permission from Cox JM, Aspegren DD. A hypothesis introducing a new calculation for discal reduction: emphasis on stenotic factors and manipulative treatment. J Manipulative Physiol Ther 1987; 10(6):287–294. Copyright, the National College of Chiropractic, 1987.)

Documenting disc bulge reduction by the disc canal percentage is one means of monitoring the stenotic effect of disc lesions. We have heard and read that the disc bulge remained following a given treatment. Yet not only total reduction but also partial reduction can bring pain relief.

Conclusion

Recognition of the need for investigation of disc herniation reduction following conservative care is called for. Teplick and Haskin (103) discuss 11 cases of spontaneous regression of herniated lumbar disc on CT scan and call for further investigation of conservative treatment effects on disc herniation. Many others (104–122) have shown reduction of disc herniation on myelography, epidurography, or CT scan, or clinical relief when flexion-distraction manipulation, bracing in flexion, and hanging or upright gravity reduction systems are applied as therapy. Also, Naylor et al. (123), Gertzbein (124), Rydevik et al. (7), Elves et al. (125), and Eyre (126) inculpate the chemical irritations (radiculitis) of the nerve by discal biodegradation products as much as the mechanical irritation. Called the "autoimmune mechanism involved with the inflammatory tissue reaction" seen around degenerative discs, this results in intraneural edema and impaired intraneural microcirculation leading to functional changes of motor and sensory deficits.

This chapter offers a CT measuring system to determine the percentage of the vertebral canal occupied by a disc bulge; changes in size of the disc herniation can be evaluated later by repeating the identical CT views.

Stenosis is the problem within the vertebral canal leading to nerve root compression. Controversy exists over what is the most important factor in stenosis—the bony vertebral canal size or the dural sac area. Regardless, soft tissue stenosis by intervertebral disc protrusion, ligamentum flavum thickening, or facet degenerative change is involved, with further narrowing of the vertebral canal or intervertebral foramen and the resultant pressure increase in the dural sac or nerve roots. Depending on the degree of developmental stenosis present, the amount of acquired stenosis becomes important. The person with pre-existing bony stenosis or a large dural sac may develop marked nerve root compression with minimal disc or soft tissue lesion, whereas someone with a large bony canal and small dural sac may have minimal or no sign of symptoms of nerve root compression when such acquired factors appear.

A case was presented with a 14% reduction of a disc protrusion following chiropractic manipulation as measured on CT scans before and after care. Less than total disc herniation reduction resulted in total relief of sciatica in this patient.

Perhaps patients can tolerate a degree of nerve root compression by soft tissue encroachment, depending on the amount of developmental stenosis of the vertebral canal or the size of the dural sac; but if the pressure reaches sufficient levels, symptoms appear. Study in this area is important and it is progressing.

WHAT HAPPENS WHEN A NERVE OR NERVE ROOT IS EXPOSED TO A HERNIATED DISC OR A NARROWING OF THE SPINAL CANAL?

The answer to this question is not known to the complete satisfaction of most physicians. Exploration of this question is exciting.

Spinal Nerve Root Changes Under Compression—A Chiropractic Paradigm of Nerve Root Compression Pathophysiology

Chronic compressive spinal nerve root pathophysiologic changes were assessed in the lumbar spine of the adult dog. At 1 month, thickening was seen of the dura mater and arachnoid membrane around the affected nerve root corresponding to the alteration of the blood-nerve barrier in the nerve root. After 3 months, large myelinated fibers decreased in number and small newly formed fibers increased in the periphery of the fascicle. At 6 months, endoneurial fibrosus and Wallerian degeneration of nerve fibers became obvious. Compound action potentials and sensory nerve conduction velocity decreased by 3 and 12 months, respectively. Intraradicular edema caused by alteration of the blood-nerve barrier is the most important factor in the nerve root dysfunction caused by chronic compression (127).

Embryologic Determination of Pain Locus

When the dorsal root ganglion is irritated by any of a variety of mechanisms, pain is referred to the various structures innervated by that root. What determines pain distribution?

The musculoskeletal system is derived from the embryonic mesoderm. The paraxial mesoderm condenses to form 42 somites that run the entire length of the embryo, located adjacent to the neural tube. The somite then differentiates to produce a ventromedial portion (sclerotome) and a dorsolateral portion (dermomyotome) (128).

The vertebral bodies originate from the sclerotomes. An intervertebral disk develops between each vertebral body. The sinuvertebral nerve sends fibers to the intervertebral disk, posterior longitudinal ligament, anterior dura, and periosteum. Afferent autonomic sympathetic branches from the paraspinal ganglion and from the sympathetic chain also develop connections to the sinuvertebral nerve.

How is pain experienced (perceived) when these structures receive noxious stimuli in the adult? These fibers can be directly stimulated, as when a penetrating object comes in contact with the periosteum. Such stimulation results in a sensation that is dull and aching in quality, which can be associated with such constitutional manifestations of nausea, vomiting, sweating, and vasoconstriction, resulting in a "sickening feeling."

Myotomes form the muscle groups of the lower extremities. The developing anterior primary ramus enters the myotome, supplying the innervation to that segment.

Differentiation of Myotomal, Sclerotomal, and Dermatomal Pain

What differentiates pain experienced by the sclerotomal structures from that derived from the myotomal structure? Both result in a deep, dull, aching sensation. Myotomal pain, in contrast, can be well localized, as in the acutely tender motor point, and it can be elicited by direct pressure.

The mesoderm overlying the myotomes also receives sensory innervation from various peripheral nerves, giving rise to the dermatomes. These dermatomes lie directly beneath the skin and are formed by the sensory afferents located within the subcutaneous tissue and the dermis of the skin.

If a nerve root is compressed, numbness and paresthesias are experienced. However, when the root is inflamed, pain is referred to the portion of the limb innervated by that segment. Patients can present with severe leg pain in a "dermatome" pattern, negative tension signs, and little radiographic evidence of nerve compression (128).

Corporotransverse and Lumbosacral Ligament Entrapment of Nerve Roots

The corporotransverse ligament of 34 cadavers was found attached to the body and transverse process of the same vertebra. The ligament may entrap the exiting nerve root below it in rotary subluxation or in complete disk space loss. The lumbosacral ligament extends from the transverse process of L5 and the L5–S1 disc to the sacral ala, forming the roof of the lumbosacral tunnel through which the L5 spinal nerve passes. This may be the site of extraforaminal entrapment if lateral disc herniations, osteophytes or tumor metastasis are also present. The nerve suspensory ligament attaches to the nerve sheath and to the disk, and it is felt to be significant as a vehicle for mechanoreception (129).

Ligamentous Nerve Root Fixation in the Vertebral Canal

The anatomy of 54 pairs of lumbosacral nerve roots was described in nine fresh adult cadaver specimens, with particular attention given to the fixation of the nerve roots to surrounding skeletal and ligamentous structures in the lumbar spine. Dural ligaments were identified fixing the dura and nerve roots at their exit from the main dural sac to the posterior longitudinal ligament and vertebral body periosteum proximal to the intervertebral disc. Distal fixation occurs at the intervertebral foramen where the epineural sheath of the spinal nerve is attached. The overall arrangement is one that tends to hold the existing nerve root anteriorly in the spinal nerve (130).

Mechanical analysis of this anatomic arrangement explains how pressure can be applied to the extrathecal nerve root by a disc protrusion without compression of the nerve root against the posterior elements. The possible role of the dural ligaments in the pathogenesis of the sciatica syndrome will be discussed.

The extrathecal intraspinal lumbar nerve root is relatively fixed in the spinal canal. Because of this fixation, the extrathecal nerve root cannot easily slip away from a disc protrusion, whereas the nerve root lying freely in the thecal sac can.

Trolard, in 1893, described and illustrated a "ligamentum sacral anterius durae matrin" as an anterior midline series of bands that fastened the dura to the posterior longitudinal ligament in the lower lumbar and sacral regions (130). Hofmann, in 1898, more extensively described several dural ligaments, including a "ligamenta anteriora dura matris" similar to Trolard's earlier description (130). This anatomic arrangement means that traction forces applied to the lumbosacral nerves are resisted by the intervertebral foraminal attachments and the dura, in effect insulating the intrathecal nerve roots from the traction forces. A new finding is an additional attachment by a ligamentous band running from the sheath of the extrathecal foramen. This ligamentous attachment may provide additional fixation of the nerve root to the spine distal to the intervertebral disc.

We are therefore suggesting that when a contact force is present on the nerve root, tension forces are developed, with the forces shared by the dural ligaments just proximal to the disc and the well-known foraminal attachments distal to the disc. Thus, traction forces are exerted on the attachments of the dural ligaments (the posterior longitudinal ligament and vertebral periosteum) and on the pedicular periosteum and connective tissue at the foraminal exit of the spinal nerve. Thus, a disc protrusion is a necessary but not sufficient condition for a contact force to be exerted on the nerve root. The pressure distribution in the nerve root as a result of this contact force, which would determine the pattern of vascular compromise of the nerve root, would depend on the contact area of force application, the tissue properties, and the particular geometric configurations involved. Simple mechanical considerations also suggest that the dural ligaments are situated such that pressure exerted by the disc on the nerve root can be transmitted via these ligaments to the posterior longitudinal ligament and vertebral periosteum. These very structures have been shown previously to produce a characteristic component of sciatic pain when subjected to traction forces (130).

Furcal Nerve

Attention must be paid to the furcal nerve when analyzing lumbosacral radicular symptoms, especially when neurologic findings are atypical and the responsible level cannot be assessed (131). An anatomic and clinical study (131) of the furcal nerve showed the following: the furcal nerve was found in all dissections, and it arises at the L4 root level in most dissections (93%); the furcal nerve has its own anterior and posterior root fibers and its own dorsal nerve root ganglion. This proves that the furcal nerve is an independent nerve root. Neurologic symptoms suggestive of two roots being involved are frequently due to furcal nerve compression.

Neurologic symptoms implying involvement of two roots may be due to four causes. First, two roots may be compressed by a single lesion. Second, two lesions may be present. Third, an anomaly of root emergence may be present with two nerve roots emerging through the same foramen. Finally, the furcal nerve may be involved. When a single nerve root block produces motor weakness and sensory deficits in two nerve root areas, and when the other three possibilities are not demonstrated by the myelographic findings, the furcal nerve should be examined (131).

Fibrous Membrane Anterior to Posterior Longitudinal Ligament

A fibrous membrane lies anterior to and attaches to the posterior longitudinal ligament. This membrane has about one fourth the toughness of the dura, and it is made up largely of fibrous tissue. The veins of Batson lie on its dorsal surface, piercing it, and going ventral to this membrane and enter the vertebral body. Batson's plexus crosses the disc space. Hofmann's ligament anterior to the dura attaches the dura to the posterior longitudinal ligament. The posterior longitudinal ligament (PLL) is tough and strong and seldom ruptures. The anulus frequently ruptures with subligamentous nuclear herniations beneath. No periosteum is found inside the vertebral canal (132).

New Blood Vessels in Extruded Disc Material

Extruded disc tissue develops new blood vessels, which may act as an irritant or help dehydrate disc mass (133) (Fig. 3.39).

Lateral Discs Leak Plasma Protein from Nerve Root into CSF

In one study, 143 patients were evaluated by myelography with regard to involvement of the dural sac and the nerve root. A medial disc herniation group (20 patients) with evidence of dural sac impingement was compared with a lateral disc herniation group (63 patients) and an extreme lateral group (9 patients) whose condition primarily affected the nerve root. The remaining 51 patients comprised a mixed group with involvement of both the dural sac and the nerve root. In the mean cerebrospinal fluid:serum albumin ratio, cerebrospinal fluid total proteins showed a significantly increasing trend from the medial through the lateral to the extreme lateral groups. Patients with lateral lumbar disc herniations more often showed neurologic deficits. These results indicate that the elevated cerebrospinal fluid total protein found in the patients with sciatica is caused by leaking of plasma proteins primarily from the nerve root into the cerebrospinal fluid (134).

Two Theories On Referred Pain

Two current theories of referred pain are proposed: convergence-facilitation and convergence-projection.

Convergence-facilitation finds continuous afferent impulses are normally coming in from cutaneous receptors but are insufficient to excite the spinothalamic tract (STT) cell bodies. Another nociceptive impulse from another afferent fiber (e.g., an anular tear stimulating the sinuvertebral nerve) synapses on the same STT cell, which then facilitates excitation that results in the referral of pain to the region of cutaneous sensation.

The convergence-projection theory of referred pain finds afferent axons from two different regions synapse on the same STT cells that receive their primary input from other structures. For example, a primary STT cell receives dermatomal

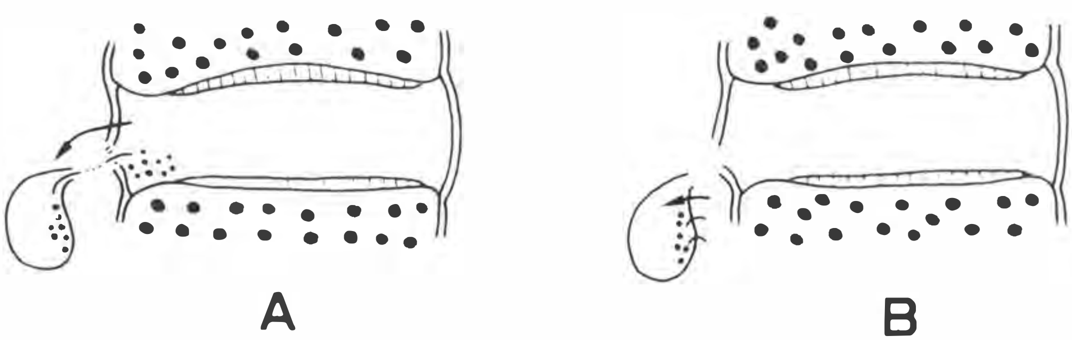

Figure 3.39. Origin of capillaries in extruded tissues of prolapse type of herniation. **A.** Capillaries extruded with intervertebral disc tissue. **B.** Capillaries newly formed after extrusion from intervertebral disc. (Reprinted with permission from Yasuma T, Arai K, Yamauchi Y. The histology of lumbar intervertebral disc herniation: the significance of small blood vessels in the extruded tissue. Spine 1993; 18(13):1761–1765. Copyright 1993, Lippincott-Raven.)

information from cutaneous receptors, but also receives sensory input from the sclerotomal (as from the anulus) and myotomal component. Once stimulated, the STT cell projects the information to the thalamus and sensory cortex through the anterolateral tract, and this pain stimulus is perceived as arising from one structure. Experimental evidence indicates that many STT cells in the spinal cord receive muscle input in addition to cutaneous input (128).

THE SPINAL CORD: ACTIVE PROCESSOR, NOT PASSIVE TRANSMITTER OF IMPULSES

The long accepted basic notion has been that the spinal cord is fundamentally a passive transmitter of information to and from the brain and the body. It is now becoming increasingly evident, however, that the spinal cord is anything but a passive set of neurons that simply receive and transmit sensations from the body to the brain and messages from the brain to the body through reflex systems. Instead, a complex group of several processes can occur in the pathways of the spinal cord to alter the transmission of information, and these alterations can vary from short to relatively permanent changes in neural characteristics (135).

Nociceptor Activity Is Six Times Greater in Inflamed Joints Than in Normal Joints

A model of arthritis in the cat knee joint shows that the inflammation leads to remarkable changes in the characteristics of the nociceptive receptors in the inflamed area. In the normal joint, approximately 400 nociceptors are activated by even severe stimuli to the joint. When inflammation is induced in the joint, the number of nociceptors activated by the same stimuli increases dramatically, often showing a sixfold or greater increase (135).

Fixation of Spinal Cord Activity

Fixation is a form of long-term alteration of spinal excitability that was first studied by DiGiorgio in 1929. It was first produced in an anesthetized mammalian preparation in which a lesion of one or more cerebellar nuclei resulted in a hindlimb flexion. When the hind limb was left in the flexed position for 3 to 4 hours, subsequent transection of the spinal cord resulted not in the expected flaccid paralysis of the limb, but in a retention of an active flexion. This unexpected retention of flexion was termed "fixation" due to the apparent "setting in" or "fixating" of the activity within the spinal cord. The fixation apparently created a spontaneously active focus of spinal neural activity that maintained the leg muscle contraction. The minimal time (fixation time) necessary for fixation to be established in the rat cord was about 45 minutes between introducing the cerebellar lesion and spinal transection.

In the early 1980s, a series of studies examining the fixation phenomenon found that retention of leg flexion could be induced not only by a cerebellar lesion, but by direct electrical stimulation of leg skin. The stimulation produced a tight flexion that lasted after the stimulus was terminated. This result could be shown in rats that had been spinalized prior to the stimulation, as well as in intact animals (135).

A long-lasting increase in the excitability of the spinal reflex showed that the alterations being observed were indeed within the spinal cord not due to sensory alterations or muscle contractures. The amount of fixation can be influenced by factors such as physical exertion and stress (fixation increases with stress), and by prior stimulation to the limb (decreasing with less intense prior stimulation and not with intense prior stimulation).

Reflex changes of fixation can be seen with as few as 20 minutes of stimulation when the stimulus is sufficiently strong. Thus, fixation depends not only on time, but on stimulus strength.

Relatively short inputs to the spinal cord can produce long-lasting alterations in the excitability of the spinal reflexes that do not depend on higher brain structures and are sufficiently robust to outlast days of intervening activity. The neurons of the spinal cord undergo a massive increase in excitability as a result of the increased afferent inputs. Studies of fixation and inflammation have indicated that abnormal inputs can alter the excitability of spinal reflex circuits for long periods, and perhaps permanently (135).

Double Crush Syndrome Increases Nerve Susceptibility to Symptoms

It is hypothesized that pathologic changes at one point along a nerve render it more vulnerable to injury at other locations. This concept was formalized in 1973 by Upton and McComas who coined the term "double crush syndrome" to describe the hypothesis that proximal compression of the nerve might have lessened its ability to withstand a more distal compression (136). By this mechanism it has been suggested that two lesions, each of which would be asymptomatic by itself, could result in clinical symptoms.

The effects of subacute nerve compression on dog sciatic nerve were studied by producing a compressive injury by way of a 2-cm long calibrated clamp that generated 27.6 mm Hg pressure. The clamps were applied either singly or at two places along the canine sciatic nerve. In those animals with a single compressive site, no "complete blocks" were noted electrophysiologically. Histologic evaluation revealed a loss of large myelinated fibers at the site of nerve compression. In animals with two clamps placed sequentially along the sciatic nerve, 10 of 17 nerves showed complete electrophysiologic nerve conduction block. The loss of myelinated fibers was greater in the group with sequential lesions than in those with single lesions. It was concluded that "loss of nerve function after a double lesion was greater than the sum of the deficits after each separate lesion" (136).

Forty-three patients with carpal tunnel syndrome were compared radiologically with 43 age- and sex-matched control patients. No significant difference in the prevalence of cervical

intervertebral disc degeneration or intraforaminal osteophyte protrusion was noted. The carpal tunnel syndrome patients had a significantly higher incidence of lateral humeral epicondylitis, and they also tended to have significantly smaller vertebral canals and relatively short intervertebral discs (when vertebral body height was compared) in the midcervical area. It was felt that these findings in the cervical spine may indicate connective tissue changes that could predispose to more distal injury. From the current experimental data, it is safe to say that two experimental lesions along the course of the nerve have greater effects than a single lesion (136). Chiropractors are keenly aware of this concept, as for example in the patient with carpal tunnel compression syndrome and a cervical disc herniation. Sufficient relief of one of the compressions results in the symptoms disappearing.

REFLEX SYMPATHETIC DYSTROPHY (COMPLEX REGIONAL PAIN SYNDROME)

Reflex sympathetic dystrophy (RSD) is a syndrome in which pain affects the extremities, and it is associated with loss of function and autonomic dysfunction. RSD occurs most frequently in the upper extremity, especially the hand, but the lower extremity (knee and ankle) may also be involved (137). A fairly common complaint of unknown origin, RSD is primarily a neurovascular pain complex that most frequently affects the limbs. The syndrome is typically characterized by burning pain, hyperesthesia, swelling, hyperhidrosis, and trophic changes of the involved tissues. These disorders are often misdiagnosed, improperly treated, or both, and patients are subjected to a prolonged and some with permanent disability.

Diagnosis

The four cardinal signs of RSD are pain, swelling, discoloration (redness or pallor), and joint stiffness (137).

Stages

Symptoms of RSD begin with a gradual, insidious onset occurring over a period of days to weeks. Disease progresses in three stages, each typically lasting 6 months. The first or acute stage is present from onset to 3 to 6 months. It is characterized by edema, early cyanosis that progresses to erythema (localized to metacarpal and interphalangeal joints), hyperhidrosis, and osteopenia. Pain is severe.

The second, or subacute, stage follows the first stage, appearing 3 to 6 months after onset and lasting up to 12 months. It is characterized by chronic burning or aching pain, which may be less severe than that seen in the first stage. Motion becomes significantly limited. Periarticular fibrosus and brawny thickening result from chronic edema.

The third, or chronic stage can last for several years. The skin becomes shiny, pale, dry and cool. Progression of stiffness produces fixed joint deformities. Osteopenia becomes severe (137).

Clinical Entities

Five clinically recognizable entities are found in RSD, each with a distinct precipitant and prognosis: minor causalgia (sensory nerve injury); major causalgia (mixed nerve injury); minor traumatic dystrophy (laceration or minor crush); major traumatic dystrophy (fracture or severe trauma); and shoulder-hand syndrome are reported (138).

Pathogenesis

Pathogenesis of RSD remains controversial. Most investigators suggest an abnormality of the central or peripheral autonomic nervous system. Several authors suggest a psychiatric cause or predisposition in patients who are "sympathetic hyper reactors," who are emotionally labile, or who have a dependent personality with a low pain threshold (138).

What Causes RSD?

Reflex sympathetic dystrophy is caused by:

- A traumatic or acquired painful lesion
- An underlying predisposition (diathesis)
- An abnormal autonomic reflex

According to one hypothesis, the abnormal sympathetic reflex associated with RSD produces inappropriate vasoconstriction. This leads to ischemia and pain, triggering the pain reflex cycle. Substance P is the neurotransmitter for noxious stimuli. Increased pain levels of SP may in turn compound the pain and perpetuate a vicious cycle (137).

RSD Now Termed "Complex Regional Pain Syndrome"

Nerve damage and even minor trauma can lead to a disturbance in sympathetic activity that leads to a sustained condition termed a "complex regional pain syndrome," the term that now replaces the term "reflex sympathetic dystrophy." This results in sympathetic dysfunction features of vasomotor and sudomotor changes, abnormalities of hair and nail growth, osteoporosis, and sensory symptoms of spontaneous burning pain, hyperalgesia, and allodynia. The dorsal root ganglion becomes innervated by sympathetic efferent terminals (139).

Treatment

The most commonly used treatment techniques are injections of lidocaine hydrochloride or some other anesthetic agent that would block the free nerve endings. An anesthetic agent is injected into the limb for up to 15 minutes, after which the tourniquet is removed. Paravertebral sympathetic ganglionectomy has proved to be an effective procedure with success rates of up to 87% (140).

When working with the upper extremity it is the stellate ganglion that is usually the target of treatment. Along with pain

relief from the stellate block, physical therapy using cool packs and active range of motion exercises is often recommended.

One female patient was treated by chiropractic adjustments and electroacupuncture in lieu of more invasive allopathic treatments such as stellate ganglion blocks. The successful conclusion of this case suggests that RSD may present one area in which allopathic and chiropractic physicians could collaborate in their pain management efforts (140).

Physical Therapy

Physical therapy is the bedrock of RSD management. Massage, desensitization, and gentle active motion exercises are combined with splinting to reduce joint stiffness and pain. Elevation and compression gloves are used to reduce swelling (137). The goal of physical therapy is to counteract the clinical changes seen. Hot/cold treatments, massage, and transcutaneous nerve stimulation are aimed at desensitization. Painful passive range of motion should be avoided as this may exacerbate the painful cycle (138).

Other Treatments

The medical treatment of RSD is controversial. Vasodilators should be used if vasospasm is present. The use of beta-blockers such as propranolol has been advocated by some. Tranquilizers may help decrease anxiety. The efficacy of nonsteroidal anti-inflammatory drugs and steroids has yet to be proved. Somatic nerve blockade may be helpful for well-localized lesions. Sympathetic blockade is a useful diagnostic and therapeutic tool. The middle and lower stellate ganglia are blocked in RSD of the upper extremity, whereas the sympathetic chain at the level of L2 and L3 is blocked in RSD of the lower extremity (138). Bupivacaine (0.25%) is the blocking agent (137).

Adjustments Seem To Alter Spinal Fixation Input

Adjustment therapies to reduce motion restrictions, increase proper fluid infusion, and decrease nociceptive inputs to the spinal cord seem to be effective in decreasing the hyperexcitable central state that leads to further alterations in spinal function (135).

Conservative treatment is not as encouraging as indicated in the literature. Nine of 10 patients contacted more than 5 years after diagnosis reported a worsening of symptoms (56%) and that their condition negatively affected their activities of daily living (78%). Of those who were employed prior to diagnosis, 67% reported a job change or unemployment directly related to the disease (138).

NERVE REGENERATION FACTORS

Time

Lumbar nerve roots regenerate as a function of time. Wistar rats were studied and found that following compression of the L5, L6, S1, and S2 nerve roots with a force of 1 N for 1 hour, and then followed for 12 weeks to study the histomorphometric and biochemical changes at autopsy, the sciatic nerve showed significant decrease in myelination and a relative increase in small nerve fibers. The collagen content in the nerve distal to the compression increased. Regeneration of the damaged nerve roots took place in 12 weeks with long-term changes in myelination and increase of collagen content in the dependent nerve areas (141).

Sectioning of the L4 nerve in 9-week-old male rats showed that partial denervation produced nearly total denervation with significant, but incomplete recovery of muscle weight and tension with recovery occurring between the second and eight week post-L4 nerve sectioning (142).

Dorsiflexion Weakness Improves After Surgery

The incidence of pronounced extensor hallucis longus paresis in lumbar nerve root compression varied between 5 to 11%. Recovery after surgery was common in disc herniation and lateral spinal stenosis but did not occur in central stenosis. Complete recovery was most common in disc herniation, and recovery occurred mainly in the first 4 months after surgery (143).

Sciatic Neuropathy Heal Time Is Up to 3 Years

Good but incomplete recovery occurs over 2 to 3 years in most patients with sciatic neuropathy, particularly in those without severe motor axonal loss.

Although only about 10% of the patients have moderate recovery of motor function within the first 6 months, 75% do by 3 years. Slow recovery is expected, because significant axonal loss is frequently present and because reinnervation of muscles below the knee is delayed because of the long distance between the injury site and the target muscles. Patients should be cautioned about falsely elevated expectations for significant recovery within the first year. Nerve regeneration usually continues for the first 3 years and most patients eventually have functionally significant improvement. The prognosis for moderate or excellent recovery depends on the presence or absence of the extensor digitorum brevis compound muscle action potential and the initial strength of the gastrocnemius and tibialis anterior (144).

Muscle Recruitment Necessary in Denervated Muscle Atrophy

Following lesions of peripheral motor nerves, electrical stimulation of denervated muscle is often recommended. This is to replace the nerve function and elicit the contractile activity of the denervated muscle through the recruitment of the muscle fiber itself. Contraction of denervated muscle induced by electrical stimulation prevents the loss of oxidative enzymes and the atrophy associated with denervation. It is thus important to start treatment as soon as possible, because whatever has been lost through delay cannot be regained. The beneficial effects of electrical stimulation must be associated with the reduction in muscle wasting and the maintenance of the contractile properties of the muscle and not to an effect on nerve regeneration. It is crucial to maintain viable muscle tissue to provide a good target for the regenerating nerve and, thus, reduce the rehabilitation time and increase the chances of complete recovery (145).

PELVIC PAIN AND DISEASE CORRELATED WITH NERVE COMPRESSION

Pelvic Organ Neurophysiology

Pelvic organs are innervated by the pelvic (parasympathetic), hypogastric (sympathetic) and pudendal (somatic) nerves. Stimulation of the pelvic nerve causes contraction of the bladder detrusor muscle whereas hypogastric nerve stimulation induces a weak bladder contraction and a stronger contraction of the bladder neck. The external striated sphincter and the pelvic floor musculature are innervated by somatic fibers from S2 and S3 that traverse the pudendal nerve.

Pelvic regional blood flow shows a fourfold increase in the bladder neck area during neurostimulation. Pudendal nerve stimulation reveals an intraurethral pressure increase with a 3.5 times increase of blood flow in the sphincteric area and in the pelvic floor musculature (146).

Interstitial Cystitis and L5 Nerve Root Compression

Interstitial cystitis is a chronic condition caused by inflammation of the interstitium between the bladder muscle and bladder lining. It is exacerbated by a variety of agents, including certain drugs, hormones, and viruses. Generally, bacteria are not present in the bladder of such patients. Interstitial cystitis is progressive and exhibits a wide range of symptom manifestations. In its early stages, urinary frequency without bacterial infection may be all that is noted. In severe cases, the bladder is ulcerated and scarred. Eventually it may shrink and hold only 1 to 2 ounces of urine. In every case, sensitive tissue is continuously exposed to an acid burn from urine. As a result, it is painful to hold urine but not to urinate (147).

It is postulated that low back problems are a leading cause of urinary tract infections in women. Although this fact has been demonstrated since 1957, it is rarely addressed, and few physicians have studied the relationship in detail.

When evaluated with an MRI, patients with interstitial cystitis are often found to have abnormalities of the fourth and fifth lumbar vertebrae and occasionally lumbarization of the first sacral vertebra. Bladder dysfunction without pelvic pain is seen with compression of the L4 nerve root, whereas frequency and pain before and after voiding are consistently associated with nerve root compression which appears to cause urologic dysfunction (147).

Typically, pelvic pain and bladder symptoms far outweigh any leg or back pain. Some deny they would have sought chiropractic treatment for these complaints. In addition to the MRI, patients are also screened with a uroflow, a noninvasive study that demonstrates the flow pattern during voiding. When the uroflow patterns are correlated with an MRI, certain identifiable patterns are directly related to compression of L4 or L5 nerve roots. For example, the pattern created by abdominal strain to void corresponds with L4 nerve root compression, whereas the pattern created by delayed relaxation of the pelvic floor indicates compression of L5 (147).

Chiropractic Treatment and Results in Interstitial Cystitis

Patients were referred to a chiropractor for flexion-distraction manipulation and myofascial therapy along the thoracic spine, lumbar spine, and buttocks. Daily low back exercises emphasizing strength, coordination, and flexibility and aerobic exercise three times a week (e.g., fast walking, biking, or swimming) were suggested.

Without formal research, positive changes after 4 to 6 weeks of chiropractic treatment were seen. Follow-up uroflow studies and bladder scans have shown that in many cases the patient's uroflow and urine retention normalized.

These results should come as no surprise to chiropractors who have always supported the concept that mechanical disorders of spinal origin could induce organic dysfunction and who have long observed that manipulation results in relief of pain for many patients suffering from chronic back pain. The use of flexion distraction therapy may open new possibilities for the management of interstitial cystitis and dysfunctional bladder patients (147).

Polk (148) reports that 70,000 of the 650,000 hysterectomies performed each year are done because of pelvic pain (149), and 20 to 50% of these patients fail to achieve their goal of pain relief (150). Gillespie et al. (151) researched the possible link between spine-related disorders and pelvic pain and bladder dysfunction and found 73% of 200 women with interstitial cystitis had abnormalities involving the fourth and fifth lumbar vertebrae (152). Polk, a chiropractor, received 50 interstitial cystitis patients at Gillespie's referral for distraction manipulation of the lumbar spine, Kegel exercises, low back exercises, and aerobic exercise. Repeat uroflow was performed after 4 to 6 weeks of this care. Most patients expressed satisfaction with chiropractic care and admitted that they would never have sought it without a urologist's referral (148).

Histories of pelvic problems have included pelvic pain, bladder dysfunction, recurring bladder infections, burning in the pelvis, painful bladder, constant low back, hip and buttock pain, and painful intercourse followed by bladder infection.

Urinary Incontinence Linked with Low Back Pain

A rare association between severe low back pain and urgency incontinence of urine, not explained on the basis of any conventional neurologic or genitourinary pathology, should be recognized and a search made for neurologic mechanisms to explain the phenomenon. Urinary bladder dysfunction is reported in patients with confirmed disc herniations without nerve root compression (153).

One explanation is the production of a parasympathetic discharge stimulated by pain neuropeptides acting directly on the S2, 3, 4 nerve plexus, resulting in detrusor contraction or bladder neck relaxation. The S2, 3, 4 pudendal nerve, on the other

hand, motorizes the external urethral sphincter and mediates sensation from much of the perineum. If it is overridden by the parasympathetic discharge, this could explain the flooding complained of by a few patients, and furthermore explain the reduced perineal sensation found in most patients.

The nature of the relationship between the experience of pain and the reduced sensation in the perineum remains obscure. The patient with chronic low back pain and urinary incontinence in the absence of a cauda equina compression should not be additionally burdened with the pejorative label of "inappropriate" symptomatology. The association may be real even if as yet it is unexplained (153).

Bladder Dysfunction Is Relieved Following Decompressive Laminectomy in the Elderly

Elderly patients with lumbar spinal stenosis often manifest varying degrees of bladder dysfunction, which benefits from lumbar decompressive laminectomy (154).

Testalgia Caused by Thoracolumbar Dysfunction and Relieved by Manipulation

Ten men aged 30 to 55 years suffering from long-term unilateral testalgia revealed a unilateral thoracolumbar dysfunction in all cases. Nine of the patients experienced spasms of the psoas muscle; five men also had dysfunction of the sacroiliac joint on the same side. After a single or repeated manipulation, testalgia completely disappeared. The dysfunction of the thoracolumbar junction was found in all cases (100%), spasm of the psoas muscle on the same side in nine cases (90%), and dysfunction of the sacroiliac joint in the direction of the side of pain in five cases (50%) (155).

Abdominal or Flank Pain May Be Spinal in Origin

Thoracic pain mimicking musculoskeletal disorders is sometimes related to visceral diseases such as gastroduodenal ulcer, pancreatitis, or aortic dissection. Inversely, abdominal or flank pain may be due to rib or spine disorders. Costovertebral arthropathies are anatomically frequent, particularly during ankylosing spondylosis and osteoarthrosis (156).

Interspinous Ligament Irritation Causes Cardiovascular Changes

Wistar rats had the interspinous ligaments stimulated by noxious chemicals, which caused a pronounced elevation of mean arterial pressure and a prolonged depression of sciatic nerve blood flow (157).

Treatment of Pelvic Dysfunction with Flexion-Distraction Manipulation

The mechanically induced pelvic pain and organic dysfunction syndrome (PPOD), which is characterized by various disturbances in pelvic organ function, has been successfully managed by chiropractic distractive decompressive manipulative procedures. Patients who present with symptoms of bladder, bowel, gynecologic, and sexual dysfunction secondary to the impairment of lower sacral nerve root function as a result of a mechanical disorder of the low back were treated (158).

Two Types of PPOD (Table 3.2)

Type I PPOD Patient: These patients present with low back and/or leg pain in addition to pelvic pain and either no changes in pelvic organic function or relatively mild disturbances of bladder, bowel, gynecologic, or sexual function.

Type II PPOD Patient: These patients present predominantly with symptoms of pelvic organic dysfunction. They generally complain of low back and/or leg pain and more severe and widespread symptoms of pelvic pain and pelvic organic dysfunction (Table 3.2) (158, 159).

Table 3.2

Symptoms of Mechanically Induced PPOD

Pelvic Pain	Bladder Dysfunction	Bowel Dysfunction	Gynecologic/Sexual Dysfunction
Inguinal	Frequency	Constipation	Miscarriage
Suprapubic	Urgency	Diarrhea	Vaginal discharge
Para-anal	Dribbling	Excessive flatus	Vaginal spotting
Coccygeal	Incontinence	Anal sphincter spasm	Painful/irregular menstruation
Rectal	Difficulty	Encopresis	Menstrual migraine
	Sluggishness	Mucorrhea	Decreased genital sensitivity
	Retention	Loss of rectal sensory perception	Decrease or loss of orgasm
	Nocturia	Spontaneous bowel discharge	Dyspareunia
	Dysuria		Genital pain/paresthesias
	Infection		Pelvic pain on orgasm
	Enuresis		Deficient (pre)coital lubrication
	Loss of vesical sensory perception		Depressed libido
			Impotence

Chronic Pelvic Pain

Chronic pelvic pain is continuous or episodic pain that persists for at least 6 months and is severe enough to affect a woman's daily functioning and relationships. A 5% risk of developing it exists for the lifetime of a woman. Musculoskeletal causes of it can include coccydynia, disc problems, degenerative joint disease, fibromyositis, hernias, herpes zoster (shingles), low back pain, levator ani syndrome (spasm of the pelvic floor), myofascial pain (trigger points, spasm), nerve entrapment syndromes, osteoporosis (fractures), pain posture, scoliosis/lordosis/kyphosis, and strains/sprains (160).

The chiropractic profession has a historical interest and ability in treating visceral diseases with spinal adjustments intended to reduce nerve root compromise. Validation by studies just cited pave the future study of such nervous system-induced malfunctions as a cause of visceral diseases. This is a thought-provoking end to this neurophysiology chapter.

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Spinal Stenosis

James M. Cox, DC, DACBR

chapter 4

Security is mostly a superstition. It does not exist in nature, nor do the children of men as a whole experience it. Avoiding danger is no safer in the long run than outright exposure. Life is either a daring adventure or nothing.

—Helen Keller

STENOSIS

Stenosis is an abnormal narrowing of the bony or ligamentous structures of the vertebral canal (1). The incidence of lumbar spinal stenosis has been reported to be 50 per 1 million inhabitants annually with 17 patients per 1 million inhabitants operated on for stenosis annually. Neurologic problems are not prevalent and few emergency cases are seen (2).

Classification

I. Congenital
  A. Achondroplasia
  B. Developmental—here the central canal is narrowed in both the sagittal and lateral dimensions (3). Short pedicles and overdeveloped lamina can cause the narrowing.

II. Acquired stenosis (degenerative)
  A. Thickened, irregular laminae (4)
  B. Ligamentum flavum hypertrophy (5)[a]
  C. Soft tissue hypertrophy—from mechanical instability and degenerative disease (6)[a]
  D. Posterior articular joint disease
  E. Trefoil configuration
  F. Intervertebral disc protrusion
  G. Spondylolisthesis—with forward L5 displacement on the sacrum, the fifth lumbar nerve root may be kinked around the lower border of the pedicle or compressed by degenerative changes occurring between the pedicle and the upper sacral border (3)[a]
  H. Posterior intervertebral body plate hypertrophic osteophytes in the foramina
  I. Narrowing of lateral recess by hypertrophic articular processes

III. Iatrogenic stenosis

Iatrogenic stenosis occurs with excessive stress placed on a motion segment above a level of spinal fusion (7).[a] The interspinous ligament and ligamentum flavum become thickened, the spinous process base projects into the canal, and the laminae protrude ventrally. Bone may proliferate under the fused area, with thickening of the laminae and ligamentum flava associated with bulging of the posterior articular process. Disc herniation is common in both sets of circumstances.

Laminectomy and discectomy can also cause progressive deterioration of the intervertebral disc, with consequent migration of the superior articular process and continued degenerative changes. Scar formation at the operative site can contribute to local stenosis (6).

IV. Foraminal (lateral recess) stenosis

Trauma or recurrent inflammation leads to hypertrophy and intrusion of the superior facet into the lateral recess (8).

Figure 4.1 shows the various diameters of the vertebral canal. The lateral recess is bordered anteriorly by the vertebral body posterior surface, posteriorly by the superior articular facet, and laterally by the pedicle, and it opens medially into the vertebral canal.

[a] See case presentations at the end of this chapter, which depict these causes of stenosis.

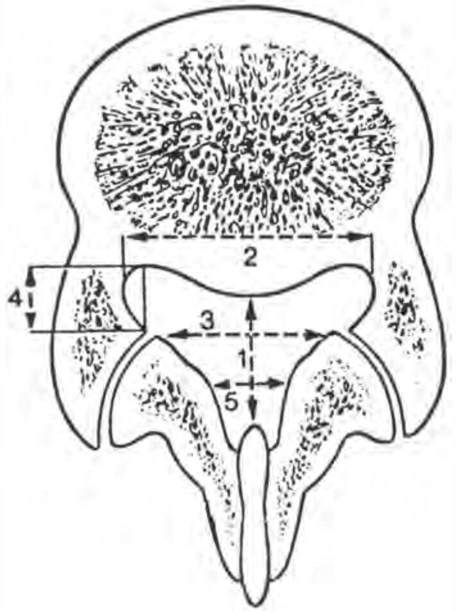

Figure 4.1. Cross-section of vertebral canal at L5 with various diameters. *1*, sagittal diameter of spinal canal; *2*, interpeduncular distance; *3*, interfacet distance; *4*, lateral recess; *5*, interlaminar distance.

From Figure 4.1, it can be seen that the lateral recess can be encroached, or stenosed, by the following:

1. Facet joint hypertrophic degenerative changes, probably best seen in superior articular facet *arthrotic* hypertrophy entrapping a lumbar nerve root coursing through the lateral recess.
2. Posterolateral disc protrusion or prolapse.
3. Ligamentum flavum hypertrophy.
4. Spondylolisthesis (9).
5. Secondary to lumbar fusion bone overgrowth.
6. Degenerative disc disease.

PATHOGENESIS

Traction is produced on neural tissue as the spine rotates, flexes, or extends itself. Normal persons have sufficient room in the canal and lateral recesses for molding and gliding; hence, movement produces no clinical symptoms. However, if the size of the canal is further narrowed by bony or ligamentous proliferations, symptoms appear (10).

Pain can result from direct nerve impingement, but it has been postulated that high-grade obstructions could at least partially block lymphatic and venous channels in the dura or its sleeves. Build-up of cerebrospinal fluid below the obstruction could cause collapse of venous return and produce stagnant anoxia (11). Axon reflexes via the autonomic nervous system have also been postulated to account for the pain (12).

Integration of causative factors cause nerve root irritation, which helps to explain low back and leg pain symptoms. Stenosis may be the most important element in determining symptoms, and their severity, response to treatment, and prognosis. A patient can have a large disc protrusion and also a large-diameter vertebral canal and lateral recess, and, therefore, have no symptoms, whereas the same disc protrusion can cause severe motor and sensory findings in a patient with a stenotic canal. Figure 4.2 demonstrates how the nerve roots lie snugly within the lateral bony recess prior to exiting the intervertebral foramina. The L5 and S1 nerve roots lying within the lateral recesses are more vulnerable to compression from a protruding intervertebral disc than the higher lumbar roots lying within a rounder vertebral foramen (Fig. 4.3).

Figure 4.4 exemplifies how a patent, nonstenotic canal can accommodate a relatively large disc protrusion without creating symptoms, whereas a stenotic lateral recess compresses the nerve root, creating marked pain and motor findings. Therefore, disc protrusion size is not as important as the size of the canal it bulges into.

CAUSES OF NERVE ROOT SYMPTOMS

Large portions of the population have disc protrusion yet have no symptoms. Weisel et al. (13) reported that three neuroradiologists, in a blind study, found 35% of 52 asymptomatic patients to have a herniated nucleus pulposus on computed tomographic (CT) scan. Further, it was pointed out that 24% of normal patients with no history of low back or sciatic pain showed significant abnormalities on myelography. Perhaps the reason for the absence of pain in these "normal" individuals is absence of sufficient pressure on the nerve root by the herniated disc to elicit "pain." Keep in mind that stenosis increases the probability of nerve root compression. A decrease in vertebral canal size by as little as 15% (2 mm) separates persons with and without back pain. Indeed, more than 53% of patients with low back pain may have spinal stenosis (14).

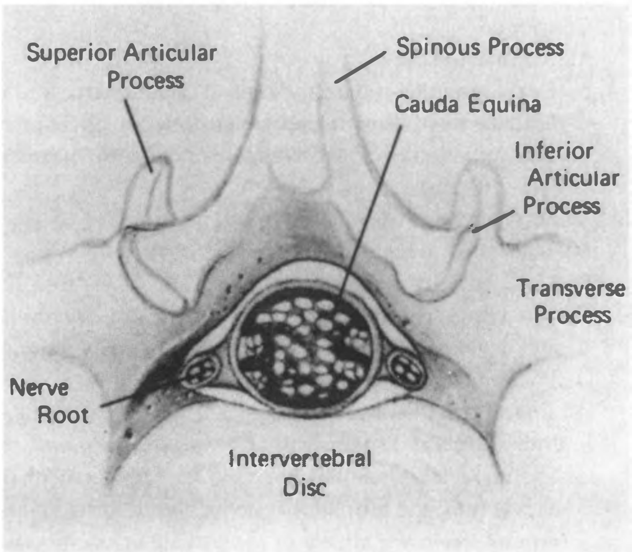

Figure 4.2. Prior to exiting from the intervertebral foramina, the nerve root lies at the lateral-most portion of the vertebral foramen. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:9.)

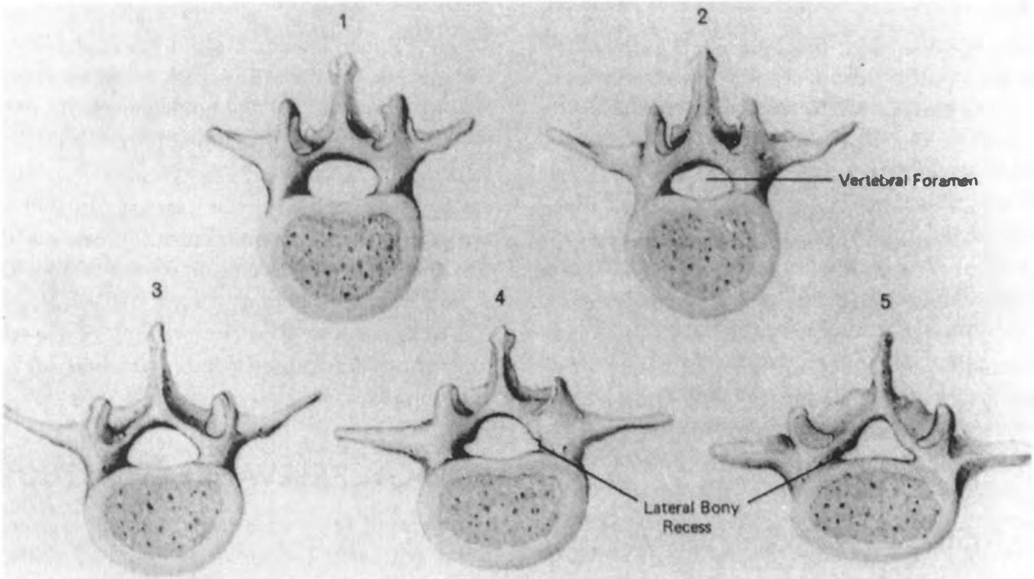

Figure 4.3. The five lumbar vertebrae. Note the *lateral bony recess* formed by the last two vertebrae. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:8.)

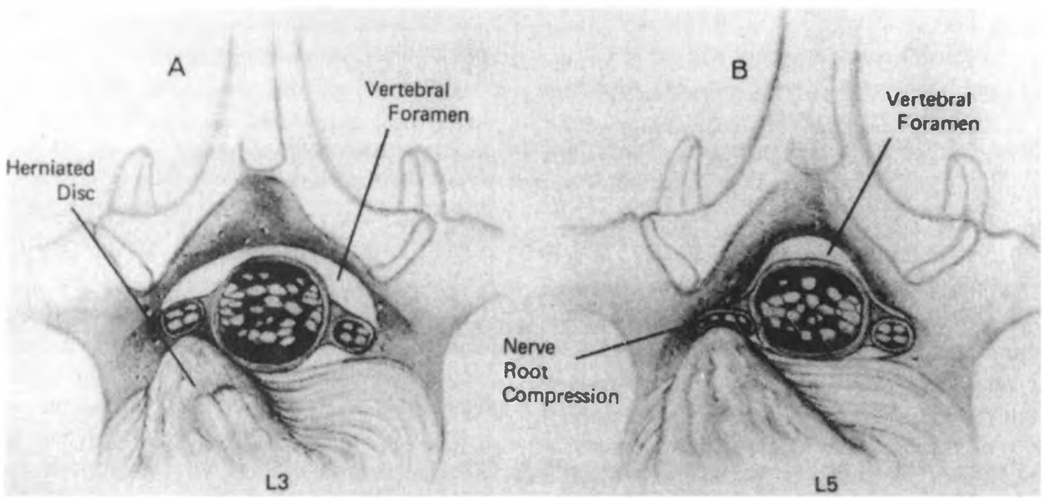

Figure 4.4. **A.** A relatively small intervertebral disc protrusion may not produce significant nerve root compression when the vertebral foramen is oval, and it may permit elevation of the root. **B.** When the nerve root lies within a lateral bony recess, even a small disc protrusion may produce severe root compression. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:9.)

Rydevik et al. (15) showed that the functional changes induced by nerve root compression can be caused by mechanical nerve fiber deformation associated with intervertebral disc herniation and spinal stenosis; also, the changes may be a *consequence of changes in nerve root microcirculation,* leading to ischemia and the formation of intraneural edema. Nerve root compression can, by different neurophysiologic mechanisms, induce motor weakness and altered sensibility or pain. Intraneural edema and demyelination seem to be critical factors for the production of pain in association with nerve root compression. Rydevik et al. compare the jeopardized microcirculation of the nerve to a "closed compartment syndrome" within the foramen.

Although no in vivo measurements of the pressures that act on a human nerve root (e.g., disc herniation) are known either to me or to Rydevik et al. (15), who extrapolate from existing knowledge on the swelling pressure of a nucleus pulposus herniation. They state that the pressure demonstrated on in vitro nucleus pulposus specimens could reach several hundred millimeters of mercury if the specimen was exposed to free fluid

within a confined space. A sequestered fragment in the foramen could be speculated to create high pressure levels in the nerve root, but the validity of the hypothesis needs to be proven. Nerve roots are more susceptible to mechanical deformation than peripheral nerves, considering that the peripheral nerve has a perineurium but nerve roots do not and that the peripheral nerve has well-developed epineural connective tissue where it passes close to bone and joint, whereas the nerve root has a poorly developed epineural lining (15).

A peripheral nerve, at 30 to 50 mm Hg compression, demonstrates change in intraneural blood flow, vascular permeability, and axonal transport (16–19). Rabbit tibial nerve showed these changes at 20 to 30 mm Hg; with complete ischemia at 60 to 80 mm Hg and higher pressures resulting in delayed recovery of intraneural blood flow (15).

PAIN MECHANISMS IN NERVE ROOT COMPRESSION

Compression of normal peripheral nerve or nerve root can induce numbness, but it usually does not cause pain. Experimental investigations of human peripheral nerves, in vivo, have indicated that the numbness induced is a result of ischemia, not mechanical nerve fiber deformation, of the compressed segment. If a nerve root—or a peripheral nerve—is the site of chronic irritation, however, even minor mechanical deformation may induce radiating pain. This has been demonstrated by placing sutures or inflatable catheters around nerve roots at the time of surgery for herniated discs and postoperatively inducing stretching or compression of the nerve root (15).

Stenotic problems can create pressure levels on nerve roots but not sufficient to cause motor or sensory findings. Thus, it seems that a nerve root can tolerate some degree of pressure. Some persons who have never had pain reveal disc protrusion on computed tomography or myelography, yet they may have a large vertebral canal, small dural sac, no ligamentum flavum or facet hypertrophy, and a small disc protrusion. This combination of factors could result in minimal pressure on the nerve root, but not enough to cause symptoms. On the other hand, a person with a small vertebral canal, a large dural sac area, and facet lateral recess hypertrophic stenosis with ligamentum flavum thickening could have severe pain with a moderate or even small disc protrusion causing high compressive forces on the nerve root.

THECAL SAC SIZE IN STENOSIS

The need for a system to measure stenosis has recently been shown by Schonstrom et al. (20, 21) who introduced a new measurement for the transverse area of the dural sac on CT scan. They felt that bony measurements alone did not reliably identify patients with spinal stenosis; the dural sac transverse area is the most accurate method of identifying stenosis, with the critical size for the dural sac below 100 mm. Further, they found the most common causes of spinal stenosis to be intervertebral disc and ligamentum flavum soft tissue encroachment, as well as facet degeneration hypertrophic changes (20). By careful manometric monitoring of highly pressure-sensitive catheters in the dural sac of seven spines removed at autopsy, Schonstrom et al. (21) found that circumferential restriction of the transverse area of the intact cauda equina to 60 to 80 mm caused a build-up of pressure in the dural sac. Once that critical size was reached, even a *minimal* further reduction of the area caused a distinct pressure increase on the nerve roots. These authors note that again we see that the dural sac can tolerate a degree of compression above which pressure increases create symptoms. The compression of the cauda equina was most commonly caused by intervertebral disc protrusion or ligamentum flavum hypertrophy (20).

CLINICAL RELEVANCE OF STENOSIS

A review of the clinical and radiographic records of 214 patients with spinal spondylosis found 63 (29%) were symptomatic with cervical spondylosis, 123 (58%) presented with symptoms of lumbar spondylosis, and 28 (13%) presented with complaints referable to both the cervical and lumbar spondylotic changes. Segmental sagittal diameters of the spinal canals of the symptomatic areas were measured (22). A narrow spinal canal was present in 64% of patients with cervical spondylosis, in 71% with lumbar spondylosis, and in 64% with combined degenerative disease of the cervical and lumbar spine.

In the cervical area, myelopathy will likely occur when the midcervical diameters approach 10 mm. Myelopathy may be predicted with developmental midcervical diameters of 10 to 13 mm. From 13 to 17 mm, patients may be susceptible to symptomatic cervical spondylosis, but few of these will be susceptible to myelopathy. Above 17 mm, patients may be less prone to symptomatic disease.

In the lumbar area, patients with small developmental sagittal diameters seem susceptible to refractory disease and spinal stenosis with *neurogenic claudication* when the canal is narrowed *below 15 mm* radiographically. Patients with canal diameters of 15 to 20 mm comprise a large group of clinically symptomatic patients who may require more surgical treatment. Conversely, when the lumbar sagittal diameters are 20 mm or more radiographically, patients require more spondylotic change for the expression of the same clinical symptoms (22).

Degree of concavity (i.e., the scalloping) on the posterior surface of the lumbar vertebral bodies has been evaluated quantitatively by means of a simple measuring device. Scalloping in the median sagittal plane was found to differ from that in the lateral plane near the pedicular attachments. In the medial plane, an increase in scalloping from L1 to L4 is noted, with a subsequent decrease at L5 (23). Laterally, the concavity deepens from L1 to L5, the values here being larger than those medially at all levels. Scalloping in the lateral sagittal plane, especially at the fourth and fifth lumbar levels, is presumed to be caused mainly by pressure exerted by the spinal nerves. The medial scalloping is presumed to be partially caused by hydrostatic pressure of the cerebrospinal fluid in the dural sac. At the edges of the superior and inferior end plates, this pressure will be

counteracted by the tractional stresses of the fibers of the discal anulus fibrosus, which are inserted at the vertebral surface that constitutes part of the anterior wall of the spinal canal. Therefore, its shape has relevance in cases of spinal stenosis (23).

Figure 4.5 schematically shows how the posterior vertebral surface is modeled by tractive and pressure forces. Figure 4.6 shows myelographic changes induced in the cauda equina by these pressure forces as flexion and extension occur.

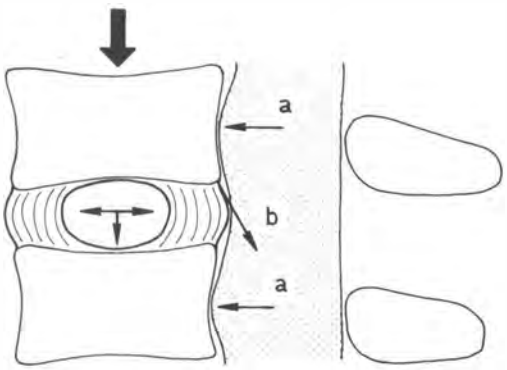

Figure 4.5. Modeling forces on the posterior vertebral surface (*PVS*). The body weight (*thick arrow*) is transmitted to the nucleus pulposus. This gives rise to a tractional force (*b*) mediated by the anulus fibrosus, which counteracts that caused by cerebrospinal fluid pressure (*a*). No such opposing force is seen at the midvertebral level. (Reprinted with permission from Larsen JL. The posterior surface of the lumbar vertebral bodies. Part I. Spine 1985;10(1):55.)

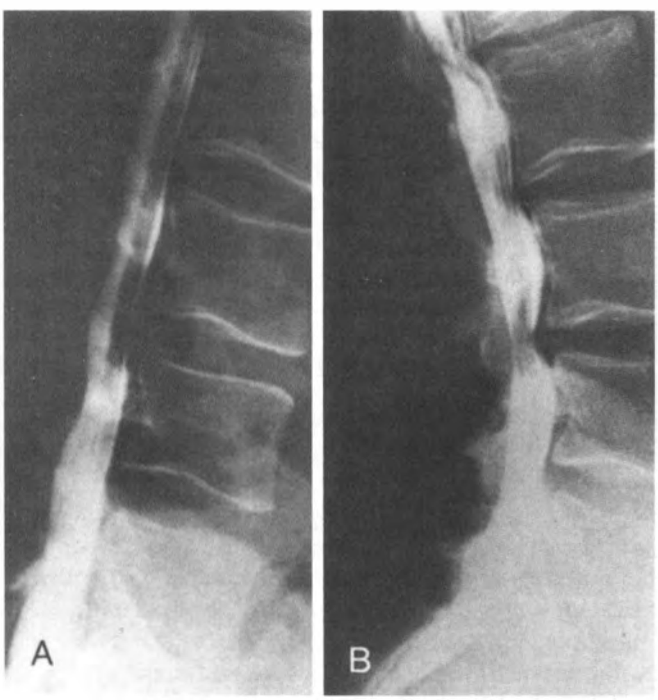

Figure 4.6. **A.** In ventral flexion, the dural sac is closer to the posterior surface of the intervertebral discs. **B.** In extension, it is closer to the central parts of the posterior vertebral service as shown in these lateral myelograms. (Reprinted with permission from Larsen JL. The posterior surface of the lumbar vertebral bodies. Part I. Spine 1985; 10(1):54.)

Herniated lumbar disc or definite sciatica was diagnosed in 16 of 195 men and women who had reported a history of low back pain in a health survey. Measurements relating the size and shape of the lumbar spinal canal were subsequently made from the survey radiographs and compared between various types of back syndrome. Age, body height, body mass index, occupation, and parity of women were controlled as potential confounders using analysis of covariance. Several dimensions of lumbar vertebral canals appeared more shallow in the subjects who had a herniated disc or definite sciatica than in the others. In particular, the interarticular distance of the first sacral vertebra was found to be narrowed in cases of sciatica; the difference of the adjusted distances to the back pain category in men was 30.5 mm versus 35.1 mm ($P = 0.02$), and in women was 23.8 mm versus 30.3 mm ($P = 0.002$), respectively (24). Herniated lumbar intervertebral disc is often symptomless (13).

Measurements of the size and shape of the lumbar spinal canal obtained from survey lumbar radiographs have been shown to be valid as compared with bony specimens from cadavers. The radiologic measurements performed in the present series (13) have proved repeatable, as described in previous reports (25). The results of Hellovaara et al. (25), in general, accord with the hypothesis that a shallow spinal canal contributes to lumbar radiculopathy. This is consistent with the findings of Ramani (26), Porter et al. (27), and Winston et al. (28), except, unlike their data, in this series no significant difference was found in the midsagittal diameter.

Plain films are useful in the diagnosis of lumbar spinal stenosis, contrary to the opinions of some authorities who feel that plain films are of little value. In many cases, clinical presentation and careful analysis of plain films are sufficient to provide an almost certain diagnosis (24).

Root Entrapment Signs

Four criteria are used to recognize lumbar root entrapment within the root canal: (*a*) severe, constant root pain to the lower leg, (*b*) pain unrelieved by bed rest, (*c*) minimal tension signs, and (*d*) patients over 40 years of age. In one study (29), 249 patients fulfilled these criteria, representing 11% of patients attending a back pain clinic. Most had restricted spinal extension, but few had abnormal neurologic signs. Degenerative change was common, especially disc space reduction. Central canal size measured by ultrasound was normal, which is compatible with a variable history of back pain. Eighty percent of patients showed a long history of back pain, and 90.4% of them were managed by nonoperative means. Although 78% of these still had some root pain between 1 and 4 years after first attendance, most of them were not troubled sufficiently to have sought alternative help (29).

The disc has been the focus of attention for several decades; at one time, root pain was almost synonymous with a diagnosis of disc lesion. It is now recognized that a lumbar root can be

affected by other pathology and at a different site from the acute disc lesion.

Patients with a root canal lesion are identified by four criteria: severe root pain, older age, unrelieved by bed rest, and without gross limitation of straight leg raising.

Root canal pathology can occur from spondylolysis, congenital facet hypertrophy at L5–S1, and previous trauma to the apophyseal joint, but the high incidence of *disc space resorption* suggests that previous disc pathology *is a major cause of this syndrome*. Radiographs of the lumbar spine showed a greater incidence of disc space narrowing than would be expected (30).

Intraosseous pressure (IOP) and cerebrospinal fluid pressure (CSFP) in the lumbar region were measured simultaneously in two groups of patients with either spinal canal stenosis or disc herniation to compare dynamic changes with positional changes, and to learn whether these pressure changes have some role in the onset of claudication. IOP and CSFP showed almost the same change patterns with positional shifts in two groups. They were lowest in the prone position and highest in the standing position. In standing with flexion, they were almost the same as in the prone position, but *in extension, they increased above the standing pressure*. Dynamic pressure changes could act as a compression force to the cauda equina in the patient with spinal canal stenosis (31).

Stenosis Determination and Treatment Influence

The existence of the stenotic lumbar canal is another factor to be considered in the effectiveness of lumbar spine manipulation. Certainly, the congenital presence of this abnormality cannot be reversed without surgical relief. Yet the best of manipulation may well render a measure of relief for the patients without providing 100% relief from symptoms. Alterations occurring with stenotic changes, namely, ligamentum flavum hypertrophy and disc degeneration, may not be reversible. Weinstein et al. (32) and others have had a 70% success rate with surgery in decompression laminotomy of patients with a stenotic lumbar spine. Thus, clinical investigation and statistic keeping eventually will provide an answer to the effectiveness of manipulation versus surgery in the treatment of patients with this condition.

With the techniques of measurement outlined in this chapter, it certainly is possible to determine the existence of lumbar canal stenosis and the prevalence of spondylotic canal radiculopathy by clinical investigation. Clinically, follow-up will show the effectiveness of manipulation. In a report in the *Journal of the Canadian Chiropractic Association* (33), 744 patients with neck and back pain were treated with spinal manipulation. These patients were referred from the Orthopedic Clinic at the University Hospital in Saskatoon. The reports covered only those suffering with low back and leg pain and the effects of manipulation. It was found that 70% of the patients did well and that spinal manipulation now receives top priority in the conservative management of back problems at this center. One of the main points I wish to stress is that the postsurgical patient did well under chiropractic care and that patients at that center were routinely referred back 3 months after surgery for manipulative care. Spinal surgery was regarded not as the end but rather as the beginning of manipulative involvement. Thus, a strong possibility exists that in the treatment of the patient with a stenotic lumbar canal, a combination of surgical decompression and manipulation may render the greatest benefit.

Figure 4.7 shows various stenotic formations. Radiograph may reveal the osteoarthritic involvement of facets that enter and reduce the lateral recess of the vertebral canal. Myelographic studies to define it are performed by injecting 30 mL of dye into the subarachnoid space and taking films with the patient in an upright position. An anteroposterior diameter of less than 14 mm is suggestive of stenosis (34). The lumbar spinal canal usually becomes progressively wider from L1 to L5 (35), and is most shallow at L5.

Figure 4.8 illustrates the Jones and Thomson (36) formula used to measure the ratio of the canal to the vertebral body, which is an accurate radiographic indicator of lumbar stenosis. This technique eliminated misinterpretation of the plain radiographs caused by patient size, magnification, and rotation and provided good clinical correlation in 12 of 13 patients. Ratios of 1:2 to 1:4 (small normal) were considered normal, and ratios of 1:4 to 1:6 were considered stenotic. Of course, this technique does not provide as accurate a measurement as does

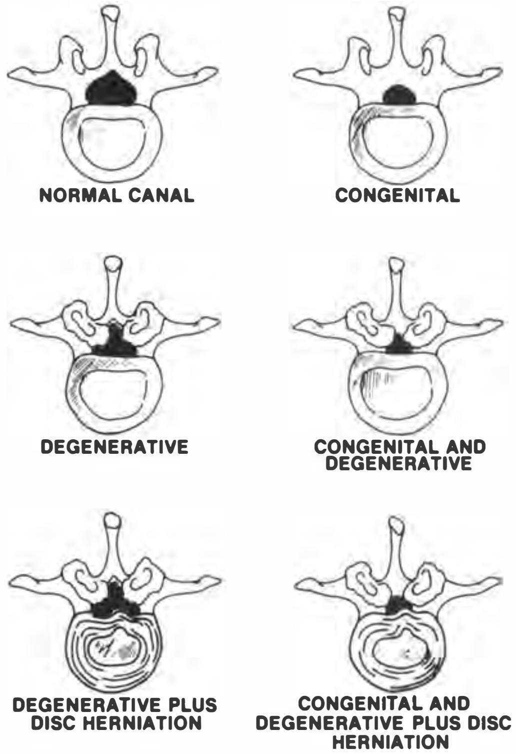

Figure 4.7. This diagram shows the normal canal and various combinations of conditions that may cause spinal stenosis. Congenital stenosis with disc herniation alone, not pictured here, is another possibility. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:293.)

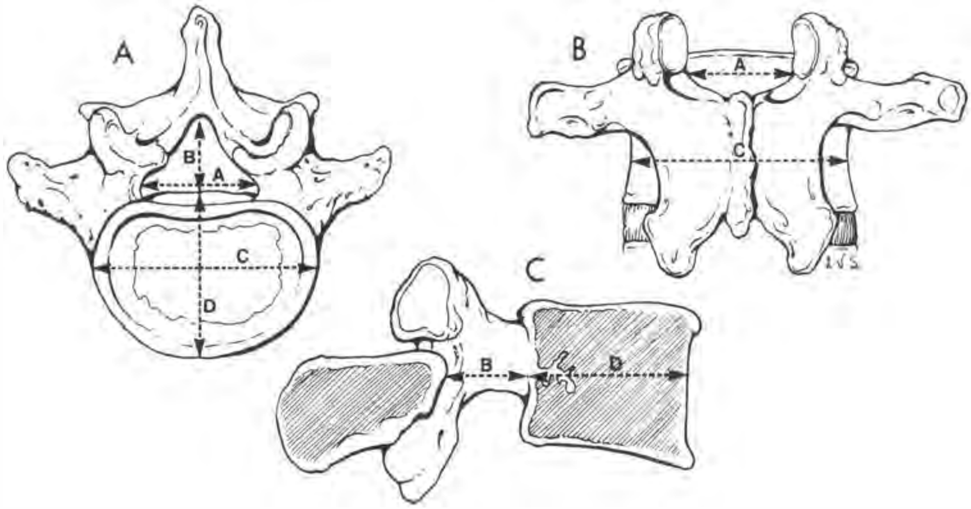

Figure 4.8. **A.** Axial section showing anteroposterior diameter of a fifth lumbar vertebra. **B.** Superior view. **C.** Median sagittal view. *A*, interpedicular distance; *B*, anteroposterior diameter of spinal canal; *C*, transverse diameter of vertebral body; *D*, anteroposterior diameter of vertebral body. The products *AB* and *CD* are compared. (Reprinted with permission from DL McRae. Radiology of the lumbar spinal canal. In: Weinstein PR, et al. Lumbar Spondylosis: Diagnosis, Management and Surgical Treatment. St. Louis: Mosby Yearbook, 1977.)

CT scan, but it is a good indicator that more detailed tests such as CT or myelography are needed.

According to Epstein et al. (37), an anteroposterior spinal canal diameter of less than 13 mm (from the posterior margin of the intervertebral foramen to the posterior surface of the vertebral body) indicates stenosis. Hypertrophic osteoarthritic spurs may be tolerated in a normal canal but create severe compression of nerve roots in stenosis. Considerably more spurring can be tolerated at L5–S1 than at L4–L5 because of a "snug" bony confine at L4–L5 and a "great" amount of space at L5–S1 between neural elements and bone.

Figures 4.9 and 4.10 reveal why patients with stenosis stand in a flexed posture, that is, to maximize the sagittal diameter of the spinal canal.

Thecal Sac Pressure in Stenosis

Different morphologic measurements were studied in the evaluation of patients with lumbar spinal stenosis (20). Preoperative CT scans from 24 patients who underwent surgery for central lumbar stenosis were analyzed.

In most patients, the common tissues causing stenosis appeared to be protrusion of soft tissues, including the disc and ligamentum flavum. It was concluded that (*a*) Bony measurements alone do not reliably identify patients with spinal stenosis. (*b*) The size of the dural sac is a more reliable measure of stenosis than bony measurements. Measurements of the transverse area of the dural sac on CT scans, enhanced by contrast in the sac, is the most accurate method for identifying stenosis. (*c*) Myelography is still considered to have an important role in the evaluation of a patient with stenosis, because the size of the dural sac can be estimated from myelographic data. (*d*) Degen-

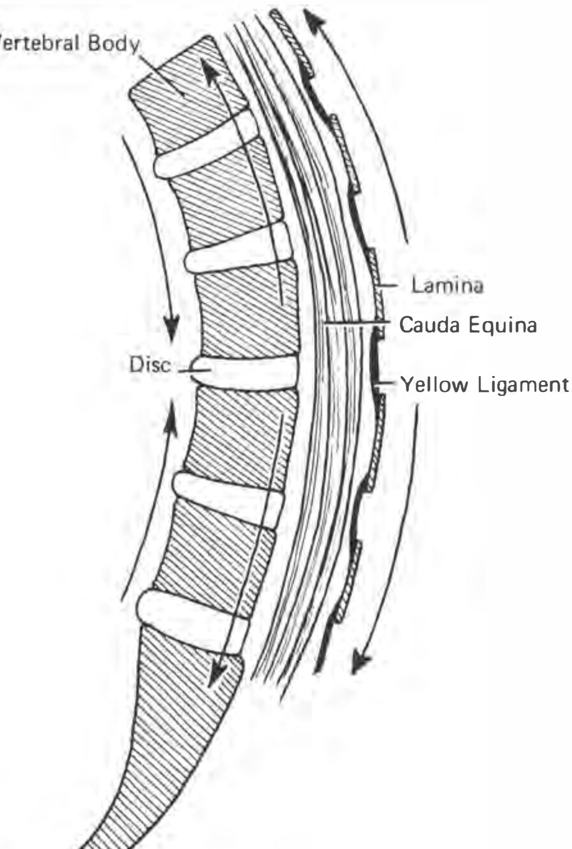

Figure 4.9. Increased spinal canal volume and decreased nerve root (cauda equina) bulk with flexion. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:432.)

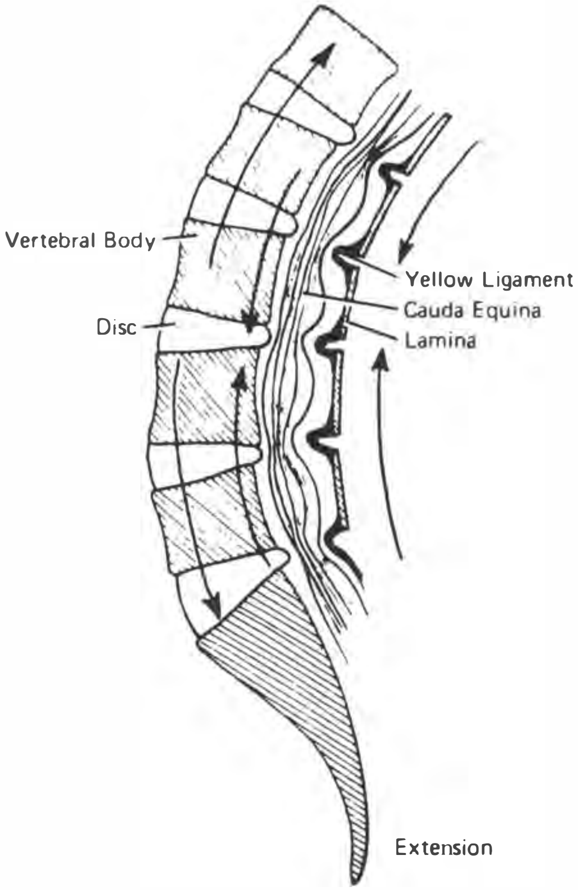

Figure 4.10. Decreased spinal canal volume and increased nerve root bulk with extension. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:432.)

erative changes within the facet joints and intervertebral discs, as well as encroachment on the canal by the ligamentum flavum, were the most common abnormalities associated with spinal stenosis. (*e*) Further investigation is needed to determine the critical size of the dural sac.

To register pressure changes within the cauda equina, a highly sensitive pressure-measuring catheter was inserted through a hole in the dural sac (Fig. 4.11) (21). Then, by circumferentially restricting the transverse area of the intact cauda equina, Schonstrom et al. found that pressure started to build up in it at a cross-sectional area of the dural sac ranging from 60 to 80 mm^2. Once this critical size was reached, even a minimal further reduction of the area caused a distinct pressure increase among the nerve roots (21).

EFFECT OF STENOSIS ON INTRAOSSEOUS BLOOD FLOW

In recent years, investigations by intraosseous phlebography have provided evidence of disturbed venous outflow from juxtachondral bone marrow of osteoarthritic joints (38).

Intraosseous stasis is accompanied by a rise of intramedullary pressure. Aching pain at rest, a typical symptom of advanced osteoarthritis, seems to be provoked by high pressure in the bone marrow. Release of intraosseous hypertension by osteotomy or critical fenestration is followed by prompt disappearance of these pains.

Intraosseous pressures in the lumbar vertebrae of patients with low back pain show that low back pain seems similar in quality to the aching rest pain experienced by patients with severe osteoarthritis, and the radiographic changes observed in spondylosis deformans are indicative of processes similar to osteoarthritis. Arnoldi (38) reported on intervertebral pressure measurements in patients with various types of lumbar pain. Pressures were measured in the spinous processes, and at least three vertebrae were examined simultaneously in each patient. In radiographically normal vertebrae, the intraosseous pressures varied within narrow limits (2 to 13 mm Hg), with a mean value of 8.3 mm Hg. In vertebrae with spondylotic changes in the radiograph, the pressure was significantly higher (28.1 mm Hg mean, 14 to 49 mm Hg range). All pressures are referred to heart level. No relationship was found between the degree of spondylotic changes in the radiograph and the elevation of intraosseous pressure. As far as I am aware, this is as yet the only report on intraosseous pressures in patients with lumbar pain. As mentioned , this is a preliminary report and its value is limited. It contains no data from healthy subjects, and

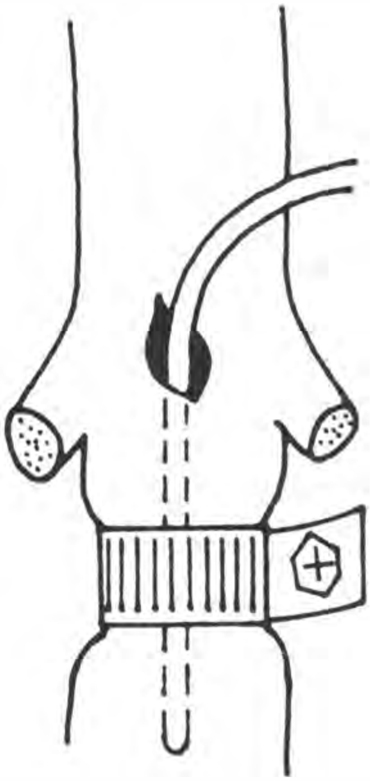

Figure 4.11. The pressure-recording catheter inserted through a hole in the dural sac. The hose clamp was applied around the sac 5 mm below the exit of the nerve roots. (Reprinted with permission from Schonstrom N, Bolender NF, Spengler DM, et al. Pressure changes within the cauda equina following constriction of the dural sac: an in vitro experimental study. Spine 1984;9(6):605.)

no measurements were performed on patients with asymptomatic spondylosis deformans. Phlebography was not done in this study (38).

Somatosensory Evoked Potential Examination For Stenosis

Cortical somatosensory evoked potential (CSEP) examinations were performed on 20 patients with lumbar spinal stenosis 1 day prior to surgery and 10 to 12 days after spinal decompression and bilateral lateral fusion (39). CSEPs were recorded following stimulation of 32 tibial, peroneal, and sural nerves and 16 saphenous nerves. A total of 110 nerves were examined. Using CSEP P1 latency as criteria for inclusion in the study, 21 tibial, 20 peroneal, and 17 sural nerves were subjected to paired two-tailed t tests to determine whether the CSEP changes that occurred postoperatively were statistically significant ($P < 0.005$). Postoperative P1 latencies of tibial, peroneal, and sural nerves changed significantly, as did N1 latencies and P1-N1 amplitudes of tibial and peroneal nerves. Ten patients improved clinically. It was postulated that pathogenic narrowing of the spinal canal stenosis leads to nerve root compression and ischemia, with resultant dysfunction primarily affecting large-diameter myelinated fibers, and that a decompression procedure may adequately relieve the underlying pathologic processes. Improvement in CSEPs may be caused by an increase in available numbers of functioning large-diameter myelinated fibers, conversion to normal from a conduction block, and, perhaps, improved axoplasmic flow (39).

Keim et al. (40) described the use of somatosensory evoked potentials (SEPs) to localize the level, extent, and laterality of nerve root entrapment. The results confirm a high incidence of fourth and fifth lumbar and first sacral nerve root involvement. The posterior tibial nerve was abnormal in 95% of cases, the peroneal in 90%, and the sural in 60% in the symptomatic lower extremity.

Significant stenosis can cause compression of the nerve roots of the cauda equina in the lateral recess or in one or more foramina. Patients with symptomatic lumbar stenosis with or without neurogenic claudication may report pain, paresthesia, or lower extremity weakness, usually patchy in distribution. Different roots may be involved unilaterally or bilaterally. The condition is much more common than has been suspected in the past, and it is probably present to some extent in most persons over the age of 60 years.

Anatomically, spinal stenosis can have the following variations: (*a*) lateral, due to hypertrophy of the superior articular process; (*b*) medial, due to hypertrophy of the inferior articular process; (*c*) central, due to bony projection (diastematomyelia) or hypertrophic spurs, thickening of ligamentum flavum or superior edge of the lamina of the inferior vertebra; (*c*) fleur-de-lis (cloverleaf), due to posterolateral bulging caused by thickening of laminae.

Patients with spinal stenosis often present with vague, sketchy clinical findings that are usually misleading. Most patients present with symptoms of pain because the sensory, not the motor, fibers are primarily affected. It is not uncommon that the usual electrodiagnostic procedures such as electromyography (EMG), motor nerve conduction, and F waves are not revealing. The "H" reflex can be used to evaluate sensory fibers, but its value is limited to the S1 function of primary afferent pathways.

Technically CSEPs are easy to perform, and they are noninvasive, and painless. The technique has proved to be a reliable diagnostic tool with a high yield of accuracy in delineating the extent and laterality of nerve root involvement in spinal stenosis (40).

Factors on Effects of Stenosis

Age

The lumbar spinal canal has no further potential for growth by infancy as regards the midsagittal diameter and the cross-sectional area. Thus, in the case of delayed development, it is not capable of catch-up growth (41). The degree of narrowing in the canal increases with senescence, the canal is most narrow by median age of 67 years (42).

Sex

No association was found between sex and degree of stenosis.

Stenotic Results on the Nerve Complex

Low Pressure on Dorsal Root Ganglion (DRG) and Nerve Diminish Nutrition Supply

A pressure of only 10 mm Hg induced a 60% reduction of efferent nerve impulse amplitude during 2 hours of compression and a complete block at 50 mm Hg. Blood flow in the uncompressed nerve root segment between the two balloons at 10 mm Hg was reduced to 64% of normal and the nutritional transport to the same nerve segment was drastically reduced.

Low compression pressures cause (*a*) changes in blood supply, (*b*) endoneurial edema, (*c*) metabolic imbalance, and (*d*) altered impulse propagation. Symptoms and signs in central spinal stenosis are likely to be secondary to these combinations of nerve tissue reactions induced by mechanical compression (43).

Narrow Canal Equates to Longer Leg Pain Duration

Cross-section areas of the disc hernia, the dural sac, and the residual spinal canal were measured on computed tomography–myelography in 58 patients with lumbar disc herniation who did not undergo surgery. After a median of 14 months from the onset of leg pain, 77% had returned to work, and only 7% were pain-free. Hernia size was not associated with the outcome measures. A high score for pain intensity and distal pain distribution was associated with a wide dural sac and a wide residual spinal canal. Patients with the longest duration of leg pain had the narrowest spinal canals (44).

Epidural Pressure

Epidural pressure at the stenotic level in patients with lumbar spinal stenosis is changed by posture. The pressure is lowest (18 mm Hg) when lying down. Pressure in sitting is two times higher than that in lying; it is four times higher in upright standing than in lying. Highest pressure (116 mm Hg) is measured in standing with extension, which is about six times higher than that in lying. In standing posture, the pressure with flexion is one fourth of the pressure with extension. These pressure changes may explain the postural dependency of symptoms in spinal stenosis. Increased pressure to the dural sac by posture may induce the compression of the nerve roots. As a result, cauda equina and radicular symptoms may appear (45).

Increased Intraosseous Blood Pressure

Intraosseous hypertension is found in the vertebral bodies in positions causing low back pain, such as sitting, whereas lying down reduces the pressures and is not painful (46).

DORSAL ROOT GANGLION CHANGES

The dorsal root ganglion is a vital link between the internal and external environment and the spinal cord. The primary sensory role of the spinal cord is to receive afferent stimuli in the form of action potentials and to relay the information transmitted to and from the brain (47).

Cells in the DRG were originally divided into two classes according to their diameters. The large cells give rise to large myelinated fibers and the small cells to the unmyelinated (C) and finely myelinated (A) fibers. The central terminations of these primary afferent fibers, derived from the small cells, end mainly in the substantia gelatinosa, lamina 2 of the spinal cord. Several peptides, including calcitonin-generated peptide and substance P, have been localized to a subpopulation of small DRG cells. To date, calcitonin gene-related peptide is the most abundant peptide in the DRG.

In 1983, Wall (47) also demonstrated the DRG to have ongoing activity and mechanical sensitivity that could be a source of pain-producing impulses and could contribute to pain in those conditions of peripheral nerve damage where pain persists after peripheral anesthesia.

Pressure on the DRG and Nerve Root

Cerebrospinal fluid plays an important role in the nutrition of the nerve roots. The dorsal root ganglion is well vascularized compared with other parts of the nerve root. These factors help support nutrition created by the increased metabolic demand of the DRG in which several important substances are synthesized. Included in some of these neuropeptides are substance P, vasoactive intestinal polypeptides (VIP), and proteins needed to maintain structural and functional integrity of the entire sensory neuron (43).

SYMPTOMS OF A STENOTIC CANAL

The mean duration of stenosis symptoms was reported at 20 (1 to 180) months with 72 patients (58%) having claudication, 43 (35%) radicular pain, and 8 (7%) mixed symptoms. Reduced power of the extensor hallucis longus and peroneal paresis were the most prevalent signs (2).

Stenosis can be present with (*a*) no symptoms, (*b*) neurogenic claudication, (*c*) symptomatic disc protrusion, or (*d*) root entrapment with degenerative changes (48). The classic symptom of central spinal stenosis is claudicating leg pain, aggravated by standing or walking and relieved by forward flexion or sitting (48).

Lateral Recess Stenosis

Radiculopathy of the L5 nerve root may be caused by L4–L5 disc herniation or by L5–S1 foraminal stenosis. Radiculopathy caused by lateral canal stenosis consists of pain in a dermatomal distribution and sensory or motor deficits of a particular nerve root (48).

Differential of Lateral and Central Stenosis Symptoms

Back and leg pain lasts longer in patients with central rather than lateral stenosis; back pain usually lasts 15 years before leg pain commences. No association with the degree of stenosis nor difference in the symptoms of patients with lateral and central stenosis was reported in this study (42). Claudication was found in both groups at all degrees of stenosis. The neurologic findings were equal in lateral and central stenosis and did not increase with the degree of stenosis.

Reflex Sympathetic Dystrophy

Reflex sympathetic dystrophy is a syndrome of burning pain, hyperesthesia, swelling, hyperhidrosis, and trophic changes in the skin and bone of the affected extremity. It is precipitated by a wide variety of factors in addition to nerve injury. It occurs outside of dermatomal distributions and can spread to involve other extremities without new injury. The diagnosis is primarily clinical, but radiography, scintigraphy, and sympathetic blockade can help to confirm the diagnosis. The most successful therapies are directed toward blocking the sympathetic innervation to the affected extremity, in conjunction with physical therapy. The theories proposed to explain the pathophysiology of reflex sympathetic dystrophy include "reverberating circuits" in the spinal cord that are triggered by intense pain, ephaptic transmission between sympathetic efferents and sensory afferents, and the presence of ectopic pacemakers in an injured nerve (4).

Previously termed "causalgia," for the condition in soldiers with persistent burning pain and progressive trophic changes in a limb following gunshot injuries, today, all such manifestations of sympathetic overactivity are termed "reflex sympathetic dystrophy."

Reflex sympathetic dystrophy can be associated with lumbar disc herniations. Both central and peripheral neuroanatomic pathways can be implicated in the development of this syndrome. Clinical findings of (*a*) vasomotor instability in the leg, supported by plain radiographs showing osteopenia; (*b*) bone scan showing increased uptake; and (*c*) a favorable response with sympathetic blocks suggest the diagnosis. Symptoms should be relieved with appropriate nerve root decompression but may also require a therapeutic lumbar sympathetic blockade (49).

Lumbar Degenerative Disc Disease-Induced Stenosis

The triple joint complex, made up of the two posterior zygapophysial joints and single anterior intervertebral disc, can be affected by trauma or degenerative disc disease. One of the joints of the complex begins the process of degeneration, and it may or may not be symptom producing. However, because the function of the three joints are so intertwined, changes in any one eventually affects the other.

With combined triple joint complex degeneration, eventual loss will occur in disc height and facet cartilage, with resultant ligamentous laxity of other ligamentous restraints. Stress will be transferred to levels above and below, where the process will repeat itself until multilevel spondylosis occurs (50). Nerve entrapment can occur at each level.

Algorithm of Stenosis Development

Panjabi et al. (51) probably best developed the stages of stenosis in the following algorithm:

1. Asymmetric disc injury at one functional spinal unit (FSU).
2. Disturbed kinematics of FSUs above and below injury.
3. Asymmetric movements at facet joints.
4. Unequal sharing of facet loads.
5. High load on one facet joint.
6. Cartilage degenerative *or* facet atrophy and narrowing of intervertebral foramen (IVF).

Patient Presentation

Degenerative stenosis patients typically present as follows:

1. Eighty-eight percent show symptoms distal to the buttocks; only 56% show pain distal to the knees, indicating that calf pain, often considered part of the pseudoclaudication syndrome, is not necessary to establish a diagnosis of lumbar spinal stenosis (52).
2. Extension increases stenosis symptoms by decreasing the spinal and neural canal areas. Absence of pain when seated is highly specific for lateral lumbar stenosis, and thigh pain with lumbar extension is an independent correlate of the diagnosis. These mechanical relationships support the concept that lumbar flexion increases and extension decreases the cross-sectional area of the spinal canal and neural foramina.
3. The most specific physical examination findings are a wide-based gait and abnormal Romberg test.
4. Numbness is noted in 51% of lateral lumbar stenosis cases preoperatively.
5. Absent ankle reflexes, muscle weakness, and sensory deficit in 58, 51, and 52%, respectively, are reported in lateral lumbar stenosis patients.
6. Lateral lumbar stenosis patients showed that 65% had reduced ankle reflexes, 42% had reduced strength of the extensor hallucis longus, 46% had sensory disturbance, and 51% had radiating pain on lumbar extension (52).

STENOSIS FACTORS IN BACK PAIN

Severe Back Pain

Patients with small canals are more likely to visit doctors and have treatment for back pain. Canal measurement is not a predictor for back pain, but it is a risk factor for severe back pain in early working life (53).

Poorer Health and Decreased Academic Ability

Impaired early programming of canal growth leaves a small adult vertebral canal, and other sensitive developing systems can be affected similarly, producing in an adult a small canal, declining health, and poor academic ability (54). The hypothesis has not been proved, but it is supported and deserves further study.

GROWTH FACTORS IN STENOSIS

Does infant malnutrition produce smaller adult spinal canals? Lumbar and thoracic vertebrae ($n = 1073$) from a prehistoric American Indian population (15 to 55 years of age) were measured for anteroposterior (AP) and transverse vertebral canal sizes, nerve root tunnel (NRT) (intervertebral foramen) widths, vertebral heights (VH), vertebral osteophytosis (VO), and tibial lengths. They underwent a dietary change from hunting and gathering, with a protein-rich (PR) diet, to maize agriculture, with a protein-deficient (PD) diet, between 950 and 1300 AD. The multivariate analyses done controlled for age, sex, culture, NRT, VH, VO, and wedging. Canal size was significantly smaller in the PD subjects. AP diameters were generally and highly correlated with NRT, and thus both spinal stenosis and sciatica may have a developmental basis. Canal size was independent of statural components. Consequently, canal size is a most powerful tool in assessing infant malnutrition. Moreover, perhaps the association between canal size and low back pain (LBP) found in living populations has been underestimated, and this component of LBP is preventable (55).

Roaf (56) provided rough estimates in inches for lumbar and thoracic spine growth from 2 to 16 years of age. The lumbar vertebrae and discs grow approximately twice as much as the thoracic area. He suggests (without data) that, in the thoracic

region, the posterior elements may grow faster than the anterior, and that this may be reversed in the lumbar region.

Using data from Porter et al. (57), Eisenstein (58), and Hinck et al. (59), it appears that at birth the canal is approximately 65% of its adult size, and by 5 years it is 90% of its adult size. In addition, within the canal, the AP diameters appear to be more advanced than the transverse (TR) vertebral canal.

First, the estimates of the association between low back pain and canal size, derived from ultrasound readings, using a 15° oblique angle, suggest that only 2 mm (a decrease in canal size of about 15%) separates persons with and without LBP (57). The frequency of small transverse diameters and LBP has been suggested to be 53% (14). Consequently, if AP diameters are most variable and most frequently associated with LBP, then even less than 2 mm may separate those persons with and without LBP. Indeed, more than 53% of patients with LBP may have AP spinal stenosis.

Dual-Level Stenosis

A single-level stenosis probably causes little neurologic dysfunction because the nerves are well supplied with oxygenated blood from a proximal and distal supply. A two-level low pressure stenosis, however, will produce more profound effects. The arterioles will supply the uncompressed segment between the two blocks, but the venous return will be impaired, and a long segment of cauda equina will become congested. Metabolites will build up, and reduced blood flow will impair nutrition. In a two-level central canal stenosis, all the cauda equina will be congested with bilateral symptoms. In single-level central stenosis, with a more distal root canal stenosis, only a single root will be congested and unilateral symptoms seen (60).

INTERMITTENT CLAUDICATION

Definition and Risk Factors

Claudication is a descriptive term for the clinical symptom complex of exercise-induced leg pain that is relieved by rest. The primary risk factor for claudication is cigarette smoking. The relative risk of developing claudication is 2.11 if a person smokes more than 20 cigarettes per day and is 1.75 if a person smokes 11 to 20 cigarettes per day. The risk remains increased for up to 5 years after smoking cessation. Diabetes, systolic hypertension, hypercholesterolemia, increasing age, and increasing body mass also are statistically significant variables related to claudication. A combination of risk factors increases the relative risk of claudication (61).

Differential Diagnosis

Although neurogenic and ischemic causes of back pain may clinically appear similar, certain clinical signs and symptoms can be used to differentiate the two. Neurogenic claudication is characterized by vague leg pain anteriorly and posteriorly over the thighs and calves. This is caused by postures in the spine that mechanically compromise the neural canal and foramina. In addition, paresthesias and dysesthesias in the lower extremities occur with these postural changes.

In contrast to neurogenic claudication, claudication associated with ischemia is manifested by pain, dysesthesias, and paresthesias that occur with ambulation, but which are relieved by rest or lying supine. The absence of pulses and the presence of pallor distally are classic signs of vascular claudication, but they are not associated with the neurogenic form (62).

Figure 4.12 is a differential diagnostic chart of lower extremity pain as caused by arterial insufficiency and neurogenic claudication. According to Weinstein et al. (32), the classic clinical symptom of a narrowed lumbar canal is pain aggravation in the lower extremities following exaggerated lordosis of the lumbar spine. This classic clinical symptom is one of numbness and tingling or a feeling that the legs are asleep. It may be brought on by standing, bending backward, or reaching overhead. Ehni (63) has shown that during myelography lumbar spine extension produces total block of the column, whereas flexion permits the dye to pass through the lumbar spine.

An interesting diagnostic point was presented by Dyck et al. who said that the ankle reflex, when accompanying intermittent claudication, may be absent after exercise and present when at rest. Furthermore, Weinstein et al. (32) noted that two patients in the claudication groups had urinary retention when ambulatory, but following rest could void normally.

Finding	Arterial Insufficient Claudication	Neurogenic Claudication
Arterial pulses of femoral, popliteal, post. tibial, and dorsalis pedis	One or more diminished	Normal
Pain in legs induced by	Exercise such as walking but not by posture change	Walking, standing kneeling, hyperextension
Relieved by	Rest	Bend forward, squat, flexion
Accompanied by low back, buttock, thigh pain	Rare	Common
Type pain	Cramping is severe if exercise is continued	Dysesthesia such as numbness, tingling, and burning
Comes at rest	No	Yes
Sensory loss	Rare	Mild
Leg raise	Normal	Normal
Arterial murmur	Yes	No
Plain x-ray findings	Arteriosclerosis of abdominal aorta or iliac and femoral vessels	Discogenic spondyloarthrosis

Figure 4.12. Differential diagnostic factors of intermittent claudication.

Peripheral and Cardiovascular Signs Coexist

Physical Examination

Evaluation of the carotid artery pulses is recommended because atherosclerosis, the most prevalent cause of occlusive peripheral arterial disease, commonly affects the carotids as well as the peripheral arteries (64).

After examination of the peripheral arterial pulses is completed, auscultation over the carotids, abdominal aorta, and femoral and popliteal arteries is useful in detecting mild occlusive arterial disease. A systolic bruit is indicative of turbulence, most often caused by atherosclerosis proximally; a bruit extending into diastole is heard when the arterial narrowing proximally is sufficiently severe to produce a gradient (and therefore flow) in diastole—a useful sign of significant occlusive arterial disease.

To determine the degree of occlusive arterial disease, the elevated extremity can be graded as follows (64):

Grade of Pallor of Elevation	Appearance at Designated Duration
0	No pallor in 60 seconds
1	Definite pallor in 60 seconds
2	Definite pallor in less than 60 seconds
3	Definite pallor in less than 30 seconds
4	Pallor with no elevation of extremity

Doppler Testing

Doppler segmental pressures with an ankle-brachial index (ABI) provide information about the physiologic significance of clinically suspected arterial obstruction. The ABI is a ratio of the ankle blood pressure:brachial blood pressure. An ABI of greater than 0.85 is considered normal, an ABI of 0.50 to 0.84 suggests arterial obstruction with claudication, and an ABI of less than 0.50 suggests significant arterial obstruction with critical ischemia.

Occasionally, patients have normal ABIs and segmental pressures at rest, but their symptoms strongly suggest claudication. For this diagnostic test, the patient walks until the onset of leg pain. Exercise may unmask the arterial obstruction, resulting in a postexercise change in the Doppler segmental pressures and the ABIs (61).

Differential Diagnosis

Disc herniation leads to operation much sooner than lateral or central spinal stenosis. Pain at rest, at night, and on coughing is as common in lateral spinal stenosis as in disc herniation, although it is probably less severe. Root tension signs are common in disc herniation, less so in lateral spinal stenosis, and rare in central spinal stenosis. Neurologic abnormalities are most common in central spinal stenosis, especially reduced or absent patellar reflexes. Profiles of symptoms and signs in the three conditions differ and are sufficiently specific to help in diagnosis (65).

Mechanisms of Intermittent Claudication Causation

Various mechanisms have been suggested as the cause for intermittent claudication—for example, ischemic neuritis of the cauda equina, narrowing of the spinal canal at standing, venous return obstruction induced by the increase of cerebrospinal fluid pressure below the stenosis, and changes of nerve root microvascularization at standing.

In spinal stenosis, the pressure on the cauda equina probably varies with posture and exercise. Clinically, therefore, a situation of intermittent, rather than continuous cauda compression, thus might correspond well to the clinical condition of neurogenic claudication (66).

An adequate blood supply is one important component in preserving the functional properties of nerve roots. A pressure level of 10 mm Hg is sufficient to induce a significant reduction of both blood flow and supply of nutrients to the nerve roots. After compression release, blood flow is restored within minutes. A pressure level of 10 mm Hg is known to induce venular congestion in the nerve roots. Therefore, it is a likely assumption that the venular congestion induced by the continuous compression can significantly affect the recirculation of the cauda equina when the intermittent compression component is released. A pressure of 50 mm Hg, in addition to the venular congestion, will also affect the capillary and arteriolar blood flow. Therefore, restoration of cauda equina blood flow is likely to be more impaired at 50 mm Hg than at 10 mm Hg (66).

Exercise-Induced Ischemia of the Nerve Roots

Twenty-two minipigs were trained to run on a treadmill. Two-level lumbar spine stenosis was created in 12 pigs, 10 were nonoperated control subjects. Blood flow of the spinal cord and nerve roots was determined with microspheres at rest, during exercise, and after exercise. Studied were the effects of lumbar spinal stenosis and exercise on blood flow of spinal neural tissue. Results suggest that exercise-induced impairment of spinal nerve root blood flow plays a role in the pathophysiology of neurogenic claudication (67).

Combined Cervical and Thoracic Spine Stenosis

Twenty patients with spinal intermittent claudication, caused by cervical and thoracic lesions, who were given surgical treatment ($n = 19$) were studied. Their main subjective symptoms were tightness, weakness, and numbness in the lower limbs and a strangulated sensation in the trunk to lower limbs. Objective findings were occurrence and/or aggravation of the spinothalamic tracts. Circulatory impairment of the spinal cord seems to be closely related to the cause of spinal intermittent claudication (68).

Treatment

In intermittent claudication, conservative treatment consists of eliminating risk factors, particularly smoking, drug treatment, and physical exercises. Exercise can prolong the pain-free walking distance of claudicants. The optimal exercise program should be supervised, performed regularly for at least 2 months, and be of high intensity. Although many fundamental questions remain unanswered, it is justified to prescribe exercise therapy for intermittent claudication more generally than is realized in today's practice (69). It should consist of exercise to or through the onset of claudication, rest until the pain resolves, and then resume exercise. The exercise is performed daily in a session lasting 30 minutes to 1 hour (61).

Claudication is an obvious marker for systemic atherosclerosis, and the long-term survival rate is lower in patients with this condition than in age-matched control subjects.

Hypertension is an independent risk factor for claudication to be treated. All patients with claudication should stop smoking. Hyperlipidemia is associated with claudication. The low-density lipoprotein cholesterol level should be lowered to below 100 mg/dL (61).

So far as drug therapy is concerned, pentoxifylline (Trental) increases red blood cell deformity, decreases plasma viscosity, decreases platelet aggregation, and increases resting and hyperemic extremity blood flow. Aortobifemoral bypass is reported successful in more than 90% of patients with aortoiliac occlusive disease. Most patients with claudication respond to conservative therapy (61).

Treadmill Stress Testing

A prospective study of patients with neurogenic claudication and lumbar spinal stenosis determined pre- and postsurgical functional status to evaluate the outcome of surgical intervention and found treadmill testing to be a useful indicator of functional status and surgical outcome (70).

DIAGNOSIS: IMAGING DIAGNOSIS OF SPINAL STENOSIS

Pathologic Sequence of Lumbar Dysfunction

Understanding the pathologic process and making a concise and precise diagnosis of which nerve or nerves are affected are important steps in the formation of a logical treatment plan (71). The pathologic process of low back pain is composed of:

1. **Dysfunction,** which is nearly always relieved by nonoperative measures. Ninety percent of patients with low back pain have this symptom. Attendance at a spine education program, a light elastic garment, manipulations, or posterior joint injections relieve most patients of their symptoms.
2. **Disc herniation.** Seventy-five percent of patients with a first herniation respond well to the measures outlined immediately above, together with a period of rest in bed. The remaining 25% may require chemonucleolysis or discectomy.
3. **Lateral entrapment (stenosis).** Approximately 50% of patients with this type of lesion respond to nonoperative measures. Manipulation is an effective method of treatment. In most cases, the remaining 50% require operative decompression with enlargement of the narrow lateral canal.
4. **Central stenosis.** Combining a clinical assessment with EMG studies, radiographic and CT scan examination, and sometimes a selective nerve block, makes it easy to identify the entrapped nerve or nerves. A few patients respond to nonoperative measures. Many require decompression.
5. **Instability.** Patients with a minor degree of instability often require no more than decompression. Those with major instability require fusion of the affected level following decompression and at the same operation (71).

PLAIN RADIOGRAPHIC EVALUATION FOR STENOSIS

Figures 4.13 and 4.14 are photographs of two lumbar vertebrae. Figure 4.13 shows the typical round vertebral canal with fairly well-developed pedicles, whereas Figure 4.14 shows a trefoil canal with underdeveloped pedicles.

Various clinicians have measured the interpedicular and sagittal diameters of the canal. Epstein et al. (72) found the sagittal diameter normally to be 15 to 23 mm, with a measurement of less than 13 mm clinically significant of narrowing. He further noted that accompanying the shortened pedicles are thickened neural arches and prominent facets, which further narrow the diameter. Paine and Haung (73) report that the

Figure 4.13. Photograph of actual lumbar vertebra showing a rounded vertebral canal with well-developed pedicles.

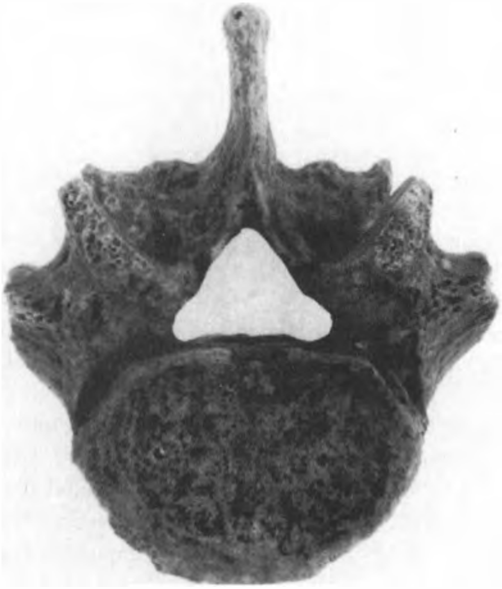

Figure 4.14. Photograph of a trefoil-shaped vertebral canal with underdeveloped pedicles.

sagittal diameter of canals in patients with stenosis is 8 mm. The pioneer and perhaps the best authority on stenosis of the canal is Verbiest (74), who states that a sagittal diameter of less than 12 mm is definitely too short. His conclusion is based on the measurements of the vertebrae of American (75), Dutch (76), Norwegian and Lapp (77), and White and Zulu skeletons (78). According to Verbiest (74), absolute stenosis is indicated when the sagittal diameter is 10 mm or less, which may produce signs of radicular compression in the absence of any additional compressive agent (e.g., disc protrusion, ligamentum flavum hypertyrophy, and lamina hypertrophy). Midsagittal diameters between 10 and 12 mm are classified as relative stenosis and serve as warning of possible future disturbances caused by the development of spondylosis and its accompanying arthritic changes in the facets. According to Verbiest, a narrow canal and mild disc protrusion or minimal ventral osteophytosis produces symptoms, which could be well tolerated in a lumbar canal of normal size.

Plain Film Markings

In our clinical investigation, we used the technique of Eisenstein (78) (illustrated in Figures 4.15–4.17), which demonstrates the use of this technique on actual x-ray films. Figure 4.16 reveals the sagittal diameter of a well-formed canal. According to Epstein et al. (72), the sagittal diameter of a good-sized canal is equal to one half of the diameter of the vertebral body. Application of the Eisenstein technique in Figure 4.17 reveals stenosis of the L5 level, because the sagittal diameter of the canal measures less than 12 mm; this underdevelopment can be seen by scanning the radiograph even if one does not measure the diameter. Remember, the L5–S1 intervertebral foramina are the smallest in the lumbar spine and that the size of the L5 nerve root exiting through them is the largest of the lumbar cauda equina. Rabinovitch (79) observed that the L3 through S5 nerve roots are less mobile than those above these levels, making these nerve roots more susceptible to compression by disc protrusion and osteophyte formation than those in the levels above. According to Hadley (80), the lumbar nerve roots occupy from 17 to 25% of the upper aspect of the foramina. Epstein et al. (72) found that intervertebral foramina in normal cadavers have a sagittal diameter approximately equal to that of both the foramen and neural canal, but they are consistently 2 to 3 mm less in the lower three lumbar segments, where the nerve roots are larger.

Eisenstein (81) measured the sagittal diameters of 2166 lumbar vertebrae of 433 adult skeletons and found the overall lower limit of normal sagittal diameter to be 15 mm. Of the 2166 vertebrae, 6.3% showed midsagittal stenosis, with none less than 11 mm. Midsagittal stenosis was twice as frequent as other types of stenosis. Eisenstein felt the structural reason to be an increase in the interlaminar angle (shortening of the laminae) rather than a shortening of the pedicle.

To evaluate the width of the spinal canal when diagnosing

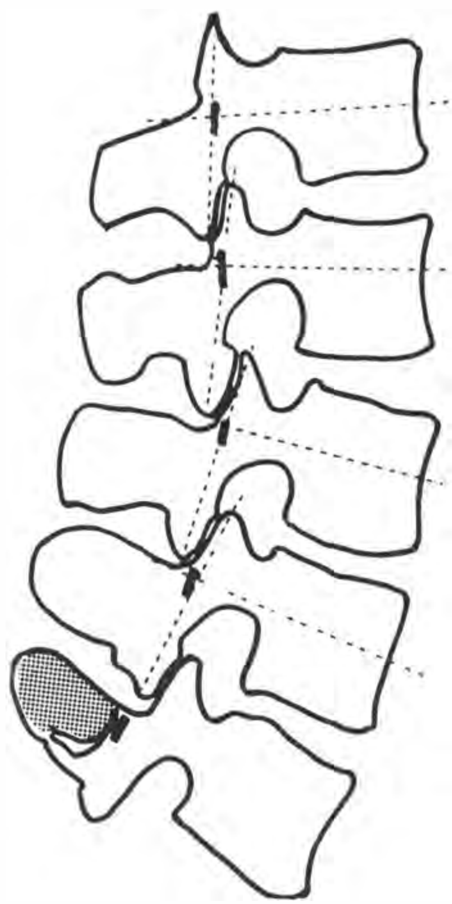

Figure 4.15. Tracing of a radiograph showing the method of locating the posterior border of the spinal canal. The posterior border of the canal at the fifth lumbar vertebra is consistently more posterior than is expected. (Reprinted with permission from Eisenstin S. Measurements of the lumbar spinal canal in 2 racial groups. Clin Orthop 1976;115:43.)

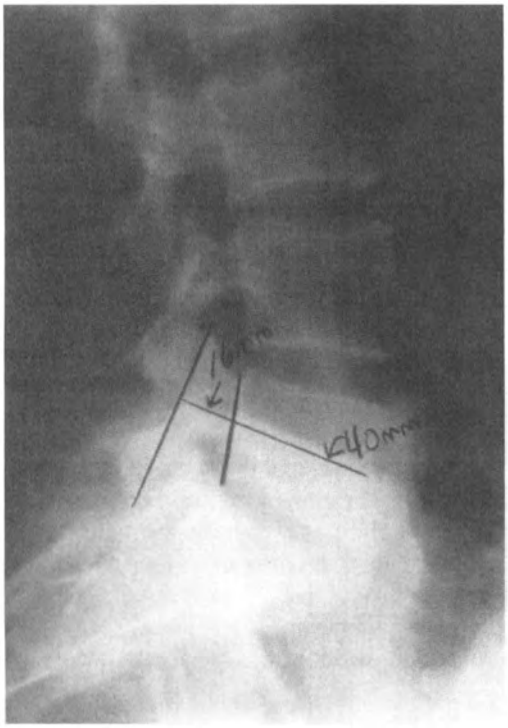

Figure 4.16. Radiograph demonstrating a well-developed sagittal diameter of the vertebral canal.

spinal stenosis a study was conducted of 91 patients who were more than 59 years of age when undergoing myelography. Using a sagittal diameter of 11 mm as the borderline value for the diagnosis of spinal stenosis, it was found that 31 of the 66 patients with spinal claudication, suspicion of spinal claudication, and sciatic pain fulfilled this criterion, and 3 of 25 of the control group and those with atypical symptoms had a sagittal diameter of 11 mm or less. Five patients showed a complete block on the myelogram, and all of them had a typical spinal claudication. The spinal canal narrows with age in asymptomatic patients as well, and the myelographic finding of stenosis in elderly patients is not always indicative of a clinical diagnosis of spinal stenosis (82).

In a prospective study, the incidence, causes, and management of atypical claudication were investigated. All patients were clinically assessed, with Doppler ultrasound studies and radiographs performed on the lumbosacral spine; some had epidural injections, myelography with computerized axial tomography, and arteriography. The incidence of atypical claudication was low—13% of all claudicants. Although difficulties in diagnosis were encountered, spinal and arterial causes were found to have an approximately equal incidence. Only one patient had a definite central spinal stenosis. The need for invasive investigations was low (18%) and the need for surgery was even lower (7%); *most of the patients' symptoms responded to conservative management* (83).

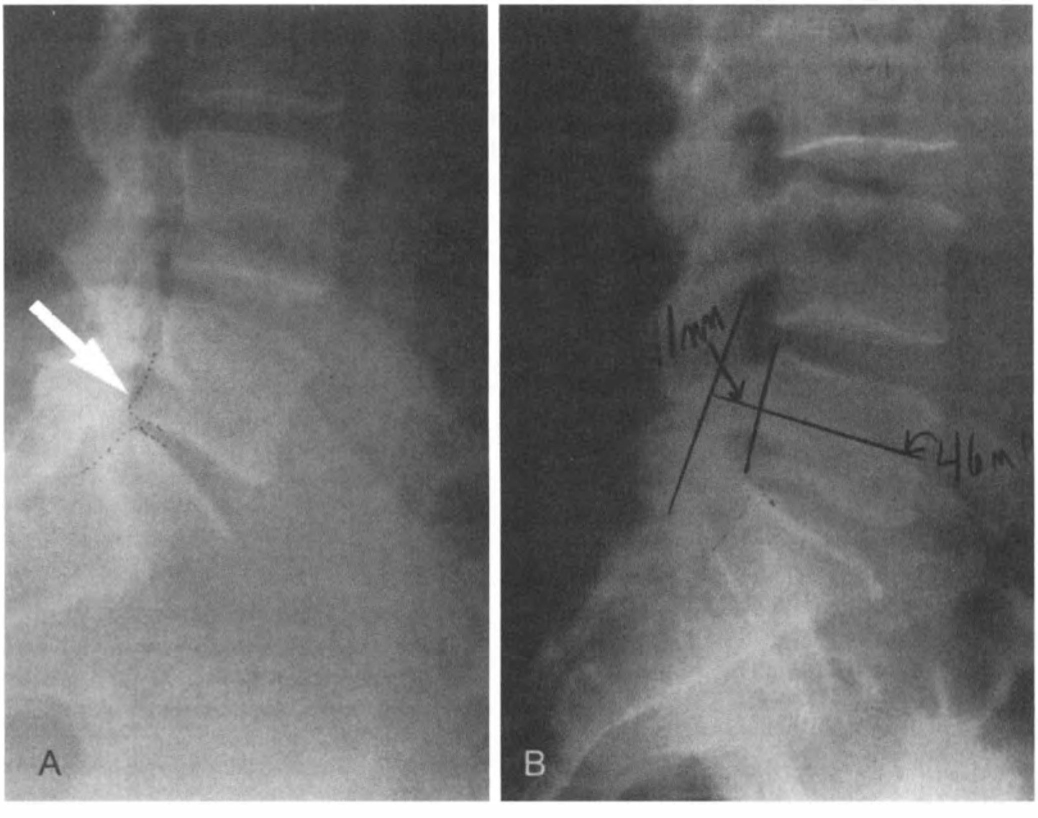

Figure 4.17. Radiographs of a stenotic vertebral canal in a patient with symptoms of intermittent neurogenic claudication. **A.** Retrolisthesis of L5 on the sacrum *(arrow)*. **B.** Stenosis measurements.

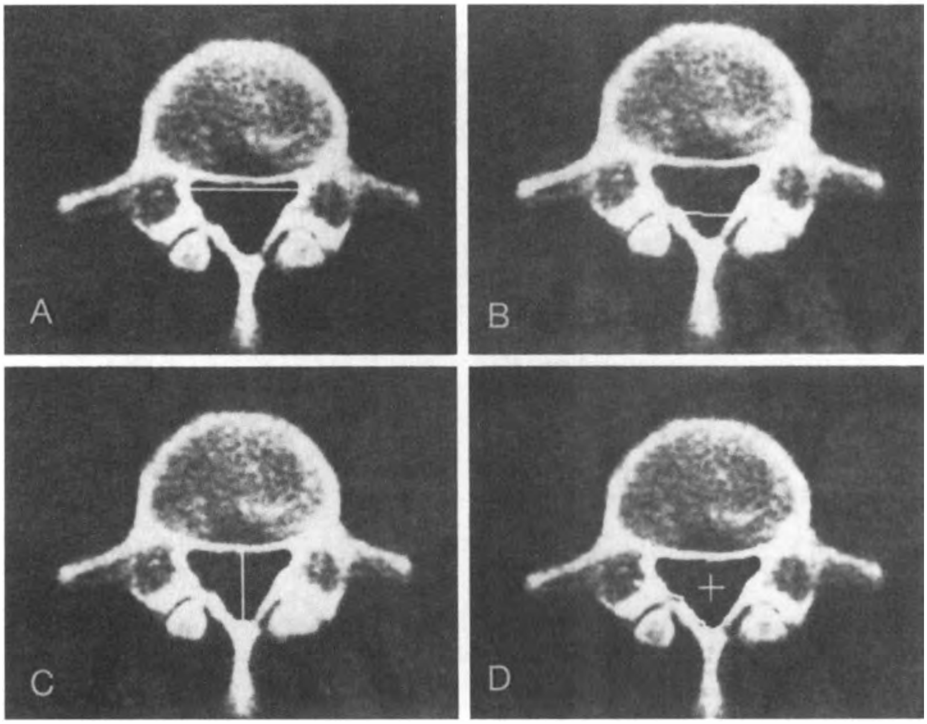

Figure 4.18. Computed tomography scan of the superior aspect of the fifth lumbar spinal canal demonstrating the measurements of interpedicular distance (**A**); interfacet distance (**B**); midsagittal diameter (**C**); and cross-sectional area (**D**). (Reprinted with permission from Kornberg M, Rechtine GR. Quantitative assessment of the fifth lumbar spinal canal by computed tomography in symptomatic L4–L5 disc disease. Spine 1985;10(4):329.)

OPERATIVE TREATMENT IN CASES OF SMALL CANALS

Interpedicular distance, interfacet distance, midsagittal diameter, and cross-sectional area at the upper aspect of the fifth lumbar spinal canal were measured from the CT scans of the spine performed during a period of 1 year (Fig. 4.18) (84). The patients were divided into four groups. Group 1 (25 patients) was the normal control group. Group 2 was composed of 29 symptomatic patients who were thought to have an L4–L5 herniated nucleus pulposus (HNP) by CT and did not undergo surgery. Group 3A was made up of 24 patients who underwent an L4–L5 discectomy and had favorable results, and group 3B (three patients) included those who failed to improve following surgery.

The patients who are likely to undergo operative treatment have a midsagittal diameter that is less than 1.6 cm and a cross-sectional area that is greater than 2.5 cm^2.

Surgical treatment is not advocated on the basis of canal size; however, a small canal size should suggest to the physician that the prognosis for resolution of symptoms is less than favorable (84).

Accuracy of Plain Film Stenosis Markings

The sagittal dimensions of five lumbar vertebra canals tended to be more shallow in patients undergoing operation for lumbar radiculopathy than in a group of controls. The more frequent occurrence of radiculopathy in patients with small canals can be explained by the fact that only a small protrusion of intervertebral disc, or any other structural abnormality, can impinge on the nerve. The sagittal diameter can be obtained easily from the lateral radiograph and, therefore, requires no invasive or expensive tests. This measurement is helpful in interpreting myelographic defects and in planning and performing operations on patients with radiculopathy (28).

An association is noted between lumbar radiculopathy and a narrow sagittal diameter of the lumbar vertebral canals. Abnormalities of intervertebral discs, vertebrae, ligaments, blood vessels, and nerves have been incriminated—individually and in combinations—in lumbar radiculopathy, but attempts to understand the pathophysiology and to improve the results of treatment have concentrated heavily on the intervertebral discs. Many pathologic, psychological, occupational, and anatomic factors that may be important in patients with lumbar radiculopathy have not been addressed.

Although often used interchangeably, the terms "spinal canal" and "vertebral canal" are not synonymous. Each person has one spinal canal bounded by bone and ligaments but many vertebral canals, one in each vertebra. Therefore, measurements based on bony landmarks apparent on radiographs, accurately speaking, are measurements of the vertebral canals (28).

Accuracy of Plain Film Stenosis Measurement

The role of the narrow lumbar spinal canal in back and sciatic pain is well established. Accuracy of measurements obtained from lumbar radiographs was therefore analyzed in lumbar spine specimens taken from 132 male cadavers. After removal of soft tissues, the same distances were measured on the bones of 80 specimens. After correction for magnification, comparison were made, and the average radiographic measurements of interpedicular distances were 2 mm greater than the osteologic ones at L3, and 4 cm greater at L5. Interarticular distances, midsagittal diameters, and pedicular lengths, on average, were 1 mm greater, and foraminal AP measurements were 1 mm less than the osteologic ones. These results confirm and amplify preliminary observations and indicate the potential value of simple measurements on lumbar spine films as an alternative to more sophisticated and expensive radiologic investigations (85).

Midsagittal diameter (MSD) and interpedicular distance (IPD) in the thoracolumbar junctional region (T10–L1) of 24 male cadaveric spines were measured both from radiographs and directly from bones after removal of the soft tissues to assess the accuracy of plain radiographs. The mean difference between bone and radiographic measurements in the IPD on different vertebral levels was 1.0 mm ($r = 0.98$) (86).

Measurements of the size and shape of the lumbar spinal canal obtained from survey lumbar radiographs have been shown to be valid as compared with bony specimens from cadavers (25).

Borderline Depth for Stenotic Canal

Radiographs were reviewed of the lumbosacral spine from 29 patients (15 men and 14 women) who had undergone lumbar laminectomy on the neurosurgical service of the Peter Bent Brigham Hospital for radiculopathy caused by protrusion of one or more lumbar intervertebral discs (28). The age and sex of each patient were recorded, along with the following measurements from each of the five lumbar vertebrae: (*a*) sagittal diameter of the vertebral canal at the midpoint of the vertebral body; (*b*) interpediculate diameter of the vertebral canal; (*c*) sagittal diameter; and (*d*) transverse diameter of the middle of the vertebral body. Measurements of the sagittal diameter of the vertebral canals were made in a manner similar to that described by Eisenstein. All radiographs were made using the standard 40-inch target film distance. Measurements were made without knowledge of which patients were the controls and which had undergone operation (28).

None of the controls had a vertebral canal that was less than 15 mm in depth—the commonly accepted lower limit of normal depths for all lumbar vertebrae—and two were exactly 15 mm. Nine of the surgical patients had a total of 10 vertebrae measuring less than 15 mm and 11 vertebrae exactly at that value.

Results showed that the mean sagittal diameters of the lumbar canals (all five were significantly more shallow in patients operated for "lumbar disc disease" than in a control group, although nearly all were within the normal range. This was determined from simple measurements taken from lateral radiographs of lumbar spines. In relatively large vertebral canals, a prolapsed or protruding disc can displace epidural fat or dura or even alter slightly the course of a nerve root but without significantly compressing it. In a small vertebral canal, little or no "extra" space is found, and therefore a small encroachment into the canal can cause the nerve to impinge against the bone. Thus, an association between the size of the canal and the occurrence of radiculopathy can help in understanding asymptomatic patient with myelographic evidence of protruding disc and symptomatic patients with small protrusions. This interpretation of the data supports Verbest's opinion that, "in the presence of a narrow although not normally narrow lumbar vertebra canal, additional slight deformities, such as posterior lipping or small disc protrusion can produce symptoms of compression" (87). In summary, less anatomic change is required to impinge on the nerve root in a small canal (28).

GRADING SYSTEM

Rothman and Glenn (88) use a grading system to evaluate pathology of the intervertebral disc, intervertebral foramen, facet joints, and vertebral canal (Figs. 4.19–4.25).

Figure 4.19 shows that this grading system is based on foraminal stenosis, disc protrusion size, and facet hypertrophy. This system allows optimal understanding of the stage of pathologic degeneration. The foraminal sagittal view demonstrates the foraminal opening and the entrapment of the nerve root by soft tissue or bony stenosis. Disc protrusion is graded by how many millimeters of bulge enters the vertebral canal. For example, a 5-mm protrusion is grade 4. Rothman and Glenn point out that a grade 4 disc or anulus protrusion has greater significance in a congenitally small spinal canal than in a large spinal canal.

The fourth row (facet joint axial view) reveals the progression of facet joint abnormality. The fifth row (central canal axial view) determines the vertebral canal shape and size and its lateral recesses. Bone or soft tissue can be responsible for the stenosis.

Specific Imaging Stenosis Causes

Disc Bulge into Vertebral Canal: Figure 4.20 shows the slight physiologic bulge of the disc, with the anulus fibrosus extending some millimeters beyond the bony end plates.

Degenerative Osteophytic Changes in the Foramen: Figure 4.21 shows marked foraminal narrowing caused by degenerative osteophytic ridging arising from the vertebral end plate.

Facet Syndrome Subluxation Changes: Figure 4.22 shows a typical facet syndrome in which the superior facet below creates foraminal encroachment as it telescopes upward. This sagittal reformation shows the marked narrowing of the L4–L5 intervertebral disc space, allowing the upward subluxation of the superior L5 facet into the neural foramen. Note how widened the facet joint space appears.

Disc Herniation: Figure 4.23 shows a grade 4 (5 mm) disc herniation at L4–L5 and a 3-mm bulge of the anulus at L5–S1.

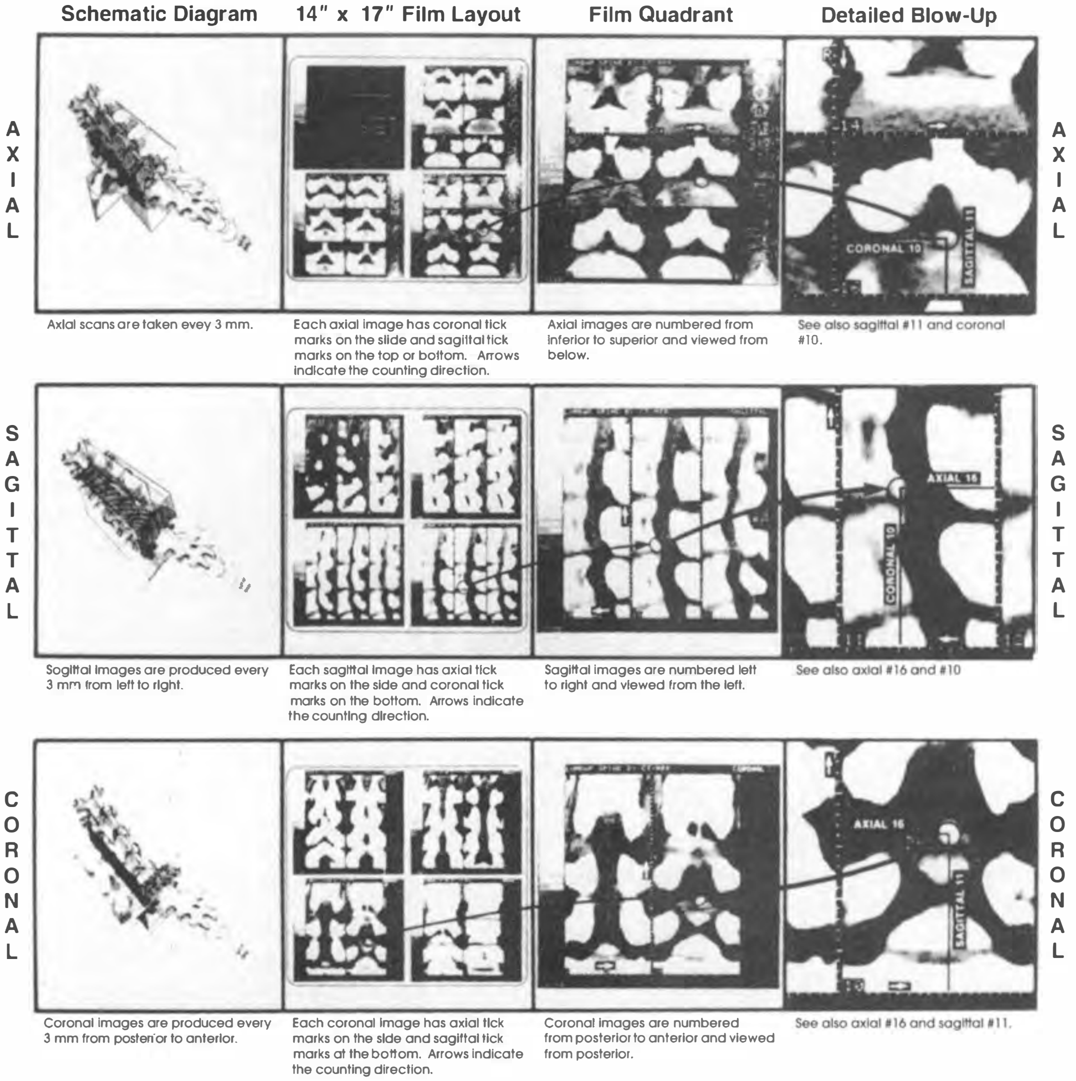

Figure 4.19. Grading system used by Rothman and Glenn to evaluate pathology of the intervertebral disc, intervertebral foramen, facet joints, and vertebral canal. (Courtesy of Steven Rothman, MD. In: Rothmann SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:28, 29.)

Stenosis: Absolute stenosis of the central canal exists when it measures 10 mm or less in its midsagittal diameter. In these cases, cauda equina syndrome can occur with no other evidence of soft tissue or bony encroachment. Relative stenosis is present when the midsagittal diameter is 10 to 12 mm (88). In that case, slight degenerative change can cause further stenosis because the reserve capacity is so reduced within the vertebral canal. Little further encroachment is required to cause symptoms.

Developmental Stenosis: Figure 4.24 shows developmental central stenosis of a 9-mm canal with a 6-mm L4–L5 disc protrusion into it. This could be symptom producing, as the potential exists for great nerve compression.

Acquired Lateral Recess Stenosis: Lateral recess stenosis caused by facet hypertrophy is seen in Figure 4.25. Here, the superior articular process has subluxated upward into the neural foramen and entraps the exiting nerve root.

CAUSES OF STENOSIS

Central Stenosis

Central stenosis is found at the intervertebral level, and it is caused by hypertrophic facets, ligamentum flavum buckling or hypertrophy, disc protrusion, and degenerative spondylolisthesis. Imaging studies (e.g., MRI or myelography) can vividly show the pathoanatomy of central stenosis. With CT, midsagittal lumbar canal diameters less than 10 mm are indicative of absolute stenosis, and less than 13 mm are indicative of relative stenosis (48).

Trefoil-Shaped Vertebral Canals

Trefoil-shaped canals are developmental in origin, and they are found at the L5 level with an overall prevalence of 25%. The

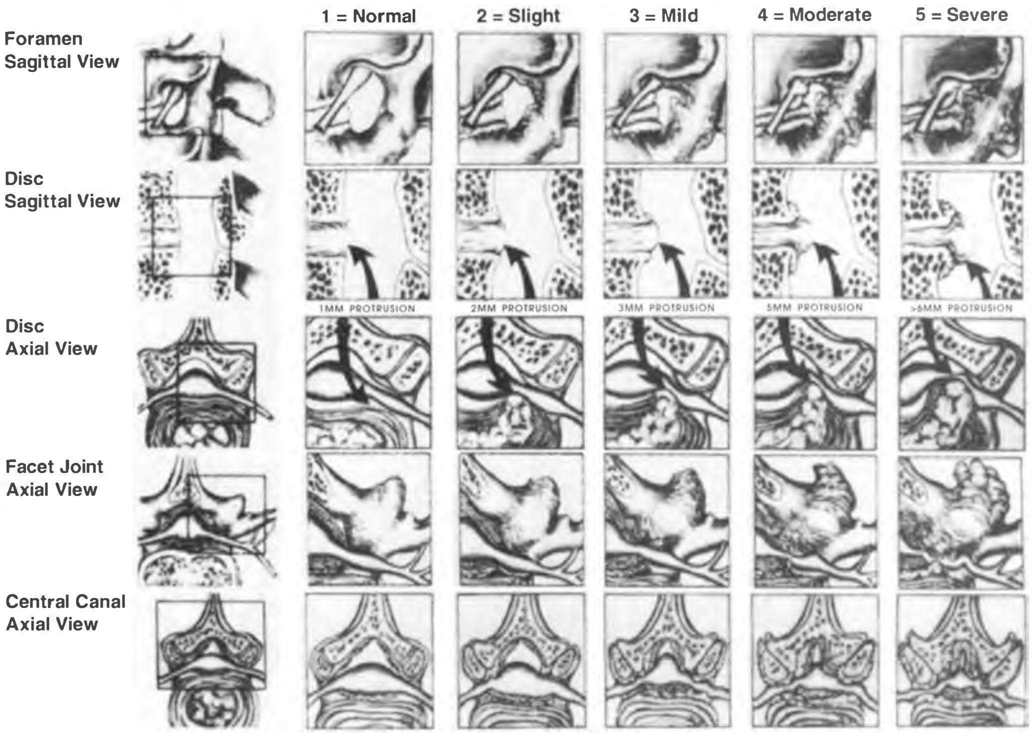

Figure 4.19.—*continued*

midsagittal diameter in the trefoil canals is significantly smaller than in the unaffected canals (89).

Stenosis Incidence in Disc Herniation

Computed tomography and transverse axial tomography (TAT) were used to study the lumbar spines of 164 patients with persistent or recurrent low back pain and/or radiculopathy. Of those patients with previous spinal fusion and those with previous discectomy, 43% and 28%, respectively, demonstrated bony stenosis of the lumbar spinal canal. Of the patients who underwent surgery for this narrowed canal, 91% showed clinical improvement (90).

Case histories are reported of four brothers with lumbosciatic syndrome caused by acute disc herniations and associated spinal stenosis. Hereditary factors, although not hitherto reported, may be implicated for these spinal lesions, as the parents had also undergone spinal operations previously (91).

Midsagittal diameter as measured by diagnostic ultrasound was smaller in patients with symptomatic disc lesions than in asymptomatic subjects, and the narrowest canals were reported in the patients who required surgical treatment (84).

Coxhead et al. (92) measured radiographs from a series of 158 patients undergoing conservative treatment for sciatic symptoms. They found an association between neurologic signs and narrowing of the interarticular distance at L4 and L5 and narrowing of the midsagittal diameter at L5.

Baddeley (93) found narrowing of the interarticular dis-

Figure 4.20. Disc bulge into vertebral canal. (Courtesy of Steven Rothman, MD. In: Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:77.)

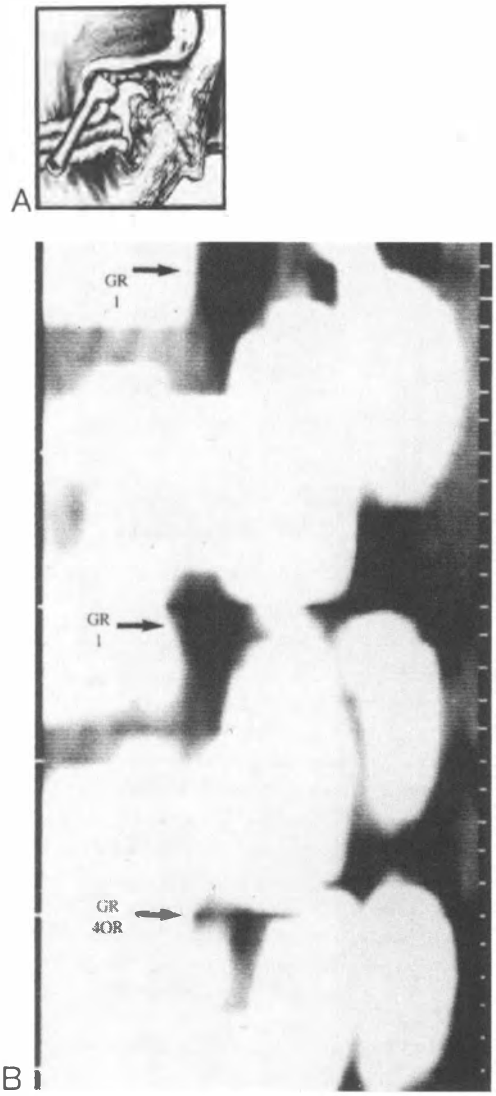

Figure 4.21. Grade 4 moderate foraminal narrowing. **A.** Diagram. **B.** Sagittal reformation on a patient with a grade 4 neural foramen. Note the degenerative osteophytic ridging arising from the vertebral end plate. The coded diagnosis is 4OR (osteophytic ridging). (Courtesy of Steven Rothman, MD. In: Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:91.)

tance, pedicular length, and midsagittal diameter in the groups of patients with disc prolapse and with the cauda equina syndrome.

Kornberg and Rechtine (84) found, in comparing normal patients with those having symptoms of herniated disc, that symptomatic patients with an L4–L5 herniated nucleus pulposus seen on CT who did not undergo operative treatment had smaller canals than did the control group. Patients requiring discectomy were found to have smaller canals when compared with the nonoperative group. Failed surgical cases were found to have smaller canals than successfully operated cases. Finally, Kornberg stated that smaller canal size should suggest a poor prognosis for these cases.

Ramani (26) found a trend toward a narrower than normal canal in patients with prolapsed discs. He concluded that, in patients with prolapsed lumbar discs, the canal tends to be narrower than normal, and that such narrowing enhances the effect of any disc protrusion, leading to severe symptoms of back and leg pain. He used plain film lateral radiographs to measure the AP diameter of the spinal canal from the midline of the back of the vertebral body to the base of the opposing spinous process. Ratios of body:canal were calculated, with 1:2.5 being normal and 1:4.5 being stenotic.

Free Fragments As Cause of Stenosis

Schmorl examined, in detail, the spines from 10,000 autopsies, and Andrae further examined some of Schmorl's material (368 spines). Findings were that 11.5% of the male and 19.7% of the female spines had a posterior prolapse of intervertebral disc beneath the posterior longitudinal ligament, and

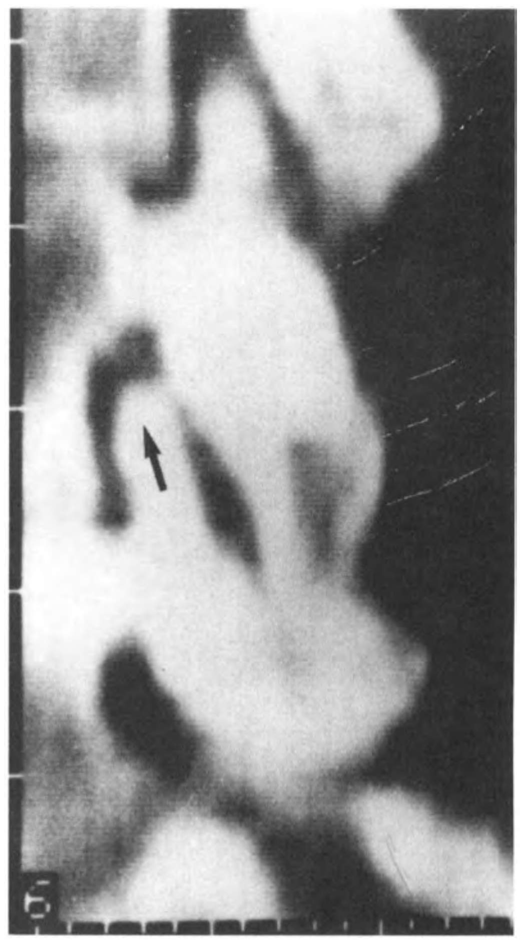

Figure 4.22. Foraminal encroachment due to upward subluxation of a facet. Sagittal reformation reveals a narrowed L4–L5 intervertebral disc space. The superior facet of L5 is herniated upward into the neural foramen (*arrow*). The facet joint space is abnormally widened as well. (Courtesy of Steven Rothman, MD. In: Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:93.)

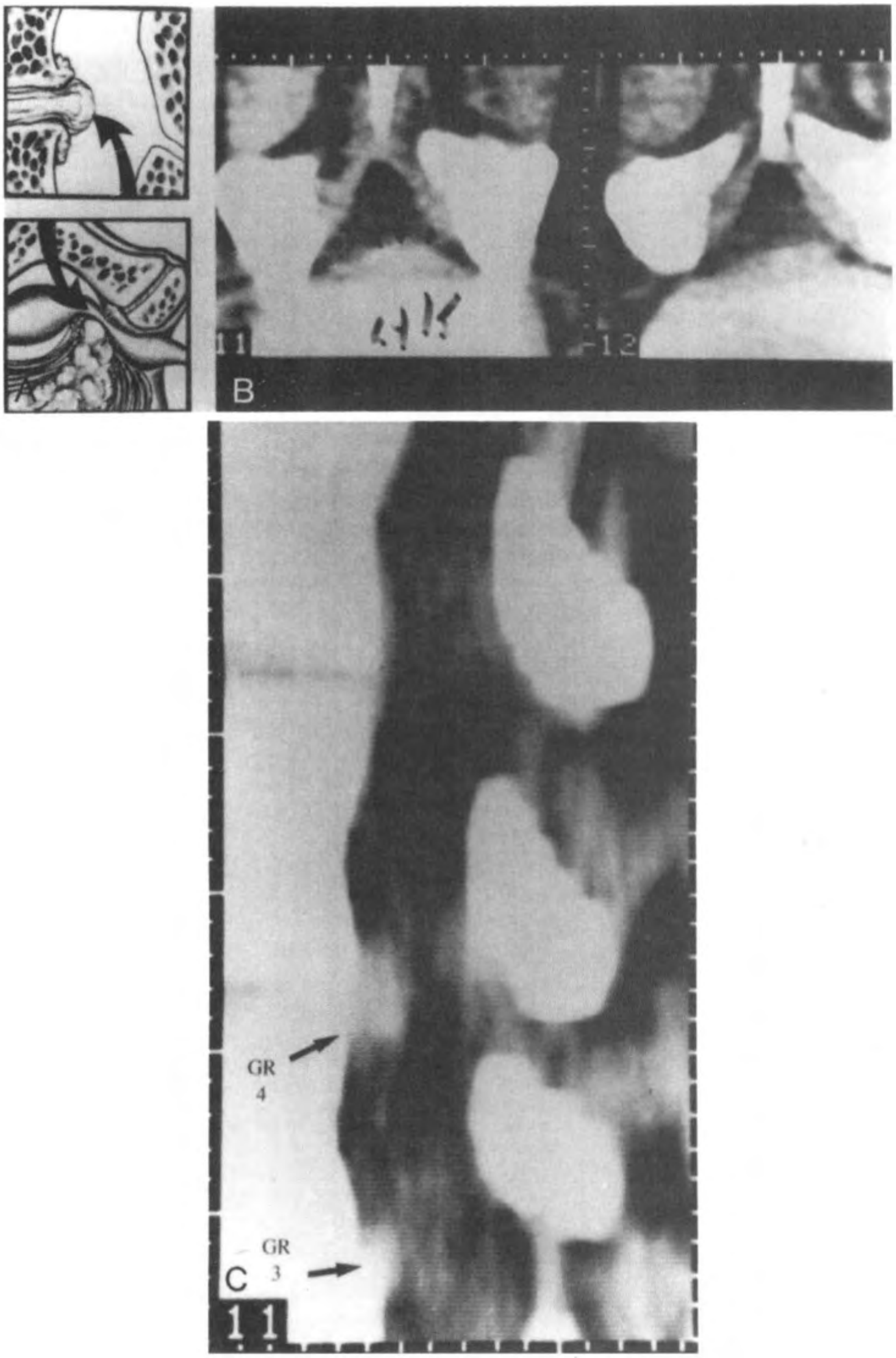

Figure 4.23. Grade 4 disc herniation. **A.** Sagittal and axial diagrams of a 5-mm disc herniation. **B.** Axial scan demonstrates a 5-mm central herniated disc. **C.** Sagittal reformation demonstrates a 5-mm L4–L5 disc herniation and a 3-mm bulge of the anulus at L5-S1 (*arrow*). (Courtesy of Steven Rothman, MD. In: Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:97.)

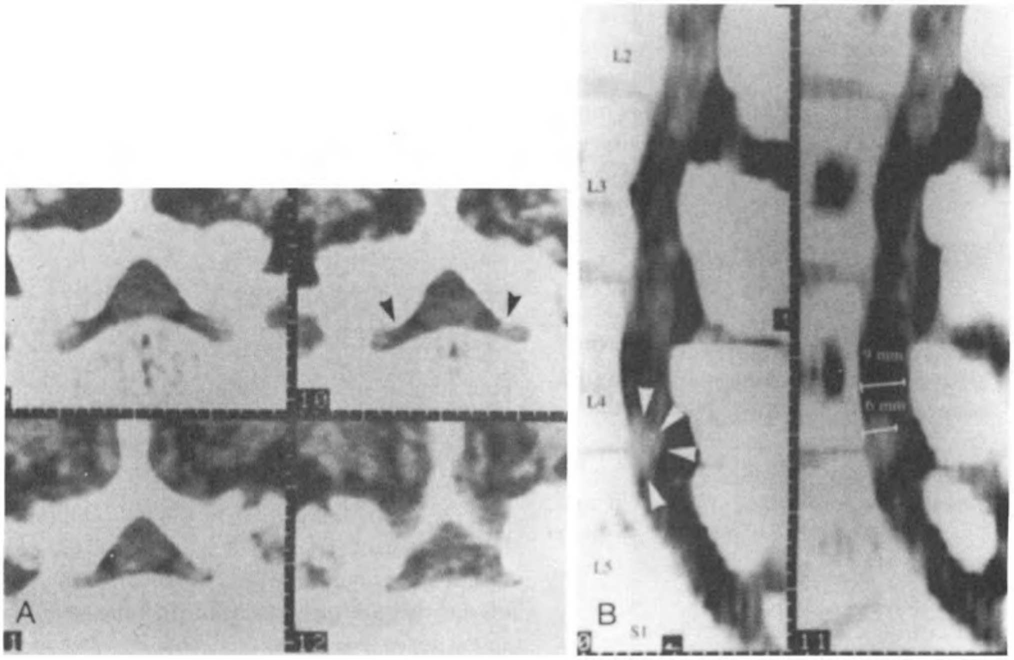

Figure 4.24. Developmental central stenosis. **A.** A sequence of axial scans demonstrates congenitally short pedicles and lateral canal indentation (*arrowheads*) by prominent superior articular processes and lamina. **B.** Midsagittal reformation demonstrates a narrow spinal canal and an L4–L5 disc (*arrowheads*). (Courtesy of Steven Rothman, MD. In: Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:199.)

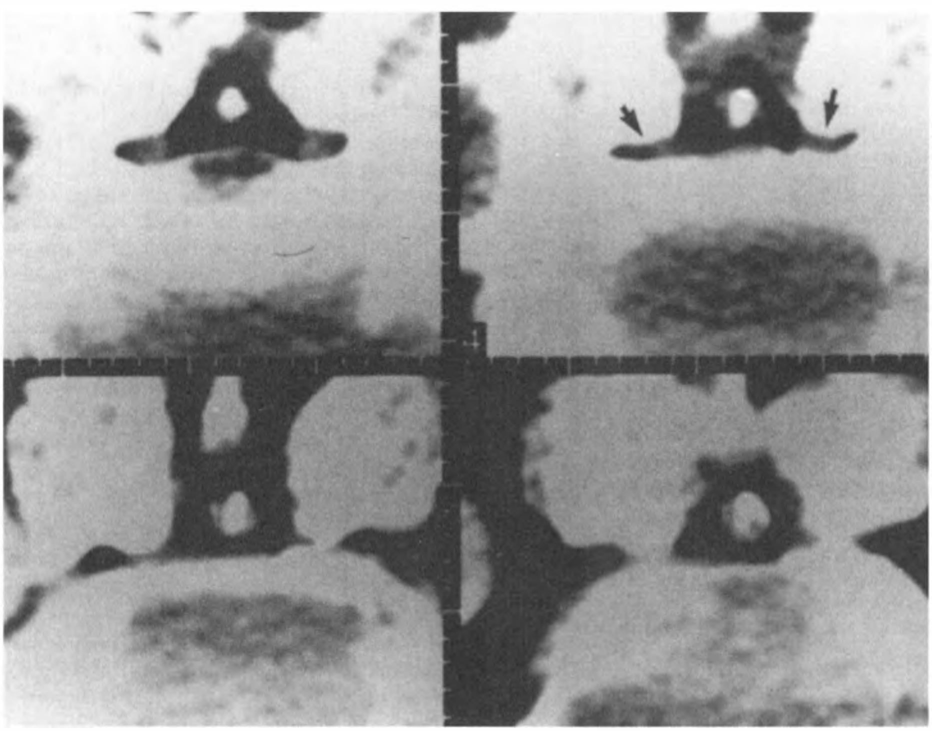

Figure 4.25. Lateral canal stenosis (subarticular recess stenosis). Axial soft-tissue views demonstrate prominent lateral subarticular stenosis. The descending roots are compressed between the superior articular process and the disc space (*arrows*). (Courtesy of Steven Rothman, MD. In: Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:205.)

more than one half of the spines with this prolapse had more than one. The percentage that was symptomatic was not known, but if the results can be extrapolated to current, living populations, then relatively few herniations become symptomatic or, at least, sufficiently symptomatic to require surgical attention (28).

Ligamentum Flavum Hypertrophy in Stenosis

Ligamentum flavum hypertrophy is significantly more often encountered in patients with spinal stenosis as seen on CT scans, and pathologic and immunohistochemical studies. This thickening is by three modes:

1. Fibrocartilage change caused by proliferation of type II collagen
2. Ossification
3. Calcium crystal deposition

It is important that hypertrophied ligamentum flavum be removed completely from the medial side of the superior facet in the capsular portion to relieve stenosis (94).

Calcium Pyrophosphate Dihydrate Crystal Deposition

Calcium pyrophosphate dihydrate crystal deposition in the ligamentum flavum occurred in 24.5% of surgical patients and may indeed be associated with the thickening of the ligament (95).

Ligamentum Flavum Bulging

In spinal stenosis, fibrotic changes, chondroid metaplasia, and calcification reduce the elasticity of the ligaments, which may thus bulge into the spinal canal in the standing position even if their thickness is normal.

In lumbar spinal stenosis, ligamenta flava play a major role in the compression of the nerve structures in the standing position and extension of the lumbar spine. It is still unclear whether the ligaments bulge into the spinal canal because they are thickened or they are simply pushed into the bulging position by hypertrophied articular processes (96).

Ligamentum Flavum Fibers Attach to the Facet Capsule

The fibrous capsule is thick in the dorsal portion of the facet joint, and its outermost fibers are intimately interwoven with the multifidi muscle's insertion on the lamellary process of the vertebra. The presence of elastic fibers appears to increase in the transition zone between the capsular ligament and the ligamentum flavum. Elastic fibers from the ligamentum flavum are particularly abundant near the superior and inferior ends of the joint (97).

Ligamentectomy for Central Stenosis

Degenerative central lumbar stenosis and complete myelographic block of the cauda equina by thickened ligamentum flavum in normal canals can cause symptoms. The dural sac can be decompressed by selective resection of the ligamentum flavum, and bilateral ligamentectomy can be performed via unilateral laminotomy (98).

TRANSFORAMINAL LIGAMENTS CAUSE STENOSIS

Four lumbar spines, including T12 and in one case T11, were obtained from embalmed cadavers and carefully dissected to expose the contents of the intervertebral foramen.

Transforaminal ligaments were found to be present at the exit zones of 71.4% of lower thoracic and lumbar intervertebral foramina. Figure 4.26 shows the types of transforaminal ligaments encountered, and the superior to inferior dimension of the compartment transmitting the ventral ramus of the spinal nerve is significantly decreased as compared with the os-

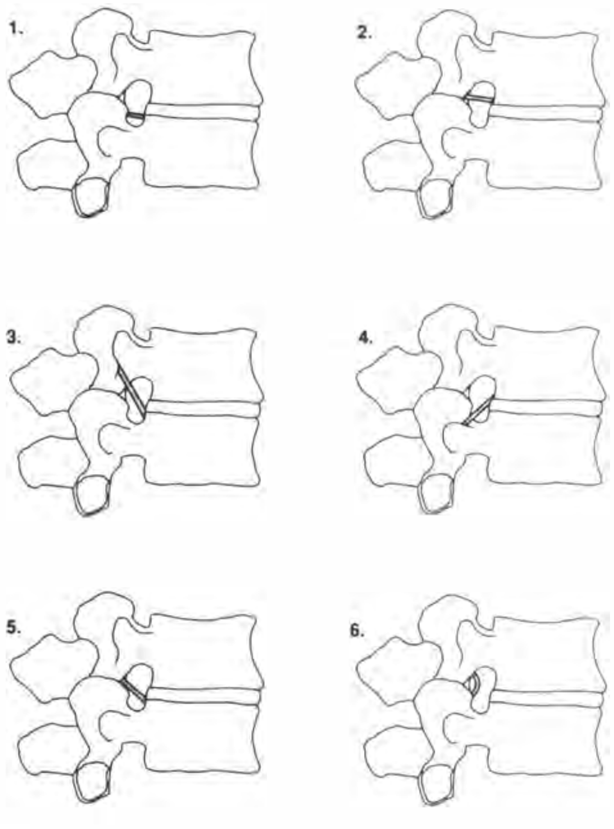

Figure 4.26. The types of transforaminal ligaments encountered in this study (n = 55): (1) inferior transforaminal ligament (n = 22); (2) superior transforaminal ligament (n = 13); (3) superior corporo-transverse ligament (n = 10); (4) inferior corporo-transverse ligament (n = 2); (5) oblique superior transforaminal ligament (n = 7); and (6) posterior transforaminal ligament (n = 1). (Reprinted with permission from Bakkum BW, Mestan M. The effects of transforaminal ligaments on the sizes of T11 to L5 human intervertebral foramina. J Manipulative Physiol Ther 1994;17(8):517–522. Copyright 1994, Williams & Wilkins.)

seous intervertebral foramen (mean decrease −31.5%). Often, less space is found at the exit zone of the intervertebral foramen for the emerging ventral ramus of the spinal nerve than traditionally thought. This decreased space may be a contributing factor to the incidence of neurologic symptomatology in this region, especially after trauma or with degenerative changes (99).

BURST FRACTURES OF THORACOLUMBAR SPINE

Five adult patients with burst fractures of the low thoracic and lumbar spines associated with intracanalar displacement showed total or subtotal resorption of the retropulsed fragment in all patients, with spontaneous remodeling of the spinal canal. Loss of mechanical loading and rhythmic respiratory oscillations in cerebrospinal fluid pressure are both important factors in the mechanism of bone resorption (100).

DEGENERATIVE SPONDYLOLISTHESIS

Fifty percent of patients with bilateral claudication have a degenerative spondylolisthesis, with a second level of more proximal stenosis (101).

Case 1

A 71-year-old woman complained of weakness of the calf muscles on walking with aching of the low back and posterior thigh muscles. In examining her, it is discovered that she cannot walk on the right toe because of weakness of the right gastrocnemius muscle and the right Achilles reflex is diminished compared with the left side.

Figures 4.27 to 4.29 are imaging showing in Figure 4.27 degenerative spondylolisthesis of L4 on L5 (*arrow*). Figure 4.28 points out that the facet joints at L4–L5 and L5–S1 are sagittal and that the L2–L3 disc is degenerated as well. Figure 4.29 shows vacuum change within the disc with extensive degeneration and internal disc disruption. Note the facet hypertrophic changes narrowing the lateral recesses and osseoligamentous canals, (*arrow*), whereas the ligamentum flavum hypertrophy (*arrowhead*) creates posterior canal stenosis by encroaching on the cauda equina.

This patient underwent decompression laminectomy and I videotaped the surgery showing that this thickened ligamentum flavum is firm and difficult to remove. Following removal of the ligamentum flavum, the patient had excellent relief of her symptoms.

Spondylolisthesis Slippage

Preoperative spondylolisthesis and a postoperative change in spondylolisthesis portend a poor outcome. Patients with mild preoperative spondylolisthesis develop a larger slip after the procedure than do those with no preoperative slip. Women and patients with preoperative spondylolisthesis may require changes in existing treatment modalities to improve outcome or alterations in long-term expectations after lumbar decompression for stenosis (102).

Figure 4.27. L4 degenerative spondylolisthesis on L5 is seen with posterior disc space narrowing, which represents instability of the disc.

Figure 4.28. The L4–L5 and L5–S1 facet joints are bilaterally sagittal and the L2–L3 disc space is narrowed with degenerative changes noted.

Figure 4.29. Axial CT scan shows vacuum change within the L4–L5 disc, which represents internal disc disruption and instability. Also note the ligamentum flavum hypertrophy (*arrowhead*) and the lateral recess stenosis due to facet hypertrophic changes (*arrow*). Note the stenotic space for the cauda equina in a patient with progressive neurologic deficits.

Figure 4.30. Dextrorotatory scoliosis of the lumbar spine is seen with extreme L2–L3 rotational and lateral listhesis subluxations at the apex of the curve. The L1–L2 disc space is markedly narrowed with vacuum change (*arrowhead*). The L4–L5 level shows indentation of the dye filled subarachnoid space by disc herniation that amputates the exiting nerve root (*arrow*).

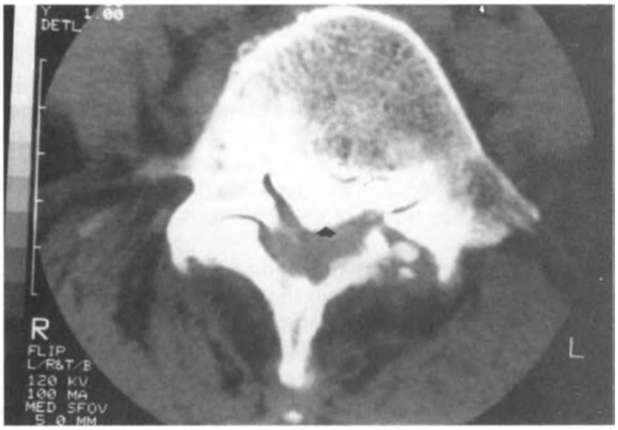

Figure 4.31. Note the distortion of the myelographically enhanced cauda equina by the rotational subluxation of the vertebra (*arrowhead*). The facet joints disclose this rotation subluxation with degeneration within the facet joints.

Degenerative Lumbar Scoliosis Causes Unilateral Claudication

In unilateral claudication, 50% of patients have a degenerative lumbar scoliosis, with central stenosis at the apex of the curve and an asymmetric distal root canal stenosis (60). Two cases of degenerative scoliosis from my practice are presented. Figure 4.30 shows dextrorotatory scoliosis of the lumbar spine with extreme right lateral listhesis subluxation of L2 and L3 at the apex of the scoliosis. Note the indentation of the right L4–L5 dye-filled subarachnoid space by disc herniation that amputates the right L5 nerve root (*arrow*). Note also the extreme L1–L2 degenerative disc disease with vacuum change within the nuclear material of the disc (*arrowhead*).

I have found these type of scoliotic degenerative spines in late middle-aged and elderly patients to be resistant to vector or forceful adjustments. The best care, in my opinion, has been gentle distraction with lateral flexion added to the distraction position of the lumbar spine. Careful tolerance testing of the patient needs to be done prior to applying distraction adjustments. Side posture adjustments in these patients can be met with resistance and pain. This is one condition in which I feel the best adjustment therapy is distraction adjustment.

A second case of degenerative scoliosis with stenosis of the lumbar spine is shown in Figure 4.31 (*arrow*), in which the axial myelographically enhanced CT scan shows distortion of the cauda equina caused by the rotational subluxation of the lumbar vertebra. In Figure 4.32, note the indentations into the anterior dye-filled thecal sac (*arrows*) caused by the combined discal bulge and rotational subluxation deformation of the dye-filled column. Tractioning of the cauda equina can occur when such scoliosis deformation takes place.

Such conditions are again treated under gentle distraction with mild derotation of the scoliosis vertebral body subluxations. Lateral flexion, gently applied, can be added into the convexity of the curve according to patient tolerance. Mobility, when gently added to these scoliotic spines, can be sedating and pain relieving to the patient.

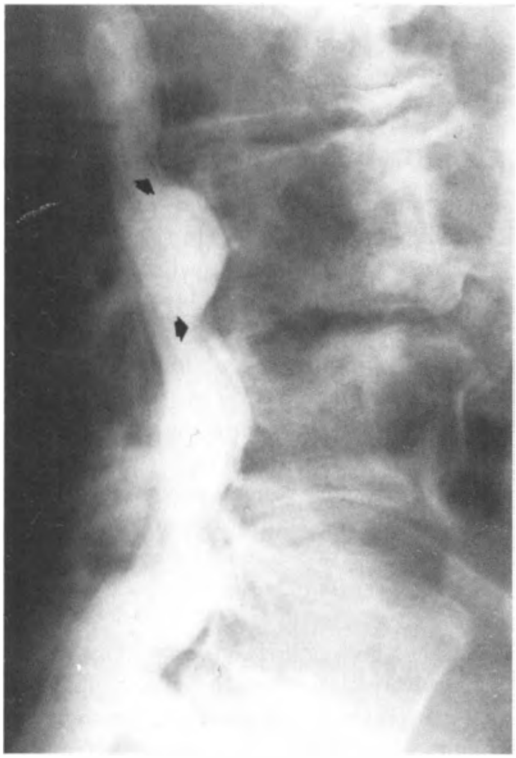

Figure 4.32. The *arrowheads* identify the indentations into the anterior dye-filled subarachnoid space by combined discal bulging and rotational subluxations of the vertebrae. Such curvature creates tractioning on the cauda equina.

Pseudogout Associated with Lumbar Spinal Stenosis

A 62-year-old man demonstrated symptoms, signs, and radiographic evidence of lumbar spinal stenosis and intraoperative pathologic findings of tophaceous deposition in the ligamentum flavum (103).

NONDISCAL CAUSES OF STENOSIS

In a consecutive series of 600 patients scanned by CT for various spinal diseases, those with low back and sciatic pain without disc herniation were selected for study. Causes of the pain proved to be joint facet degeneration (32 cases), stenosis of the neural foramina (13 cases), stenosis of the spinal canal (13 cases), lateral recess stenosis (6 cases), and spondylolisthesis (6 cases). The predominance of joint facet pathology as the underlying cause of low back and sciatic pain in the absence of disc herniation was confirmed. CT scanning of the soft tissues as well as of the skeletal structures is crucial to the causative diagnosis of the condition under study and hence to the proper planning of treatment (104).

Pagetoid spinal stenoses can occur in three stages as a progressive clinical syndrome. Several diagnostic procedures, including CT, are analyzed to introduce the concept of spinal reserve capacity (SRC). Treatment with calcitonin is recommended at the appropriate stages of the syndrome (105).

Ossification of Posterior Longitudinal Ligament in Stenosis

An enlarged and ossified posterior longitudinal ligament, a rare cause of spinal stenosis syndrome, can occupy up to 80% of the cervical spinal canal, resulting in severe, sometimes permanent, myelopathy.

Ossification of the posterior longitudinal ligaments (OPLL) has also been found in the thoracic and lumbar spine. Minorv found major differences between the clinical presentation of thoracic and lumbar OPLL and those of cervical OPLL. Thoracic OPLL is nearly always asymptomatic, and it affects women three times more often than men, whereas cervical OPLL occurs predominantly in men. The upper thoracic and midthoracic spinal area is affected most often in thoracic OPLL (106).

Dialysis

A connection between dialysis and stenosis caused by the deposition of dialysis-associated amyloid into the ligamentum flavum is reported (107). Long-term hemodialysis patients can develop cauda equina compression as the consequence of β_2 microglobulin amyloid deposition in lumbar intervertebral discs, facet joints, and ligaments. Magnetic resonance imaging is well suited to show the extent of the compression, and it supports the argument for the amyloid origin of extradural soft tissue (108).

Cauda Equina Syndrome

Cauda equina syndrome (CES) is characterized by low back pain, sciatica, lower limb motor weakness and sensory deficits, saddle anesthesia, bowel and bladder dysfunction, and occasionally paraplegia. The syndrome is classified according to onset: rapid or slow. Rapid onset CES, because of its characteristic presentation, is easily recognized. The slow, chronic progression and varying presenting signs and symptoms of slow onset CES often mimic mechanical low back pain, making the diagnosis difficult in its early stages. Anyone having multiple episodes of back pain could be suspected of having CES, because such definition appears to be diagnostic of CES from the first episode (109).

TREATMENT

Conservative Versus Surgical Care

Surgical treatment of lumbar stenosis should be considered only after an adequate trial of conservative therapy, such as exercises, supports, medications, and manipulation, has failed. Conservative therapy should be continued indefinitely as long as pain is tolerated (110). According to Wiltse et al. (111), neurologic changes alone are rarely indications for surgery.

Ben-Eliyahu et al. (112) state that recent studies show that spinal manipulation can provide relief and should be considered before surgical referral is made for decompression.

Drug Treatment

Pentoxifylline is approved by the Food and Drug Administration for the treatment in patients with intermittent claudication on the basis of chronic occlusive arterial disease of the limbs. It is not a substitute for surgical bypass or removal of arterial obstructions, but it will improve function and alleviate symptoms of the disease state. The mechanism by which pentoxifylline works is not well known, but it appears to be related to erythrocyte adenosine triphosphate (ATP) concentrations and the phosphorylation of erythrocyte membrane proteins, both mechanisms resulting in an improvement in erythrocyte flexibility. Efficacy studies indicate that pentoxifylline is significantly more effective than placebo or nylidrin hydrochloride therapy. Adverse reactions are mainly of the gastrointestinal type, and they are minimized by the use of controlled release dosage form (113).

Mesoinositol hexanicotinate (Hexopal), a derivative of nicotinic acid, has been used for some years in the symptomatic treatment of various vascular disorders including intermittent claudication. It can be concluded from a double-blind, placebo-controlled study, that Hexopal was effective, confirming previously published reports (114).

Long-term treatment produced no significant changes in intermittent claudication (IC) (140 $\pm$ 50 m), and clinical deterioration occurred in three patients. The rise in hyperemic venous resistance (VR) implies an adverse effect on blood flow properties in the ischemic limb. These findings do not support a beneficial effect on exercise tolerance, hemodynamics, or hyperemic perfusion during maintenance therapy with pentoxifylline, and they suggest a detrimental effect in some patients (115).

Side Effects of Surgery for Stenosis

Anterior vertebral body slip after decompression for myelographically verified spinal stenosis (AP diameter less than 11 mm) was studied in 45 patients (32 men and 13 women). Mean age at the time of operation was 64 years. Degenerative spondylolisthesis was found in 20 patients and acquired spinal stenosis in 25. Postoperative slipping was seen in 18 patients. An enhanced risk of further slipping was seen in degenerative spondylolisthesis, but it did not influence the result of the operation (116).

Six cases of acute postdiscectomy CES following lumbar discectomy were reviewed retrospectively in a series of 2842 lumbar discectomies over a 10-year period. Five cases had coexisting bony spinal stenosis at the level of the disc protrusion. The bony spinal stenosis was not decompressed at the time of discectomy. Inadequate decompression played a role in the postoperative neurologic deterioration. The cause of the sixth case is unknown. Bowel and bladder recovery was good when the cauda equina was decompressed early; sensory recovery was universally good, and motor recovery was poor if a severe deficit had developed before decompression. Careful review of the preoperative myelogram to rule out spinal stenosis and decompression of bony stenosis at discectomy is recommended to prevent postoperative CES. Urgent decompression of postoperative CES is advisable if compression of the cauda equina is confirmed radiographically (117).

Surgical Outcomes

Sixty-nine decompressive laminectomy patients found, at 3 to 6 years of follow-up, that 48% were very satisfied with their clinical result, 22% somewhat satisfied, 12% somewhat dissatisfied, and 19% very dissatisfied (118). Reoperations, back pain, walking capacity, and satisfaction with surgery 7 to 10 years after surgery for spinal stenosis found 23% of patients had undergone reoperation and 33% of respondents had severe back pain. Severe low back pain at the time of follow-up was strongly associated with patient dissatisfaction with the results of surgery, suggesting that patients may have expected that decompression would relieve low back pain symptoms. These results indicate that physicians should discuss with patients the differential effectiveness of surgery on back versus leg symptoms (119).

Although the 1-year follow-up results appear to be better for the surgically treated patients, few nonsurgically treated patients experienced worse pain or require subsequent surgery, and 20% of the surgically treated patients report no improvement. Therefore, the decision to undergo surgery of any type for any condition remains an individual one. Although surgery provides a greater chance for rapid relief of symptoms, such relief is likely to occur gradually without surgery (120, 121).

Unilateral Decompression and Contralateral Fusion

A new surgical technique for the treatment of lumbar spinal stenosis features extensive unilateral decompression with undercutting of the spinous process and, to preserve stability, uses contralateral autologous bone fusion of the spinous processes, laminae, and facets. Of the patients with neurogenic claudication, 69% reported complete pain relief at follow-up review. Of those with radicular symptoms, 41% had complete relief and 23% had residual pain. Low back pain was significantly relieved in 62% of all patients. This decompression procedure safely and successfully treats not only the radicular symptoms caused by lateral stenosis but also the neurogenic claudication symptoms associated with central stenosis (122).

Observation May Be an Alternative to Surgery

During 1981–1989, 32 patients (24 men and 8 women) with spinal stenosis were observed; 24 patients had neurogenic claudication, 4 had radicular pain, and 4 mixed symptoms. Nineteen patients had unilateral and 13 bilateral symptoms. All had back pain. Most nonoperated-on patients with spinal stenosis remained unchanged after 4 years and severe deterioration was not found. Observation seems to be an alternative to surgery, and immediate operation should be advised only if pain is intolerable or if neurologic symptoms develop (123, 124). Surgical treatment of spinal stenosis doesn't have an impressive

track record. Although some studies suggest a success rate as high as 80%, a meta-analysis found the overall success rate in stenosis surgery to be 64% (124).

No Correlation of Stenosis with Clinical Signs in Postoperative Patients

No correlation has been determined between the narrowest area of the dural sac, the Oswestry score, back and/or leg pain, and walking capacity. CT scanning does not provide enough evidence on which to base clinical decisions in postoperative patients with continuing symptoms. CT is a poor evaluation tool for what is essentially a soft tissue intersegmental disorder. CT scanning can adequately visualize the bony component of spinal stenosis, but not the soft-tissue component. Not everyone agrees with the view that MRI is the imaging method of choice in spinal stenosis (125).

Postsurgical Success

Only 12% of surgically treated patients for lumbar stenosis showed no bone regrowth, and the clinical results were satisfactory in most of the patients with mild or no bone regrowth and significantly less good in those with moderate or marked regrowth. The long-term results of surgery for lumbar stenosis depend both on the amount of bone growth and the degree of postoperative vertebral stability (126).

Back Surgery Satisfaction

Unsatisfactory long-term relief of symptoms after primary back operations has been reported in 15 to 40% of patients. With the number of patients needing primary surgical treatment predicted to grow by about 65% by the year 2000, an increasingly large group of failed surgical patients is likely to cause a problem in diagnostic evaluation and management for the back surgeon. Previous back surgery has a significant worsening effect on the outcome of patients undergoing surgical procedures for lumbar spinal stenosis. Patients undergoing a surgical procedure for lumbar spinal stenosis 18 months after a previous back surgical procedure obtain as good an outcome as patients who have not undergone previous back surgical procedures (127).

Patients with predominance of back symptoms are significantly less satisfied with the results of surgery than patients with predominance of leg pain. Patients with worse functional status and increased comorbidity preoperatively are also less satisfied with surgery. These results may assist clinicians in customizing patient-specific estimates of the likelihood of successful surgery (128).

Successful Surgery Reported into Eighth Decade of Life

A total of 258 consecutive decompressive lumbar laminectomies performed on 244 individuals presenting with spinal stenosis showed a high degree of success (93% pain relief, 95% return to normal activity) was achieved in the short term, which was supported by longer term follow-up data (64% pain relief, 56% activity return, 75% satisfaction).

Major conclusions arising from these data are (*a*) for all age groups through at least the eighth decade of life, decompressive lumbar laminectomy is a relatively safe operation having a high medium to long-term success rate; (*b*) lumbar instability following laminectomy is rare, even in individuals presenting prior to surgery with degenerative instability conditions; and (*c*) lumbar fusion in addition to the decompressive laminectomy procedure is rarely required for degenerative spinal stenosis (124).

Overall, 64% of the 31 patients undergoing surgical decompression for degenerative lumbar spinal stenosis had an excellent result, 17% a good result, and 19% a poor result. The authors concluded that the long-term outcome of decompressive surgery in the elderly is good; it does not differ from that reported for younger patients (129).

Bladder Dysfunction Aided by Decompressive Laminectomy in Elderly

Lumbar spinal stenosis is a common problem in elderly patients, causing typically intractable leg pain, but many patients also manifest varying degrees of bladder dysfunction. Lumbar decompressive laminectomy can have a beneficial effect on bladder dysfunction in a significant number of patients with advanced lumbar spinal stenosis (130).

Diabetes Mellitus Surgical Complications Higher

Among diabetic patients, high rates of postoperative infection and prolonged hospitalization were found compared with the rates for the control group (131). Diabetic patients who have spinal stenosis find decompressive surgery a worthwhile procedure, even in the presence of peripheral neuropathy (132).

Trumpet Laminectomy

Trumpet laminectomy is characterized by narrow laminectomy to minimize the damage to facet joints and the capsules and to facilitate complete removal of the ligamentum flavum and osseous dorsal wall of the lateral recess of the spinal canal. It has a lower incidence and lower grade of postoperative lumbar scoliosis as well as less symptom recurrence (133).

Total Laminectomy Compared with Multiple Laminotomy

Multiple laminotomy is recommended for all patients with developmental stenosis and for those with mild to moderate degenerative stenosis or degenerative spondylolisthesis. Total laminectomy is preferred for patients with severe degenerative stenosis or marked degenerative spondylolisthesis (134). Degenerative disorders of the lumbar spine in patients older than 70 years can be treated with no anesthetic complications and with 2-year results on par with those of decompressive surgery in younger patients (135).

THORACIC SPINE STENOSIS

Thoracic stenosis is defined as a narrowing of the AP diameter of the thoracic spinal canal to less than 10 mm (136). Primary thoracic stenosis becomes symptomatic when ventral spurs of the uncinate processes, discal protrusions, limbus fractures, or ossification of the posterior longitudinal ligament impinge into the canal centrally. Hypertrophied short pedicles, ligamentum flavum, or arthrotic facet joints produce comparable posterolateral spinal cord and thecal sac compression.

Symptoms of thoracic stenosis include lower extremity weakness, characterized by fatigue, leg heaviness, paraparesis, or paraplegia, and sensory complaints varying from numbness and paresthesias to anesthesia, but with preserved sphincteric function (136).

Thoracic Disc Herniation Treatment Results

Thirty-three patients were treated with microsurgical endoscopy for thoracic disc herniations using an anterior transthoracic approach. Follow-up examination revealed that all patients were independent and ambulatory and had returned to normal activities within 1 month of surgery (137).

Symptomatic thoracic discs requiring surgery are rare. Of 71 patients operated on, 37% showed evidence of antecedent trauma. Preoperative symptoms included pain (77%), motor impairment (61%), other evidence of myelopathy (e.g., hyperreflexia and spasticity) (58%), sensory impairment (61%), and bowel or bladder dysfunction (24%). Postoperative evaluation revealed improvement or resolution of pain (85%), hyperreflexia and spasticity (96%), sensory changes (84%), bowel or bladder dysfunction (76%), and motor impairment (58%) (138).

NONSURGICAL OUTCOMES

Conservative Care Is Treatment of Choice

In 145 patients with lumbar spinal stenosis, conservative treatment of physical therapy (infrared heating, ultrasonic diathermy, and active lumbar exercises) and salmon calcitonin was found to be the treatment of choice in elderly patients and in those patients without clinical surgical indications (139). Conservative treatment modalities (140) include:

- Bed rest or controlled physical activity
- Nonsteroidal anti-inflammatory drugs
- Analgesics
- Muscle relaxants
- Traction
- Manipulation
- Braces and corsets
- Exercises
- Back school
- Trigger point injections
- Physical modalities

Calcitonin Treatment of Neurogenic Claudication

Some authors reported that in patients with spinal stenosis of Paget's and non-Paget's disease who had been treated with calcitonin, some beneficial effects on neurogenic claudication were observed. Calcitonin has a powerful central analgesic effect on the receptors in the hypothalamus. A secondary response on the hemodynamics of the cauda equina is reduction of venous engorgement, and improved arterial supply. Another possible beneficial action of calcitonin might be its anti-inflammatory effect through inhibition of prostaglandin synthesis (139).

Distraction Manipulation

DuPriest (141) described the successful treatment of a patient with lumbar spinal stenosis using 12 treatments of flexion-distraction manipulation, deep tissue massage, ultrasound, therapeutic exercise, heel lift, and modification of activities of daily living. The patient was discharged from care asymptomatic in 3 weeks. Conservative treatment designed to increase lumbar flexion, thus increasing lumbar spinal canal volume, has a positive influence on the diminution of neural ischemia and its resultant neural dysfunction. Additional research is needed to elucidate these concepts.

Figure 4.33. This sagittal T1-weighted magnetic resonance image (MRI) shows extensive degenerative disc disease at all lumbar levels, more so in the midlumbar area with both anterior and posterior L2–L3 to L5–S1 disc herniations.

Lumbar Traction Found Effective for Spinal Stenosis

Lumbar traction currently performed for low back pain and sciatica management is not classically used in lumbar spinal stenosis. Lumbar tractions are well tolerated and may be effective in symptomatic lumbar spinal stenosis. Moreover, despite current opinion, lumbar tractions are not contraindicated in elderly patients who are frequently affected by lumbar spinal stenosis and in whom surgery may be problematic (142).

Case 2

[Case presentation of multiple level lumbar disc herniations with spinal stenosis in a post–decompression laminectomy patient] A 76-year-old man is seen who had undergone a decompressive laminectomy from L2 through L5 because of the chief complaint of bilateral leg pain and intermittent claudication symptoms. Four years later, he developed severe low back and buttock pain on standing for a few minutes.

Figure 4.33 reveals an MRI sagittal image showing extensive degenerative changes at all lumbar disc levels and both anterior and posterior disc protrusions at the L2–L3 through L5–S1 levels. Myelography (Figs. 4.34 and 4.35) shows the decompressive laminectomy extending from L2 through L5 (*open arrows*). Massive osteophytes have ankylosed at the right L1–L2 level and left L2–L3 level. L2 is posterior on L3 with extensive end plate sclerosis and retrolisthesis instability of L2 on L3. The L5–S1 segment shows vacuum change, and all lumbar discs demonstrate extensive degenerative changes. The myelographic column shows an anterior washboard appearance caused by the herniations of L2–L3 through L5–S1 discs (*arrows*).

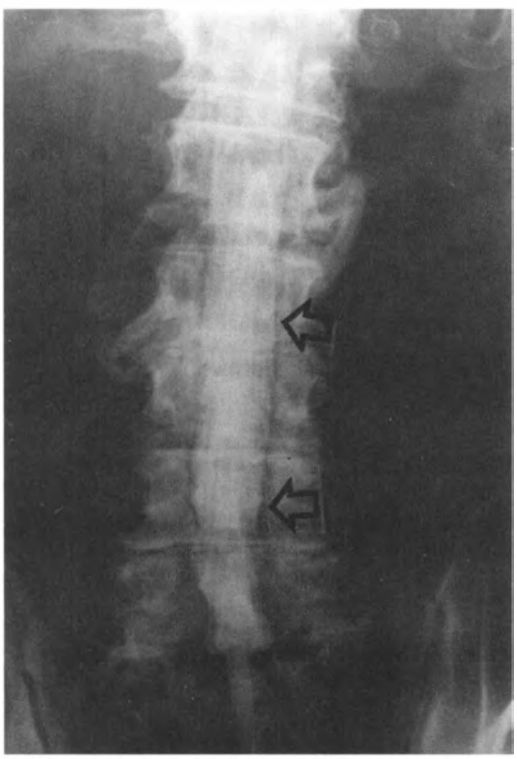

Figure 4.34. The *open arrows* reveal the dye filled subarachnoid space within the area of the decompressive laminectomy from L2 to L5. Note the massive osteophytic ankylosis at the L1–L2 and L2–L3 levels.

Myelographically enhanced CT scan (Fig. 4.36) shows the L2–L3 disc level. The cauda equina has ample space with the laminectomy decompression. Anterior, lateral, and posterior end plate hypertrophic changes are noted (*arrows*).

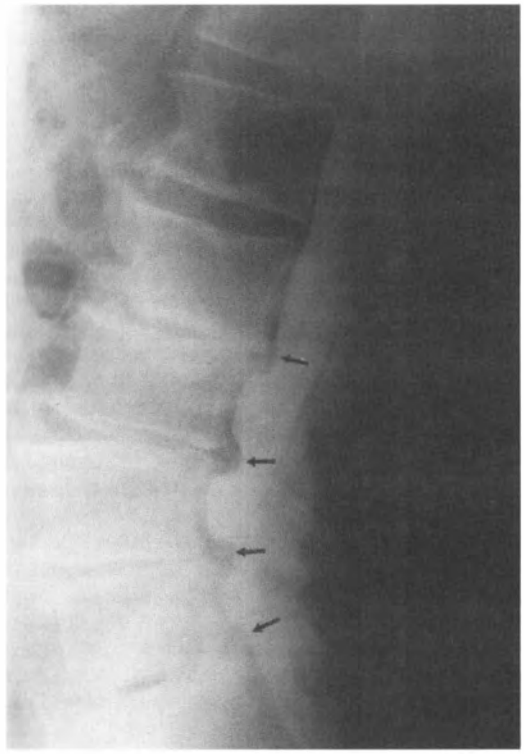

Figure 4.35. Sagittal myelographic x-ray film shows the anterior discogenic changes of the vertebral bodies with ankylosis at L1–L2. Note the indentations of the dye-filled cauda equina by posterior osteophytic changes of the vertebral bodies and disc herniations (*arrows*) to create the "washboard" appearance of the dye-filled column. Also noted is retrolisthesis subluxation of L2 and L3.

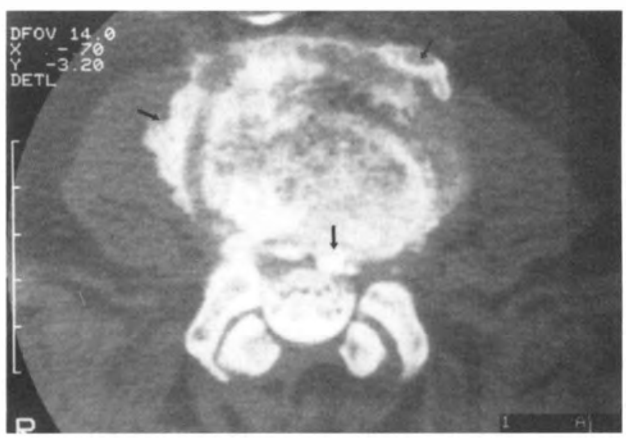

Figure 4.36. Axial myelographically enhanced CT scan at the L2–L3 level shows that the cauda equina has ample room due to the decompressive laminectomy even with the extensive vertebral body degenerative changes shown at the *arrows*.

This is an excellent example of hypertrophic bone and soft tissue disc herniation resulting in spinal stenosis with decompression laminectomy rendering good clinical relief following surgery. However, return of low back and buttock pain caused him to seek chiropractic care. Treatment of this case was gentle distraction adjustments followed by positive galvanism to the osseoligamentous canals from L2 through L5. Low back exercises consisting of knee–chest, and hamstring stretching; abdominal strengthening; adductor stretching; and abductor strengthening were performed. Excellent relief of the patient's return symptoms was obtained, showing the benefit of often required surgical and conservative chiropractic adjustments in spinal stenosis cases.

Case 3

Case 3 is of a 55-year-old white man who had low back and bilateral leg pain that was worse on the left than on the right. He also described numbness made worse on walking, leg pain aggravated by sitting, and pain in the testicles. He had been to chiropractors and was referred to us by his last doctor.

Straight leg raise was bilaterally positive at 45°, creating low back pain. Range of motion was normal. Kemp's sign was negative. Muscle strengths were normal in the lower extremities. Right ankle jerk was absent. Atrophy of the right thigh and calf was present, with the circumference being 30 mm less in the right thigh than in the left thigh and 17 mm less in the right calf than in the left calf. Milgram's sign was positive bilaterally. Nachlas', Yeoman's, and Ely's maneuvers and prone lumbar flexion all increased low back pain. Doppler testing revealed a reading of 110 mm at the left posterior tibialis (upper arm, 130 systolic) and a 50 mm reading at the right posterior tibialis. Varicose veins of the left leg were noted. Laboratory tests (complete blood count, sedimentation rate, and basic profiles) were normal. Triglycerides were 291 mg/dL (normal is 30 to 175). The prostate was normal. External hemorrhoids were present. Radiographs revealed:

1. More than 50% reduction in L5–S1 disc space height is seen, with retrolisthesis of L5 and lipping and spurring of the anterolateral body plates at L3–L4, L4–L5, and L5–S1 (Fig. 4.37).
2. Stenosis as determined by Eisenstein's measurement is evident, with the sagittal canal being 11 mm, and the body being 46 mm, the body:canal ratio being 4:1 (Fig. 4.37).

This patient had:

1. L5 stenosis with retrolisthesis subluxation of L5 on S1.
2. Discogenic spondyloarthrosis at L3–L4, L4–L5, and L5–S1.
3. An old, healed L5 disc rupture, as evidenced by an absent right ankle reflex and past untreated leg pain.
4. Intermittent claudication pain in both legs, with a marked insufficiency in the right leg where blood pressure was greatly reduced at the posterior tibialis artery. Stenosis may cause neurogenic claudication in both legs.
5. Left L5–S1 medial disc protrusion causing S1 dermatome sciatica.

Following the above diagnosis, it was decided to apply treatment four times daily at the outset, for 3 weeks. If 50% relief was obtained, both subjectively as evidenced by patient response and objectively as evidenced by tests for Kemp's sign, Déjérine's triad, range of motion, and straight leg raising, 2 more months of treatment would be given. If no relief occurred, a vascular surgeon and, possibly, a neurosurgeon would be consulted.

Cox distraction manipulation was given, followed by therapy four times daily for 3 weeks. The result was a right lower extremity blood pressure of 90 mm, which was approximately 80% the blood pressure of the left leg. The leg pain ceased and the back pain localized in the gluteus maximus muscle.

Treatment consisted of three or four distractions daily with positive galvanic current to the L5–S1 disc and B54. Tetanizing current was applied to the adductor and gluteus medius muscles. Acupressure points B24 through B31 were goaded. A belt was worn on the low back 24 hours daily. Sitting was prohibited, and exercises for the low back were given. The patient was sent home to be treated by his family chiropractor.

Prior to returning to work 3 months after the onset of treatment, the patient went through our low back pain school, where he was taught the movements dangerous to the low back, how to lift and bend, how to pick up objects from the floor or from shelves, and how to protect the back in activities of daily living.

At the end of 3 months, the patient had obtained 75% relief from pain. The major symptom was left hip and buttock stiffness on standing or walking.

Case 4

Figure 4.38 reveals a calcification projecting bilaterally from the pedicles into the vertebral canal. Helms and Sims (143) feel that these spurs most likely represent ossification of the ligamentum flavum at its point of insertion and contend that they should not be mistaken for osteophytes, free disc fragments, or fracture fragments. These ossifications are occasionally seen on CT scan, and it is noted that they are usually asymptomatic. Note that a disc protrusion is present on this CT scan.

Case 5

Figure 4.39 is a lateral radiograph showing an approximate 60% loss of vertical height of the T9 vertebral body, which occurred in this 59-year-old woman after a fall. Her pain continued, and Figure 4.40 represents the same radiograph taken 3 months following Figure 4.39. Note that the compression fracture has continued to

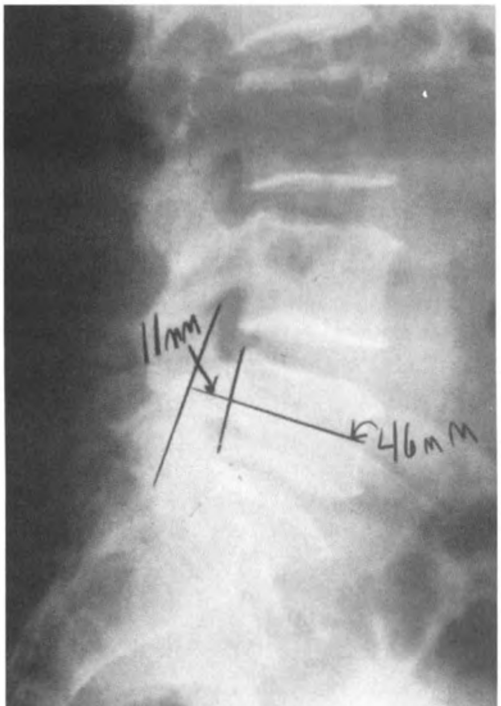

Figure 4.37. Lateral lumbar view. Retrolisthesis subluxation of L5 on the sacrum, with stenosis of the vertebral canal at L5 determined by Eisenstein's measurement.

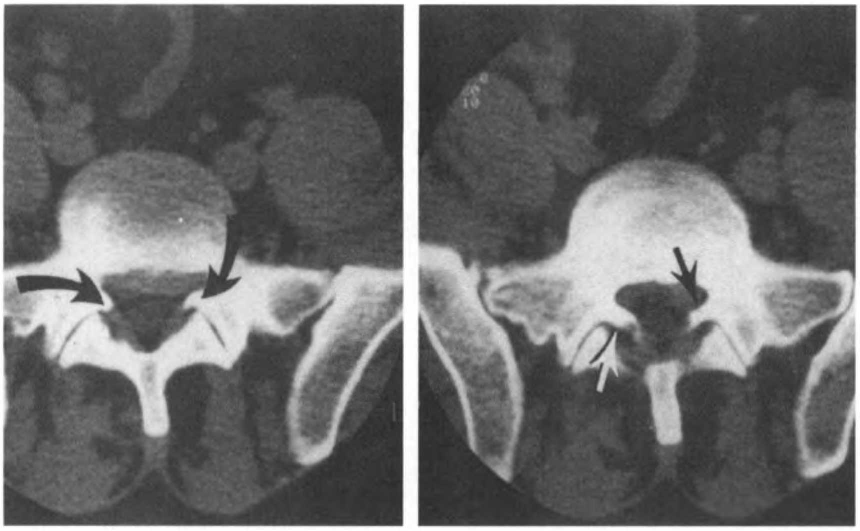

Figure 4.38. Superior facet calcifications at the attachment of the ligamentum flavum at the insertion into the facet (*arrows*).

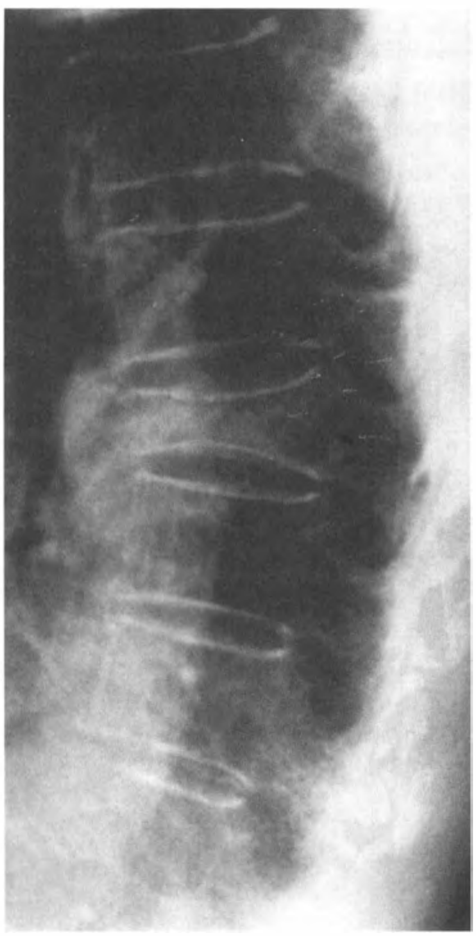

Figure 4.39. Approximately a 60% loss of height of the T9 vertebral body is seen in a 59-year-old woman following a fall. This radiograph was made the day of the fall.

Figure 4.40. Three months later, following persistent back pain, another radiograph shows progressive compression deformity of the T9 body.

deteriorate. Appropriate blood tests did not suggest any evidence of pathologic fracture. This patient became asymptomatic under conservative extension-type manipulation and physiologic rest.

This case represents the progressive collapse of a vertebral compression fracture in the months following the original injury. One must be aware of this in clinical practice, as it can explain the further pain the patient may experience within weeks following the original compression fracture. Such flexion deformity can cause narrowing (stenosis) of the vertebral canal to the point of obstructing dye flow in the subarachnoid space on myelography. Treatment of the compression defects in Cases 5 and 6 is shown in Figures 9.35 and 9.37 in Chapter 9.

Case 6

A 70-year-old man fell from a tree and sustained approximately 75% compression fracture of the L1 vertebral body. He was taken to the hospital and was catheterized because he could not urinate. He wore this catheter for 2 months and underwent prostate surgery. However, the reason for the catheter was that he could not urinate because of cauda equina compression by the narrowing of the vertebral canal following the compression fracture shown in Figure 4.41. This can be compared with the adjacent vertebra (Fig. 4.42).

When we first saw this patient, his purpose in coming was to find out whether any treatment other than a Harrington rod fusion could provide relief. Examination of this patient revealed both ankle jerks absent, whereas the patellar reflexes were +2 bilaterally. At that time, the patient was having normal urination and no other signs of cauda equina syndrome.

The cremasteric and Babinski reflexes were normal. Weakness was evident to some degree on contraction of the anal sphincter muscle, but the patient had no problem with bowel control. Hypesthesia of the S1 dermatomes was found bilaterally. In Figure 4.43, taken 7 months later, extensive ankylosis is seen of the anterolateral vertebral body plate, caused by hypertrophy and calcification. This would represent the body's own attempt to fuse this area into stability.

In considering a surgical fusion versus the body's own attempt to fuse, we consider the work of Taylor et al. (144), who studied compression fractures of the dorsolumbar spine without neurologic involvement. A long-term follow-up study was carried out on 216 patients with fractures of the dorsolumbar spine. None of these patients had neurologic impairment. The average period of follow-up was 9 years, and it was found that the functional results did not differ between patients with a single fracture and those with multiple fractures, nor could statistical clinical differences be established between patients whose fractures went on to spontaneous fusion and those whose fractures did not. Correlations could not be established for residual symptoms, reduction

Figure 4.42. Normal vertebral body and canal of the adjacent segment for comparison with Figure 4.41.

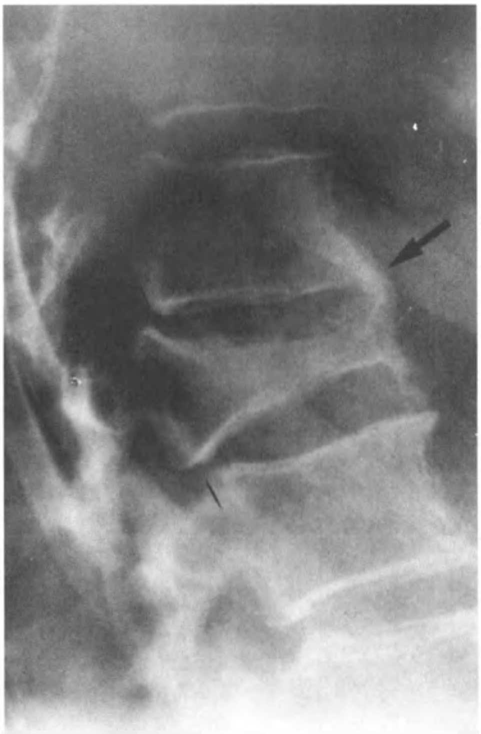

Figure 4.43. Three months after the film in Figures 4.41 and 4.42, extensive anterior ankylosis is seen, caused by calcification of the anterior longitudinal ligament and hypertrophic changes (*arrow*). The body has provided its own natural fusion at the site of instability.

Figure 4.41. Computed tomography scan reveals the impaction compression deformity of the L1 vertebral body with invasion of the vertebral canal to create stenosis of the canal and spinal cord.

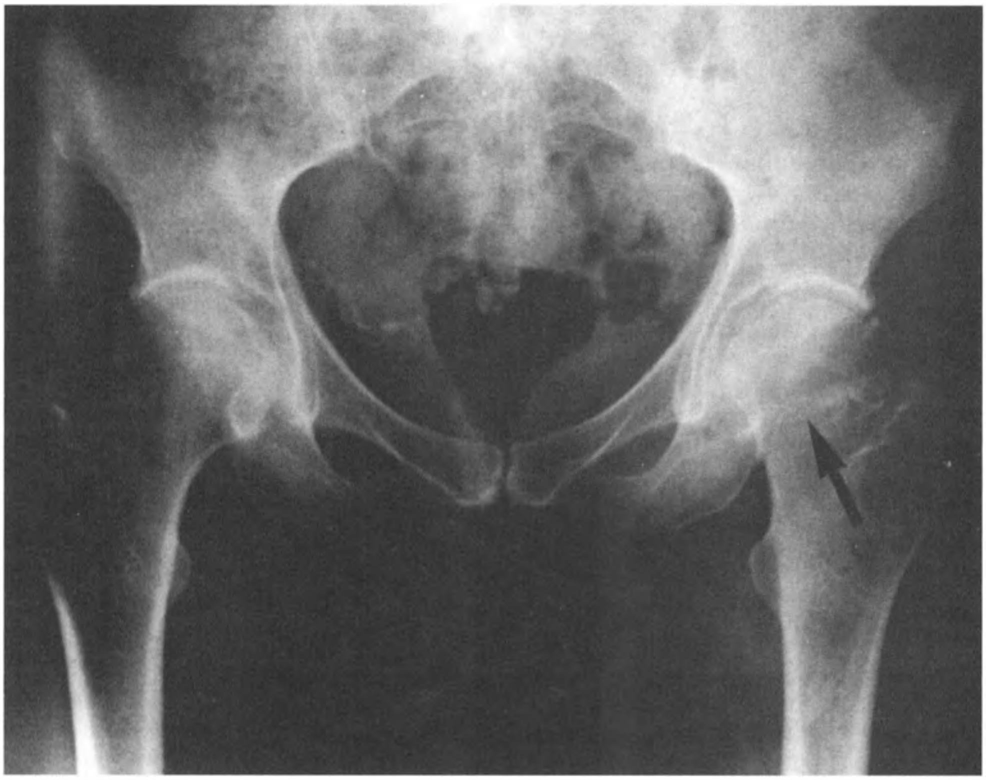

Figure 4.44. An impaction fracture of the left femoral cervical area is noted (*arrow*).

in vertebral height, encroachment on the spinal canal, and persistent kyphotic deformities. It was concluded that the nonoperative treatment of these fractures was a sound method and that attempts at reduction were not justifiable. No patient in this series had undergone a surgical procedure because of persistent symptoms.

With these thoughts in mind, we suggested to the patient in Case 6 that, because fusion was occurring and it had been 6 months since his initial fracture, he should question strongly what could be guaranteed to him through the use of a Harrington strut and fusion. He did not want the surgery. In fact, he stated that he would rather die than have the surgery done. Therefore, we treated this man, as we have other compression fracture cases, with mild flexion and extension manipulation followed by tetanizing current applied paravertebrally over the fracture site. The results of this care were persistent loss of pain in the dorsal lumbar spine and regaining of enough physiologic range of motion to be compatible with the patient's everyday living. In the past 3 years that we have followed this case, this patient has been comfortable without cauda equina symptoms.

Case 7

Figure 4.44 reveals an impaction-type fracture within the cervical area of the left femur. Figure 4.45 shows an eventual hip arthroplasty that was performed. Chiropractic involvement with this case came about because of the persistent pain in the left buttock and hip area.

Figure 4.46 is a CT scan showing ligamentum flavum hypertrophy at the L4–L5 level. Clinically it was felt that perhaps this ligamentum flavum hypertrophy was creating some degree of stenosis at this level. Flexion-distraction manipulation was given to this patient. She also attended low back wellness school and was instructed in how to prevent hyperextension motions that

Figure 4.45. A hip arthroplasty is performed on the hip joint in Figure 4.44.

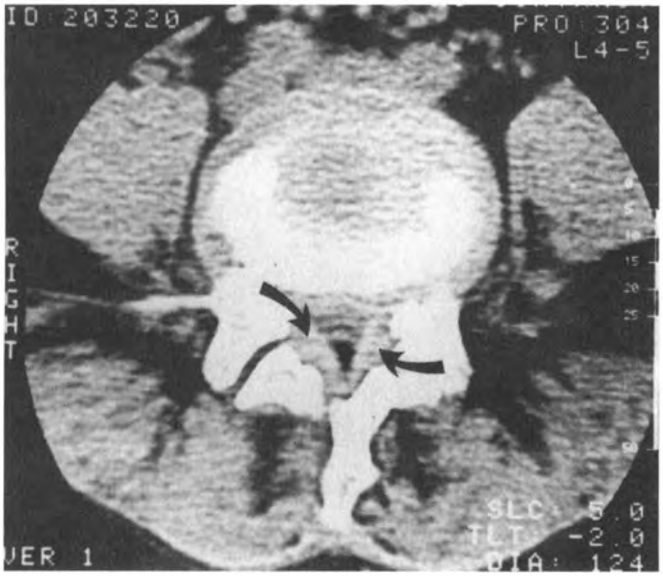

Figure 4.46. Computed tomography scan reveals ligamentum flavum hypertrophy (*arrows*), creating stenosis of the vertebral canal felt to have been responsible for the persistent pain in the left hip, which was relieved by spinal flexion-distraction manipulation.

could further aggravate her stenotic condition. Accompanying exercises in the flexion mode to strengthen abdominal muscles, stretch hamstring muscles, and maintain a slight flexion of the lumbar spine resulted in gradual relief of approximately 50% of this patient's pain.

This case is presented to show the possibility of ligamentum flavum hypertrophy as a cause of persistent hip and buttock pain that perhaps was masked by the fact that this patient had a hip arthroplasty. Further, the effects of manipulation in this type of case are demonstrated.

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The Sacroiliac Joint

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chapter 5

In seeking absolute truth we aim at the unattainable and must be content with finding broken portions.

—Sir William Osler

It has been well documented that 80% of the population suffers from lower back pain at some time in their lifetime (1). What has not been so well documented and understood is the cause of this common condition. Lower back pain (LBP) can be caused by many structures found in the skeleton, muscles, ligaments, viscera, and nerves. Yet there continues to be great difficulty in localizing the incriminating structure responsible for the generation of pain in most patients. The ever elusive definitive diagnosis in LBP patients continues to be an enigma plaguing most practitioners treating such patients. This is especially true of the sacroiliac joint and its inherent conditions, which has a controversial history ranging from being the principal cause of lower back pain to having no role at all in the generation of painful conditions.

After the seminal paper in 1905 by Goldthwaite and Osgood, the sacroiliac joint (SIJ) became regarded as the main cause of LBP (2). They claimed that "sacroiliac sprain is the common cause of low back pain." However, following the publication in 1934 by Mixter and Barr (3), attention moved away from the SIJ and began to focus on the disc and its surrounding structures. The SIJ was neglected because it is a deep-seated, complicated oblique structure, seeming to have little or no movement, as well as being difficult to access for examination (5–7).

However, with the development of sophisticated imaging procedures, examination techniques, new treatment protocols, and outcome measures, new information about the pain generators in LBP was being published. Researchers were finding that not all LBP syndromes could be simply attributed to involving the disc and facet joints (7, 8). Attention was again directed to the other structures in the low back, including the SIJ. Clinicians reported that patients presenting with SIJ pain or postsurgical pain were being effectively managed by conservative treatment directed to the SIJ (9, 10). Researchers focused on the SIJ to define better its anatomy, movement, and clinical characteristics.

This chapter thus reviews the structure, movement, clinical presentation, and management of the conditions involving the sacroiliac joint.

ANATOMY

Morphology of the Sacroiliac Joint

The sacroiliac joint is a true diarthrodial joint formed by the articulation between the sacrum and the anteromedial aspect of the ilium. Even in the mid 1700s the synovial nature of the SIJ was recognized by Siegfried Albinus and William Hunter, who first described its anatomy. The SIJ's anatomy was examined further by Albee in 1900 who dissected and analyzed 50 postmortem specimens and confirmed earlier studies (11). However, unlike the structural aspects of this joint, our knowledge of its function and biomechanics remains limited primarily because of the difficulties inherent in the analysis of motion at the articular surfaces of this deep-seated joint (see description of SIJ biomechanics below).

Anatomic Relationships of the SIJ

Located centrally in the pelvic girdle, the SIJ is designed primarily for stability and transmission of relatively significant forces during the gait cycle and, in particular, during running and jumping. The bony elements of the joint include specifically the posterolateral aspect of the sacral ala at the level of the first and second (and, occasionally, third) sacral segments and the anteromedial surface of the ilium adjacent to the posterior inferior iliac spine (PIIS). The joint can further be subdivided into two components (12, 13): (*a*) the synovial por-

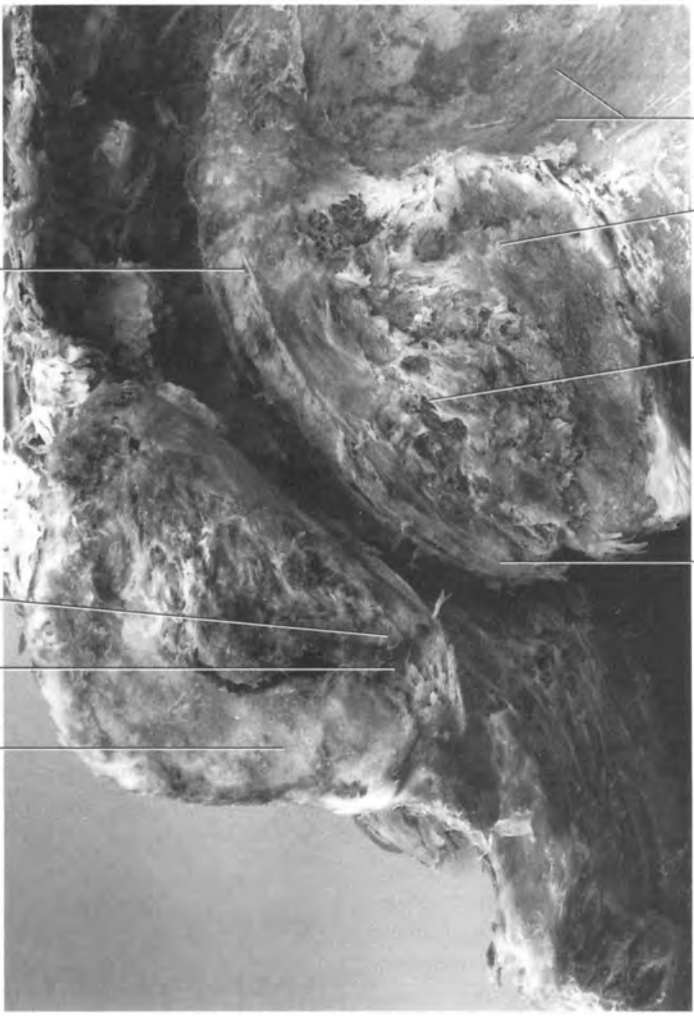

Figure 5.1. Anatomic section illustrating the synovial and syndesmotic parts of the left sacroiliac joint.

tion, which is anterior and which consists of the auricular surfaces of the sacrum and ilium; and (*b*) the syndesmotic portion, which is more posterior in position, and which consists of the roughened sacral and ilial tuberosities that attach the interosseous sacroiliac ligaments (Fig. 5.1).

The synovial auricular surface is shaped somewhat like the pinna of the external ear with a broad superior limb oriented posterosuperiorly and an elongate inferior limb oriented posteroinferiorly (Fig. 5.2). On the sacral surface the superior limb occupies approximately two thirds of the posterior sacral ala and the inferior limb extends down to the second sacral transverse tubercle of the lateral sacral crest. The most anterior part of the SIJ is formed by the apex of the convexity of the auricular surfaces at the level of the first anterior sacral foramina. Within the sacral auricular surface a central longitudinal groove roughly parallels the anterior and posterior borders of the joint. This articular groove is complementary to a bony articular ridge on the iliac auricular surface (Fig. 5.3), and it may function in an interlocking mechanism to stabilize the joint (Fig. 5.4). In older specimens the posterior rim of the inferior limb may be more ossified and may exhibit a prominent bony ridge.

In some older individuals an accessory SIJ may be present at the level of the first and second posterior sacral foramina (Fig. 5.2). When present, the iliac position of this accessory joint is adjacent to the posterior superior iliac spine (PSIS) and may extend onto the iliac tuberosity (Fig. 5.3). It has been postulated that accessory SIJs may be more common in quadrupeds and may develop in response to limited hip extension. Similarly, human accessory SIJs appear to be more common in individuals who use a wheelchair or sit for prolonged periods (15–17).

The articular surfaces of the SIJ are unique with respect to the type of cartilage that lines them. The sacral auricular surface is lined by a 3 mm layer of hyaline cartilage, typical of synovial joints in general, and this layer is approximately three times thicker than that on the iliac side (11, 13, 18–20). Histologically, this hyaline cartilage is homogeneous and is composed of large, round, paired chondrocytes distributed throughout the chondroitin sulfate matrix and arranged in cell columns parallel with the articular surface (11). In contrast, the iliac auricular surface is lined by a thin 1 mm layer of fibrocartilage characterized by smaller, spindle-shaped chondrocytes embedded in a collagenous matrix. Interestingly, the chondrocytes are

again organized into columns of cells but these are oriented perpendicular to the articular surface on the iliac side (21). As in hyaline cartilage, the extracellular matrix of this fibrocartilaginous layer is composed primarily of chondroitin sulfate as well as other glycosaminoglycans, but it has a much higher density of type II collagen fibers as is characteristic of fibrocartilage in general (9, 13, 19). The two kinds of articular cartilage present on opposing sides of this joint suggest a disparity in function between the two articular surfaces; however, this possibility remains poorly investigated.

The syndesmotic portion of the SIJ, which is more posteriorly located, consists of the interosseous sacroiliac ligaments (ISL). The superior, middle, and inferior sacral fossae on the sacral side and the iliac tuberosity on the iliac side form the bony attachments for these ligaments (22–25).

Structurally, the ISL consist of short fibers in the deepest part of the joint and these fibers become progressively longer the more posteriorly and superficially they are found.

The longest and most superficial part of the ISL blends imperceptibly with the fibrous capsule of the SIJ. The ISL is considered to be the strongest ligament in the body and it is thought to maintain the stability of the SIJ posteriorly by resisting posterosuperior gapping. Functionally, it is believed that the point where the iliac tuberosity meets the middle sacral fossa forms an axial articulation (26) with the iliac tuberosity acting as a pivot point during rotary sliding movement along the apposed articular groove and ridge on the sacral and iliac sides, respectively (Fig. 5.4).

Phylogenetic Differences

Interesting morphologic and functional differences exist between the SIJs found across the animal phyla. For example, fish have fins that are not connected to the vertebral column, but instead are connected to each other by a primitive pelvic symphysis (Fig. 5.5). The amphibian pelvic girdle is connected to a sacral rib. The SIJ of quadrupedal animals is more like that of bipeds, such as humans, but it does have unique differences. The quadruped SIJ is completely syndesmotic, and it is positioned rectangular to the spine. In bipeds, the SIJ is thought to bear twice the load as the trunk owing to gravitational influences when compared with that of quadrupeds (27). Conse-

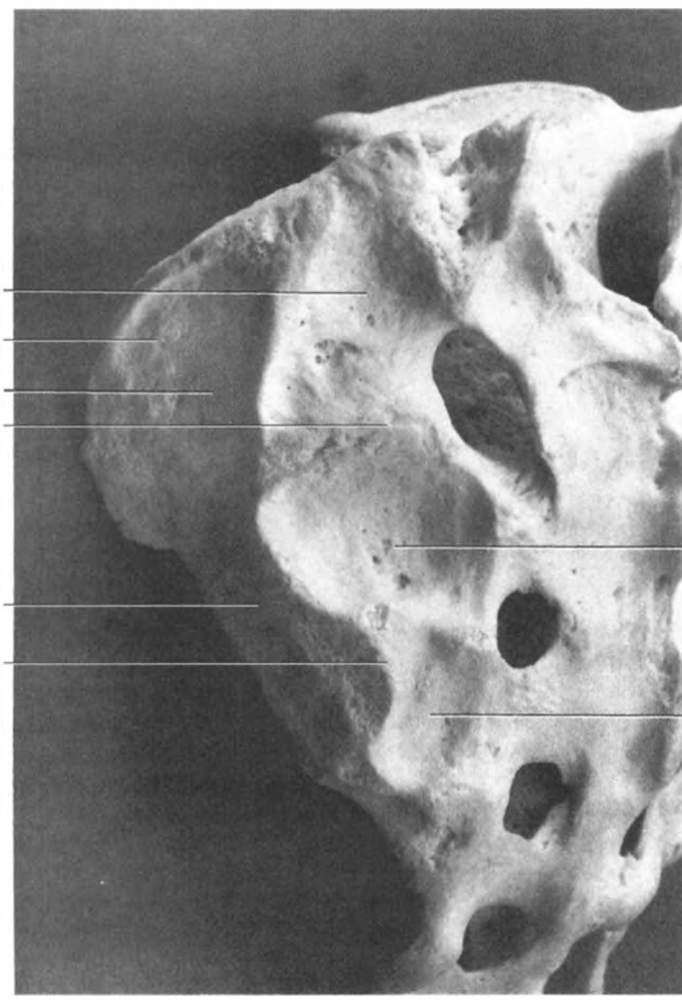

Figure 5.2. Posterolateral view of the sacral auricular surface and related bony features.

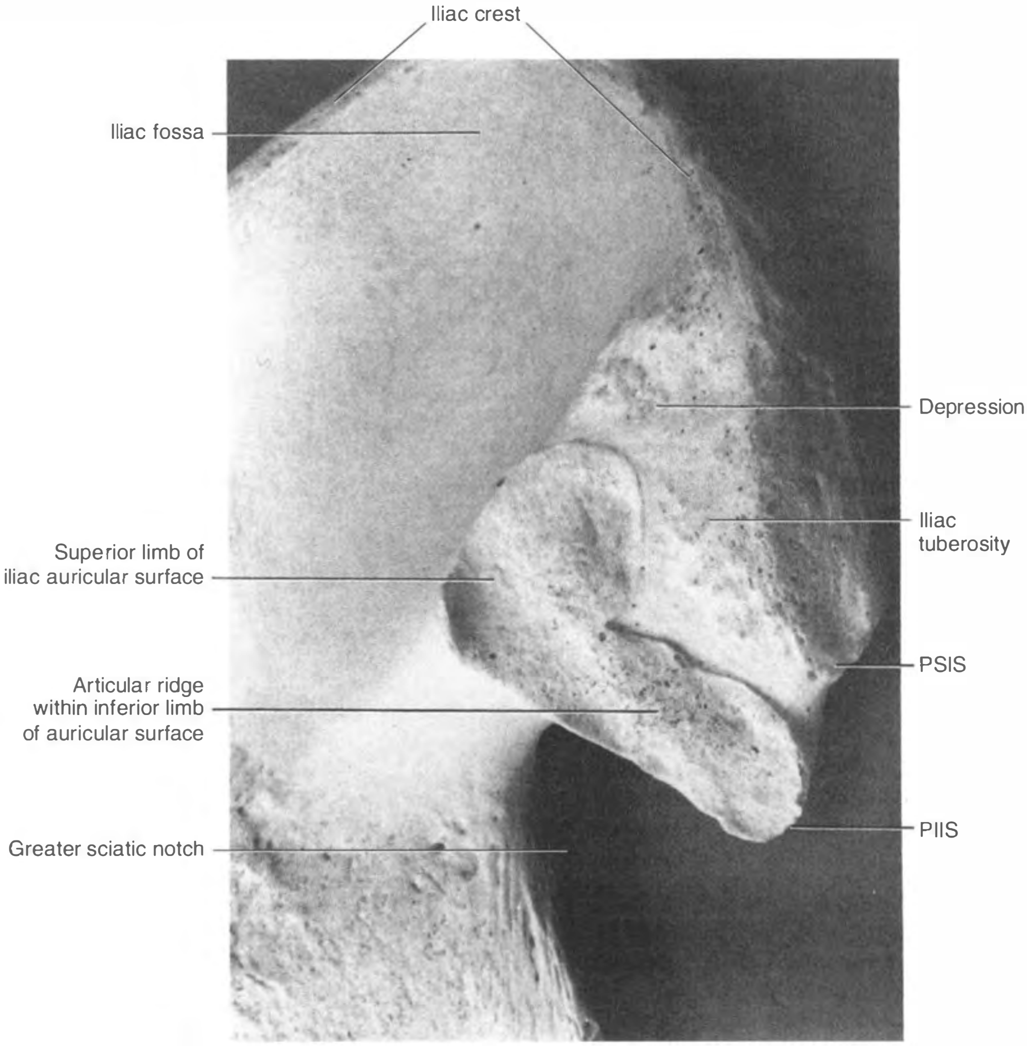

Figure 5.3. The iliac auricular surface and related bony features.

quently, because of the need to balance and contend with such increases in load, the biped SIJ is believed to have undergone a positional transformation and is more aligned in parallel with the spinal column. Also, as described above in humans the SIJ is half syndesmotic and half synovial. To ensure stability and yet deal with its bipedal functional role, the transformed SIJ is strengthened by the interosseous ligament and congruent bony surfaces that facilitate bony interlocking. Further, this positional transformation occurred in conjunction with changes in the spinal curvature and altered muscular attachments (e.g., the fascia lata in humans is stronger and incorporates an iliotibial tract to facilitate the upright stance and efficiency of muscle actions).

Postnatal Development of the Sacroiliac Joint

At birth the articular surface of the SIJ is oriented vertically and is morphologically flat and straight (11, 28); not until puberty does the joint morphology develop a more adultlike auricular shape. The planar articular surface during infancy and childhood allows freedom of movement in all directions and the stability of the joint is entirely dependent on its supporting ligaments during this early period (28). At puberty the elongate inferior limb and a broader superior limb of the auricular surface can be identified (25). The stability of the joint is enhanced during the second decade of life by the appearance of the sacral articular sulcus and iliac articular ridge which continue to become more prominent through adolescence. This remodeling process occurs as a result of secondary ossification centers that after 12 years of age appear near the joint in the hyaline cartilage model of the developing bone. Throughout this initial developmental period to the age of approximately 18 years, the sacral vertebrae and pelvic bones (ilium, ischium, and pubis) remain separate by cartilaginous regions that gradually ossify. Synostosis occurs after the age of 18 and is completed by the 25th year, at which time the SIJ has completely acquired adult morphology (29).

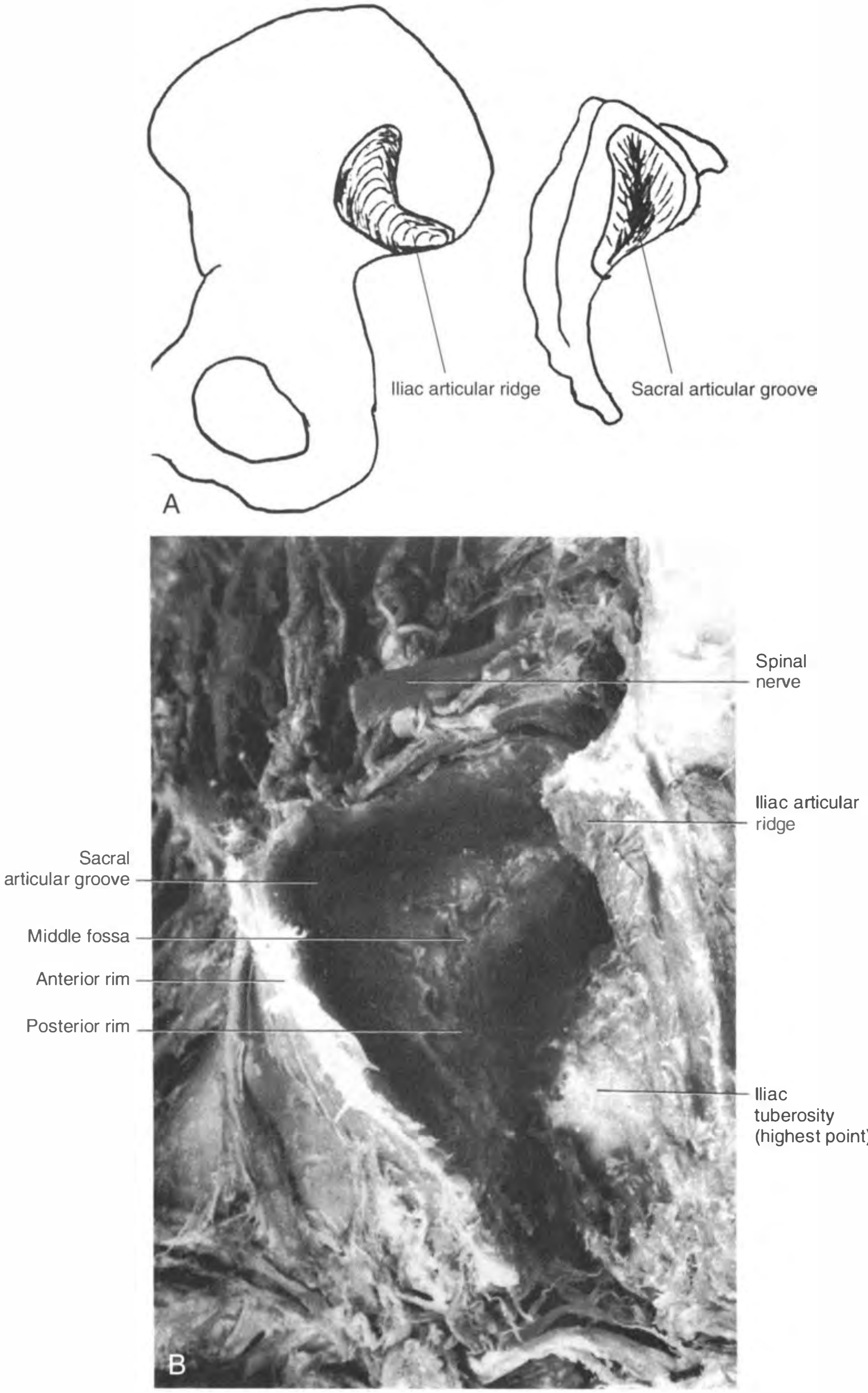

Figure 5.4. A schematic representation (**A**) and the related anatomic section (**B**) illustrating the relationship between the iliac ridge and sacral groove.

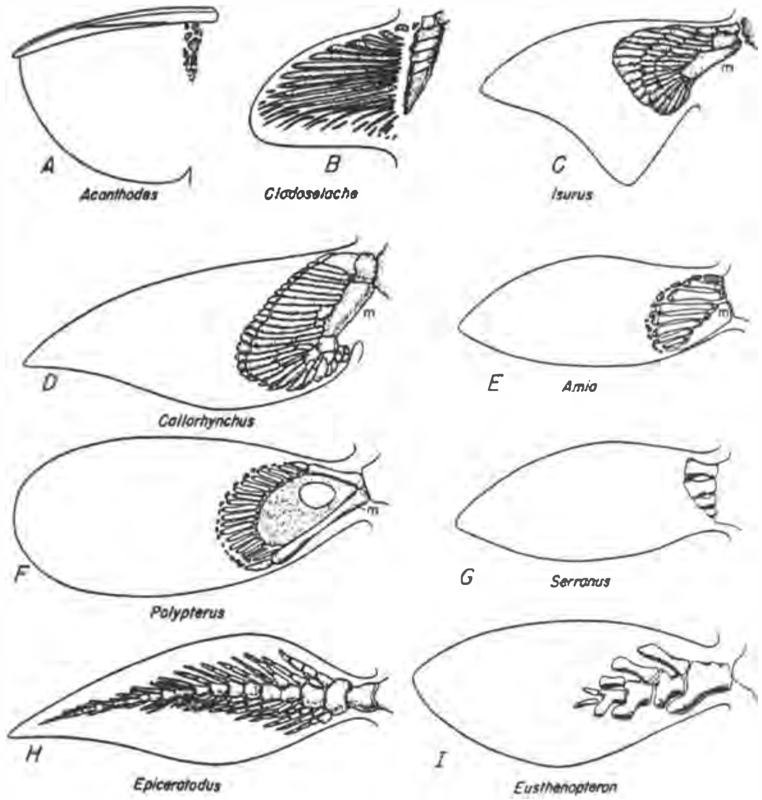

Figure 5.5. Illustration of fish fins connected by primitive symphysis.

The SIJ continues to acquire stronger stabilizing elements throughout adult life. During the third decade the sacral and iliac tuberosities become enlarged and fibrosis of the interosseous sacroiliac ligament strengthens the joint posteriorly. The bony margins of the auricular surface continue to ossify and marginal osteophytes appear during the fourth and fifth decades (Fig. 5.6). This process of osteophytosis appears to be more prominent in males and is believed to occur to further stabilize the joint in response to strenuous physical activity (11, 30, 31). Following the fourth decade the cartilaginous elements of the joint gradually become thinned and a process of marginal ankylosis may ensue. In many individuals this leads to total fibrous and bony ankylosis of the joint until by the eighth decade mobility of the SIJ is lost completely in most individuals. To date, the physiologic mechanisms that underlie this gradual process of fibrosis and ankylosis of the SIJ are poorly understood. It remains unclear whether this process is part of normal aging that occurs to decrease mobility and further stabilize the joint or whether these changes are pathologic.

Intrinsic Ligaments of the Sacroiliac Joint

The fibrous capsule of the SIJ is strengthened anteriorly and posteriorly by intrinsic capsular ligaments (Fig. 5.7). The ventral or anterior sacroiliac ligament (VSL) strengthens the inferior half of the anterior capsule. Its fibers, which are thin superiorly and become progressively thickened inferiorly, attach horizontally across the joint. The strongest part of the VSL attaches anteriorly to sacral ala at the level of the second sacral segment and crosses the most inferior part of the SIJ to attach to the subauricular sulcus on the ilium as far back as the PIIS (Fig. 5.7).

The dorsal or posterior sacroiliac ligament (DSL) is divided for descriptive purposes into two components: short and long DSL. This ligament occupies the deep recess between the sacrum and the ilium posteriorly, called the "sacroiliac fissure." The short DSL attaches medially to the sacral tuberosity along the lateral sacral crest (Fig. 5.7). Its fibers, which are greatly thickened relative to the VSL, course laterally and superiorly to attach to the anteromedial aspect of the PSIS of the ilium. This portion of the DSL may or may not be continuous with the interosseous sacroiliac ligament (Fig. 5.8) which lies deep to it within the syndesmotic compartment of the SIJ.

The long DSL is more vertically oriented with dense fibers attaching superiorly to the sacral ala above the first posterior sacral foramen and the PSIS posterior to the attachment of the short DSL (Fig. 5.7) along with the longest fibers of the sacrotuberous ligament (see below). The fibers of the long DSL, which are thick and strong, course inferiorly and medially to attach to the lateral sacral crest at the level of the third and fourth sacral segments and blend superficially with fibers of the sacrotuberous ligament.

Both the VSL and the DSL function to counteract gravita-

tional forces and prevent distraction of the SIJ, particularly during upright posture and through the gait cycle. The DSL also serves to provide attachment for the deep fibers of the multifidus and gluteus maximus muscles. One additional intrinsic capsular ligament has been described by Illi (32), which is sometimes referred to as "Illi's ligament" (33). This ligament strengthens the SIJ capsule superiorly by attaching across the margins of the auricular surface; it may be an extension of the interosseous sacroiliac ligament (Fig. 5.9).

Extrinsic Ligaments of the Sacroiliac Joint

The iliolumbar, sacrotuberous, and sacrospinous ligaments are extrinsic to the fibrous capsule of the SIJ; however, they assist the VSL and DSL in stabilizing the joint. The iliolumbar ligament attaches superiorly to the transverse process and body of the fifth (and sometimes fourth) lumbar vertebra. Its fibers course laterally, for the most part, to attach along the superior border of the medial third of the iliac crest. This ligament may also have vertical fibers that blend anteriorly with the VSL and posteriorly with the long DSL (Fig. 5.7). The iliolumbar ligament helps to prevent distraction of the SIJ superiorly. The sacrotuberous and sacrospinous ligaments, on the other hand, function to prevent posterior displacement of the sacral apex during nutation of the sacral promontory. Structurally, the sacrotuberous ligament attaches medially to the lateral sacral crest from S3 to S5 (Fig. 5.7). Long fibers of the sacrotuberous ligament also originate from the PSIS and join the lower fibers to course inferiorly, laterally, and anteriorly to attach to the medial aspect of the ischial tuberosity. Sacrospinous ligament fibers attach to the anterolateral border of the sacrum at the level of the third to fifth sacral segments and course laterally and anteriorly to reach the ischial spine.

Muscles Surrounding the SIJ

No typical intrinsic muscle exists for the SIJ. However, about 40 muscles can influence SIJ motion (34) (Fig. 5.10). Some of these muscles attach at three points, including a small portion connecting the sacrum and ilium of the SIJ (19, 35). These are the erector spinae, multifidus, iliopsoas, gluteus maximus, and piriformis muscles (36). The muscles covering the anterior sur-

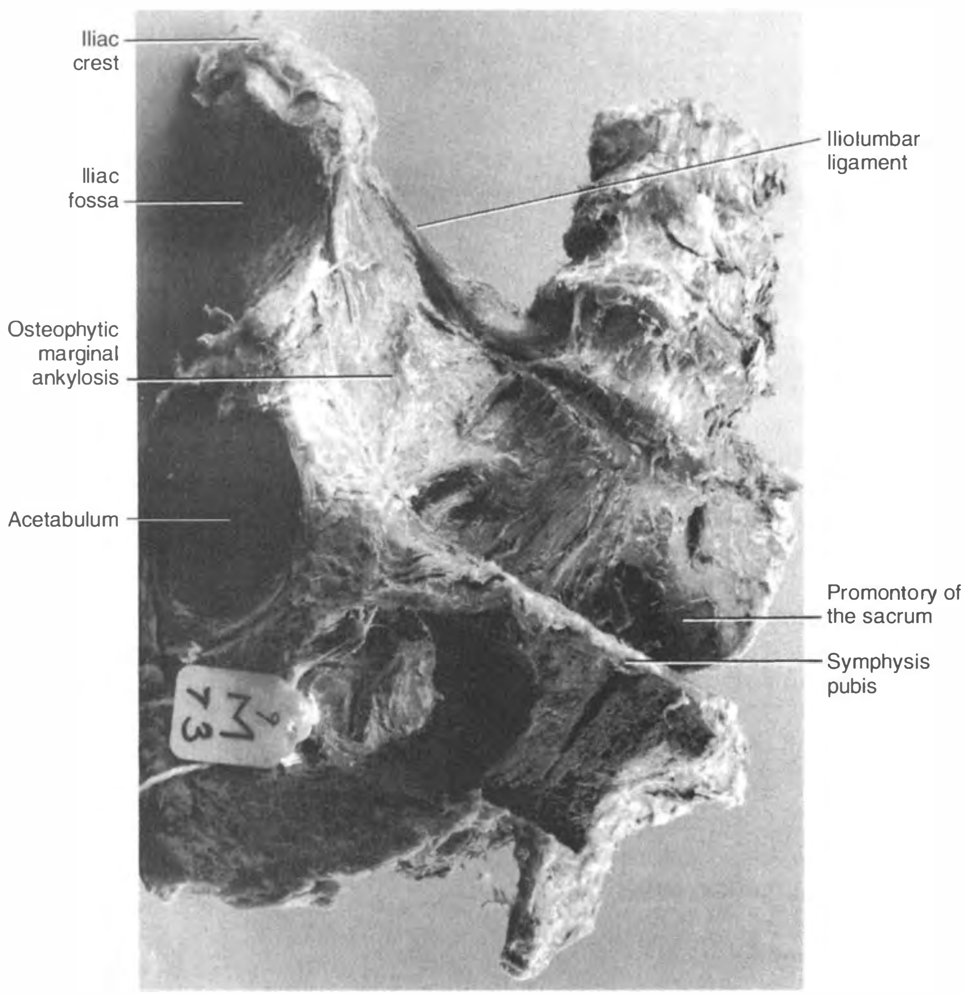

Figure 5.6. An anatomic section illustrating the anterosuperior surface of the sacroiliac joint, which is the most frequent site of osteophytic marginal ankylosis.

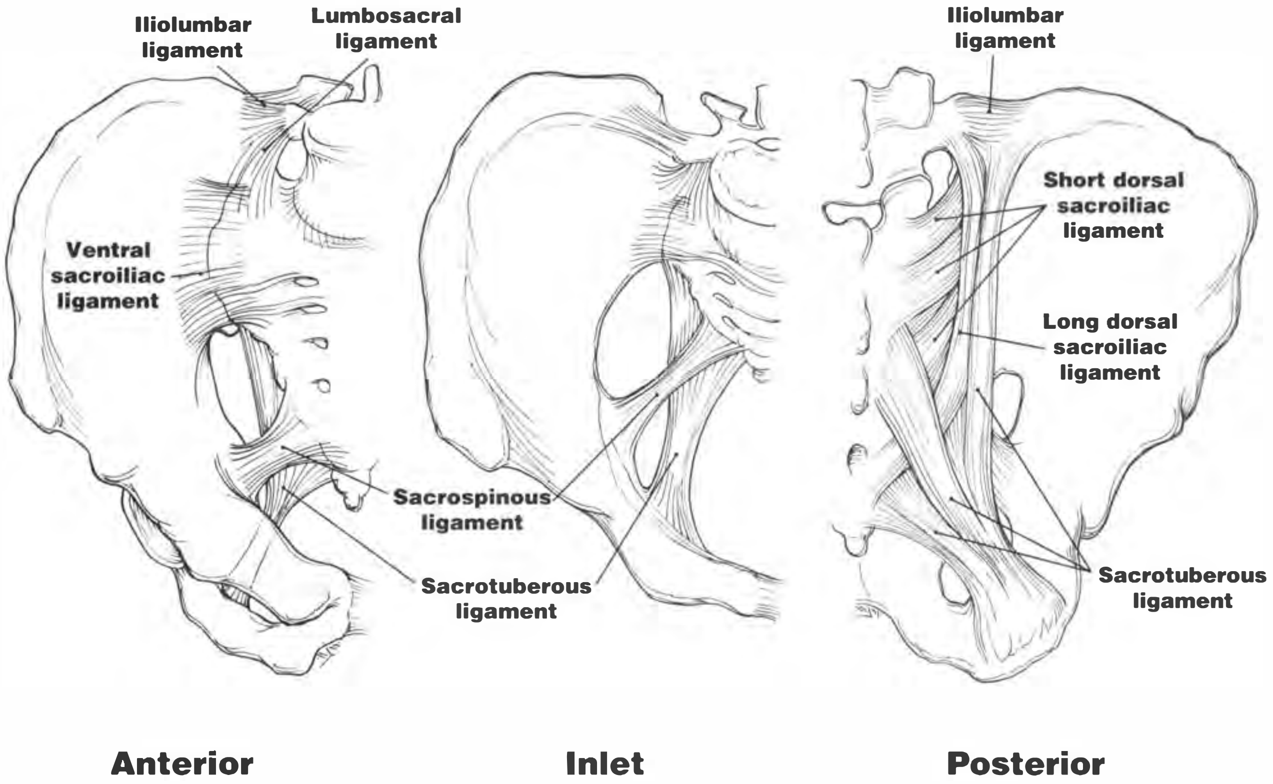

Figure 5.7. The ventral (anterior) and dorsal (posterior) sacroiliac ligaments.

face of SIJ are the iliopsoas, which is innervated by L1, L2, and L3, and the piriformis, which is innervated by L5, S1, and S2. The muscles covering the posterior surface of the SIJ are gluteal muscles, which are innervated by L4, L5, S1, and S2.

Because the SIJ has no intrinsic muscle of its own, its movement occurs through various mechanisms: The sacrum moves when the spinal column changes position, and the ilium moves when the lower extremities change their position.

The SIJ is also affected by the muscles capable of tilting the pelvic ring (28, 34, 37–41). SIJ movements are created by (*a*) the muscles that flex, extend, or rotate the vertebral column, moving the sacrum; (*b*) the muscles that flex, extend, abduct, adduct, supinate, and pronate the thigh, moving the ilium; and (*c*) the muscles that tilt the pelvis anteriorly, posteriorly moving the sacrum, and tilt right or left laterally, moving the ilium. The sartorius muscles extend the ilium, whereas the hamstring muscles flex ilium. The rectus abdominis muscles tilt the pelvic ring posteriorly and the erector spinae muscles tilt the pelvic ring anteriorly by moving the sacrum.

Arterial Supply to the Sacroiliac Joint

Branches of the posterior division of the internal iliac artery supply the anterior aspect of the sacroiliac joint. The primary branch to the joint anteriorly is the lateral sacral artery, which usually is a direct branch of the posterior division of the internal iliac artery. The lateral sacral artery passes inferiorly along the sacrum lateral to the anterior sacral foramina into which it may send radicular branches and penetrates the piriformis muscle, which it supplies, to reach the SIJ. In addition, the iliolumbar branch of the internal iliac artery may send articular branches into the anterior, superior aspects of the SIJ (Fig. 5.11).

Posteriorly, the SIJ is supplied by penetrating branches of the superior gluteal artery—also a branch of the internal iliac artery—which enters the gluteal region deep to the gluteus maximus muscle through the greater sciatic foramen. The superior gluteal artery then divides into superficial and deep divisions; the superficial division branches medially to supply the overlying gluteus maximus and penetrates the multifidus muscle to reach the posterior aspect of the SIJ. The deep division of the superior gluteal artery does not supply the SIJ; instead it courses laterally deep to the gluteus medius muscle.

Innervation of the Sacroiliac Joint

Innervation of the SIJ is highly variable even from side to side in the same person (42). This variation contributes to the different reported pain referral patterns and, ultimately, in diagnostic confusion (43–45). Nerves from L2 to S4 can all be found in the SIJ. Posteriorly, the nerves run between the superficial layer of interosseous sacroiliac ligaments and the dorsal sacroiliac ligaments. The anterior surface of the joint is most frequently innervated by the anterior primary rami (PPR) of S1

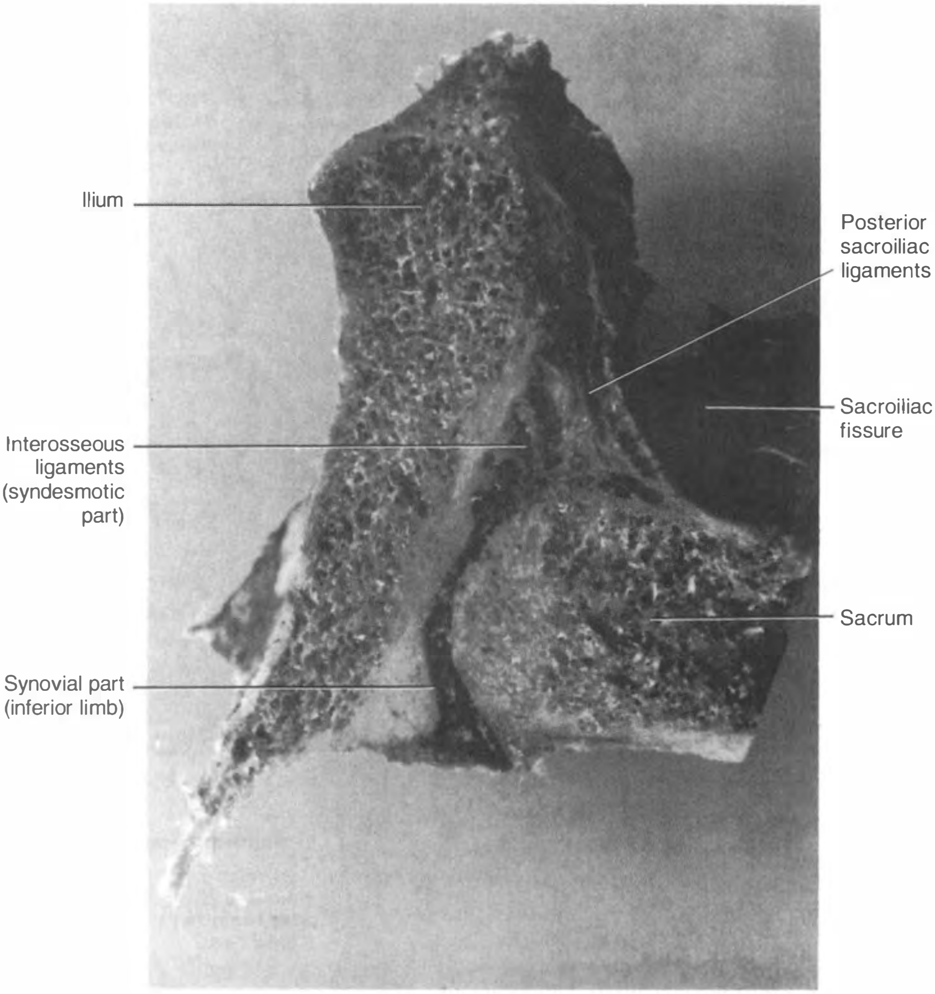

Figure 5.8. The interosseous sacroiliac ligaments.

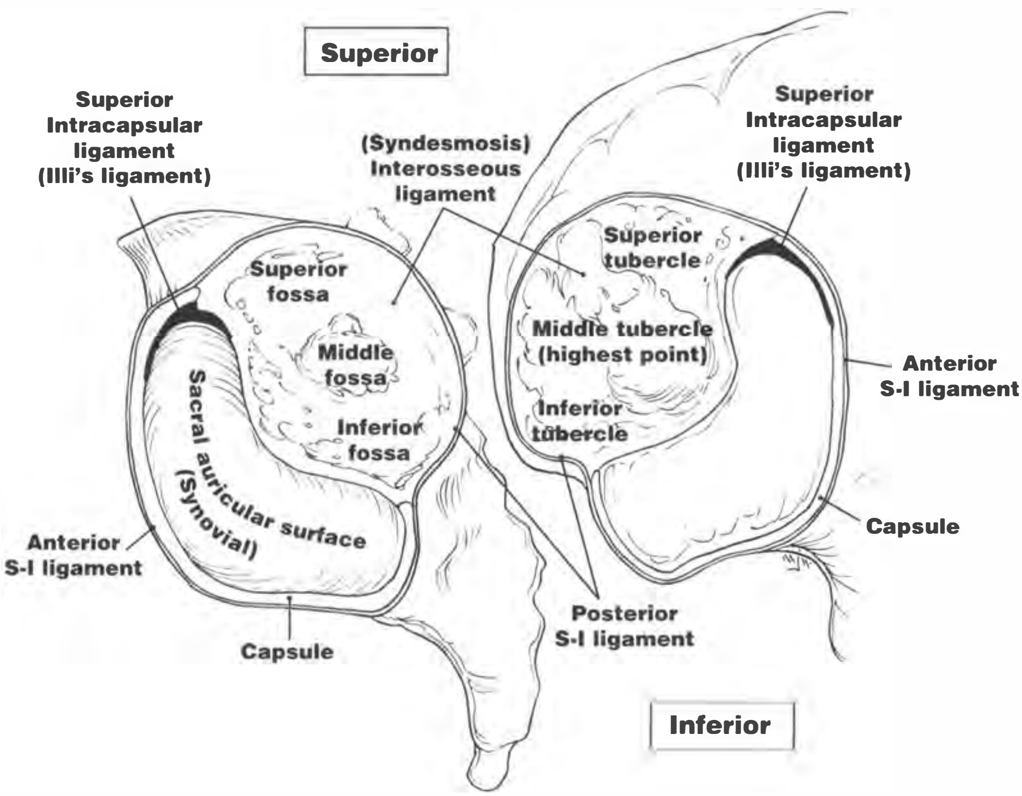

Figure 5.9. A cross section of the sacroiliac joint illustrating the intimate relationship between the anterior and posterior ligaments.

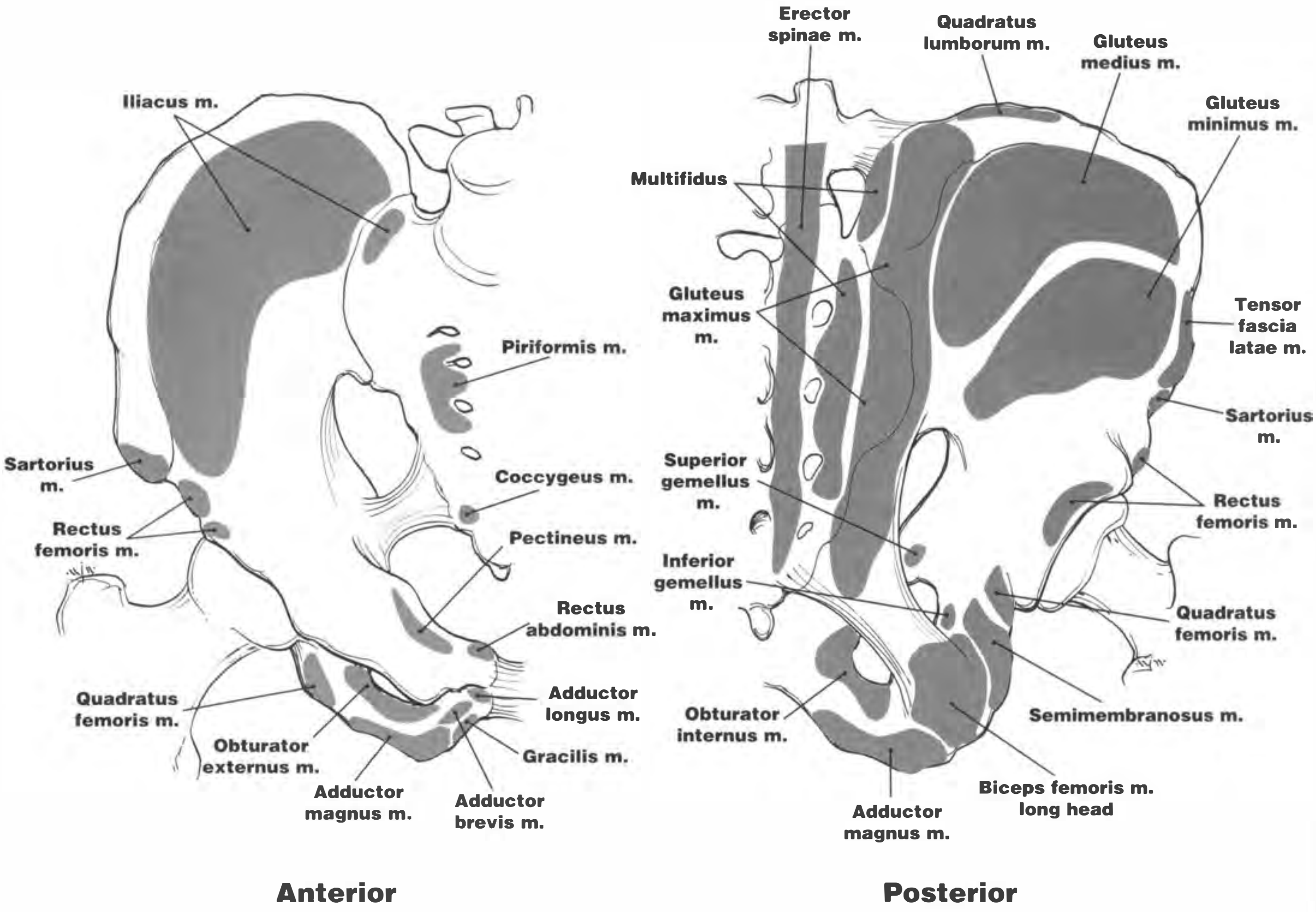

Figure 5.10. A schematic illustration of the numerous and powerful muscles that attach to the pelvis and which can directly or indirectly affect the function of the sacroiliac joint.

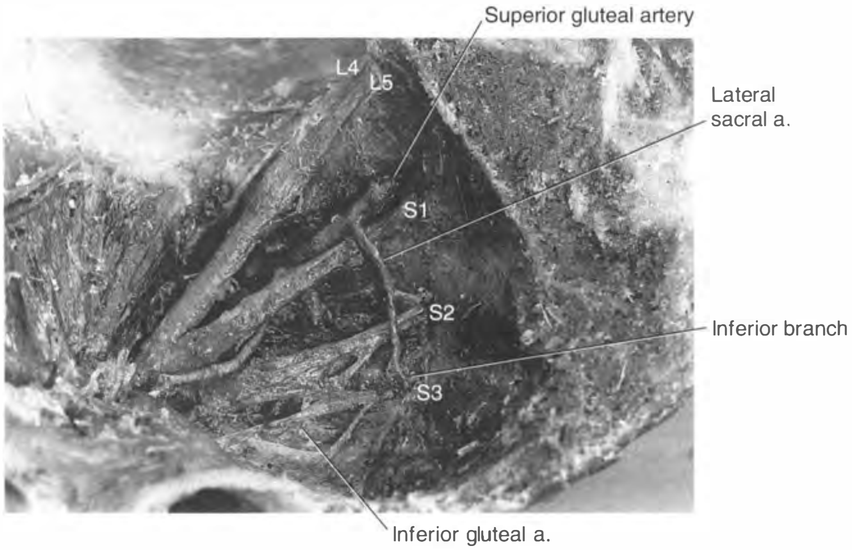

Figure 5.11. An anterior section of the pelvis illustrating the vessels that supply the sacroiliac joint.

and S2 (42). Bernard and Cassidy, however, have reported that the SIJ is innervated by L4 to S3 (46). Ro found lateral branches of the PPR of L5 extending distally onto the joint as illustrated in Figure 5.12.

The SIJ is richly endowed and innervated by nociceptors (pain receptors) and proprioceptors (movement and position sensors) (25, 28). This rich innervation may be because the joint monitors the movement and position of the pelvic ring, thus contributing to keeping the body balanced and upright.

BIOMECHANICS

Biomechanics of the SIJ are difficult to study. Direct palpation and access are impossible and its variable shape and symmetry makes modeling complicated (24, 47–49). The synovial and syndesmotic parts of the joint, and the inherent interdigitating irregular articular joint surfaces contribute to creating variable patterns of movement. Movement of the joint is not only influenced by muscle action but also by many external forces, including gravity and ground reaction forces. Further, the SIJ is surrounded by some of the body's more powerful muscles attached to various areas of the pelvis and the connecting symphysis pubis (50–53). The consequence of this unique structure and function creates the trabecular patterns visualized in the bony pelvis that suggest the SIJ and symphysis pubis are interdependent functional units in the pelvic ring (54).

Kinematics

Studies to understand what occurs to the pelvis during pregnancy and delivery have provided interesting insights into SIJ motion. Hippocrates believed that SIJ movement occurred only in pregnant women. Even today authors believe that no movement occurs in the SIJ except in the pregnant woman when the hormone, relaxin, is released. This hormone has been found to lengthen the true conjugate measure of the female pelvis from 8 to 13 mm via the "loosening" of SIJ and symphysis pubis (55). Interestingly, the true conjugate length has also been found to increase or decrease with changes in posture because of the movement of the SIJ and pubic symphysis, regardless whether the person was pregnant (50, 51). For example, it has been reported that the "Walcher position" (extension)

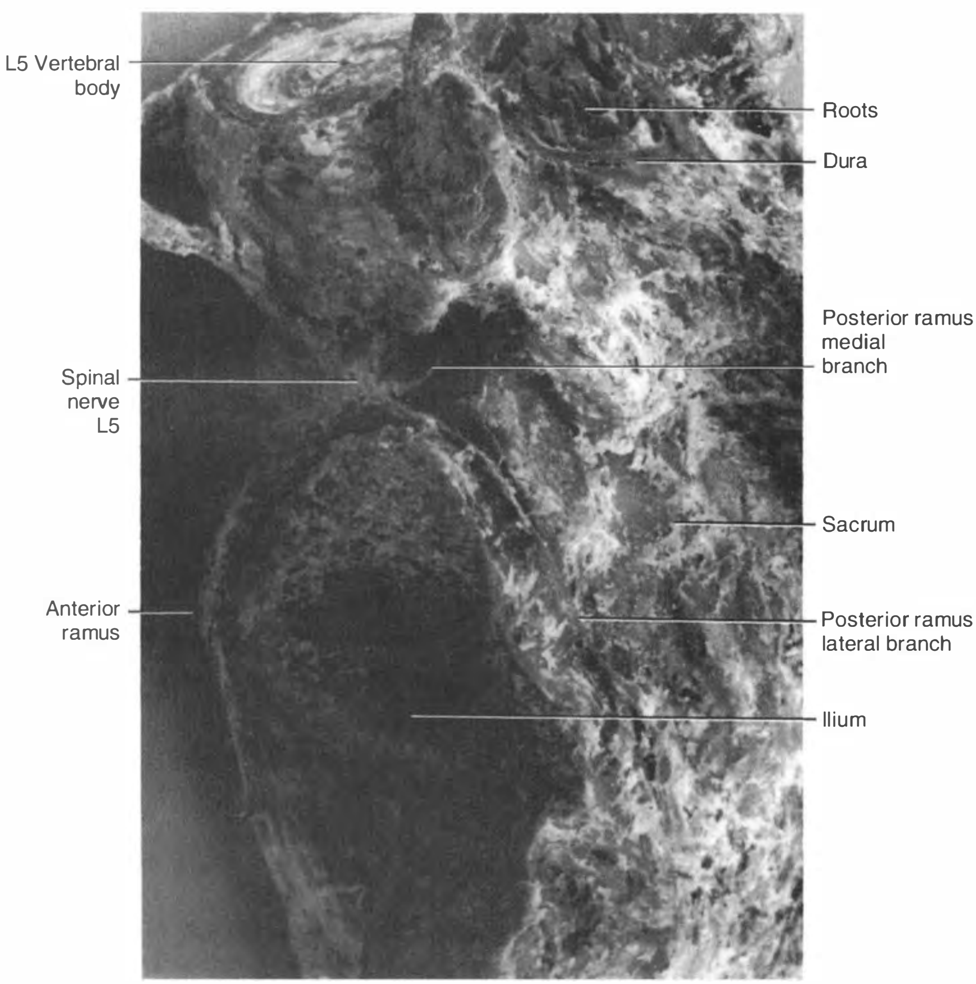

Figure 5.12. Anatomic section illustrating the innervation of the sacroiliac joint.

increased the pelvic inlet, whereas the lithotomy position (flexion) decreased the pelvic outlet (37, 38).

Traditionally, SIJ motion has been assessed by observing the movements of the sacral promontory. Researchers using radiographic studies have reported sacral promontory nodding (nutational motion) of about 5 to 6 mm occurring about a transverse axis located 5 to 10 cm below the promontory; rotating through an average angle of 8°, ranging from about 4 to 12° (18, 28, 39, 55). The nature of this combined upward and downward translation with the rotation of the sacral auricular surface is consistent with the behavior exhibited in saddle-type joints (Fig. 5.13).

Others have attempted to measure the relative movement of the sacrum to the ilium by using the PSIS as a landmark. Pitkin and Pheasant recorded motions of 2 mm and 2°, while the level of inclination between the right and left anterior superior iliac spine (ASIS) was about 11° (38). Colachis implanted Kuschner pins in the pelvis and used cineradiography to assess the movement (39). He found a small degree of movement, with the greatest range being observed during forward flexion from a standing position.

In another study, Sturesson et al. implanted four 0.8 mm diameter tantalum balls into both the pelvis and the sacrum. Stereoroentgenograms were taken of patients in five different positions and movements were recorded. Findings were relatively minor three-dimensional movement ranging from 1 to 2° and 0.5 to 1.0 mm (56).

Frigerio et al. were the first to quantify the movement of the SIJ by adapting a system of stereoradiography and mathematical modeling (57). They combined the measurements obtained from two radiographs taken orthogonally to reconstruct the SIJ in three dimensions and then correlated the relative motion of the joint. Findings were that the movement of the ilium relative to the sacrum averaged 2.7 mm, and that movement between the innominates (measured between the right and left ASIS) was a maximum of 15.5 mm. Egund, using stereophotogrammetric (not radiography) and mathematical modeling found 2° of rotation and 2 mm of translation (58). Drerup and Hierholzer in 1987, used rasterstereographic surface measurement together with surface curvature analysis of the PSIS dimple and computer mathematical analysis (59). They reconstructed the true spatial position of the joint and found the average PSIS dimple displacement was ±1.5 mm and the torsion angle was ± 1.5°.

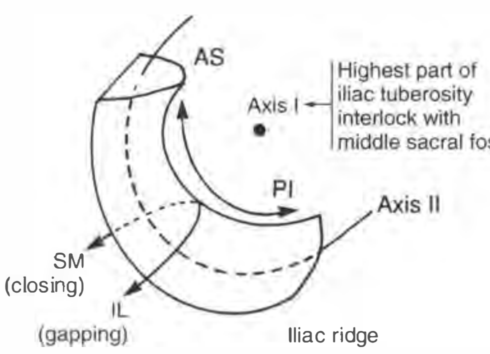

Figure 5.13. A line drawing illustrating the saddle shape of the sacroiliac joint and the outline of the translational and rotational movements that may take place depending on the axis of motion selected.

More recently, Kissling using a three-dimensional stereophotogrammetric method, reported considerable variation in the SIJ motion within and between 24 healthy volunteers ranging in ages between 20 and 50 years (60). Although the position and direction of the movement axes varied, a characteristic pattern was detected. The average degrees of rotation and translation ranged from 1.8° and 0.7 mm for men and 1.9° and 0.9 mm for women, respectively. He reported no statistically significant age or sex differences in rotational and translational movements. Kissling's findings that motion does not significantly differ as one ages, at least to 50 years, are interesting considering that many believe that the degree of motion in the SIJ decreases with aging because of inherent degenerative joint changes.

Two studies assessing movement in the SIJ of older patients support the notion that the degenerative changes observed macro and microscopically may not necessarily imply that the aged SIJ does not move. Miller et al. assessed the kinematics in eight fresh cadavers, aged 29 to 74 years, with the muscles removed (47). They loaded the SIJ and measured the displacement of the sacrum relative to one or both ilia. They measured the average degree of lateral translation as 0.76 mm (standard deviation [SD]1.41), anterior translation as 2.74 mm (SD 1.07), lateral rotation as 1.40° (SD 0.71), and axial rotation as 6.21° (SD 3.29).

In another study conducted on five fresh nonembalmed cadavers, aged 52 to 69 years, radio-opaque markers were implanted in the pelvis and spine; computed tomography scans were used and then converted to computer images for measurement analysis. The total SIJ range of motion for double leg flexion–extension in the sagittal plane was 8° on the right and 7° on the left. In the coronal plane 2° of motion was recorded, whereas in the transverse plane 1° was recorded on the right and 2° on the left (61). Although the movements were small, they do add preliminary support to the claims made by clinicians that the SIJ does move, and it can be manipulated in older patients.

Regarding pubic symphysis movement, Stern et al. reviewed the related literature and reported that the average transverse width of the adult symphysis pubis is 5.9 mm in males and 4.9 in females, which widens to 7.1 mm during pregnancy (62). Radiographic studies have been used in detailing symphyseal movement in adults, which has been found to measure 0 to 0.5 mm in males, and 0 to 1.0 mm and 0 to 2.0 mm in nulliparous and parous women, respectively. Walheim and Selvik reported translations of up to 2 mm and rotations of up to 3° in normal subjects (63). Pubic symphysis instability was considered present when symphyseal width was greater than 10 mm and vertical displacement was greater than 2 mm. The cause of symphysis diastasis is varied; it can be the result of single or repetitive injury or hormonal influences (47–50, 62, 64).

Instantaneous Axis of Rotation

If the quantification of the movement of the SIJ is discrepant, it is even more so when the instantaneous axis of rotation (IAR) is discussed. The actual location of the axis of rotation of the SIJ has long been debated (26, 34, 65). Because of the inherent variations in the anatomy of the SIJ—from aging, gender differences, mechanical loads, endocrinologic effects, and other factors—no one distinct IAR is found.

Historically, Farabeuf suggested a fixed axis of rotation located around a transverse axis passing through the interosseous ligament (66). The resultant movement would be seen to have the sacrum following an arc of a circle whose center is located posterior to the joint. Bonnaire and Bve' suggested that this transverse axis passed through the sacral tubercle, thereby creating an angular displacement about the center of the joint (67). Pitkin and Pheasant contended that the axis for flexion and extension fell through the interosseous ligament (38). Weisel described two different axes to explain the translational and rotational movements observed in the range of motion studies outlined above (55). He proposed that for translation to occur a pure linear displacement of the sacrum would take place as the sacrum slid along an axis at the caudal portion of the SIJ. Rotational motion would take place around an axis that was anterior to the joint and anterior and inferior to the sacrum.

Wilder et al. assessed the axis of rotation of the SIJ by analyzing its topography (23). They used gross contour profiles of the joint in the frontal and sagittal planes and statistically redefined the axis of rotation by defining a best fit axis of rotation for each profile. They found that the IAR in both planes was scattered broadly in and outside the joint and, therefore, rotation could not occur about a single axis of rotation as previously proposed. Further, translation occurred about a "rough axis" of each slice only after sufficient force was applied to overcome the resistance of the ligamentous structures, thus allowing the SIJ to separate and move. Finally, they found that the IAR varied considerably among the specimens used. These findings along with the others reported herein help to explain the difficulty in obtaining an accurate assessment of the movement characteristics of the SIJ.

Kinetics

The SIJ's position as a link in the kinetic chain between the spine and legs makes it imperative that it have stability and mobility and yet be able to withstand considerable forces affecting it (Fig 5.14). Miller et al. undertook to study the load displacement behavior of fresh cadaver SIJ (47). The SIJs were found to be able to resist loads of 500 N or 50 N-m without failure in the eight primary directions tested. Failure tended to occur in the bone medial to the SIJ except in torsion tests, where the failure was ligamentous. The degree of motion measured in the SIJ was insignificant when both ilia were fixed compared with the displacement observed when one of the ilia was fixed and the other subjected to various loads in different directions. Also observed was variation in the ability of each of the specimen to resist loads. In comparison with the stiffness measured at the L3 to L4 motion segment of the lumbar spine, the SIJ was six times stiffer in medially directed forces (lateral shear on a lumbar motion segment) but 20 times weaker in inferiorly directed forces (axial compression on a lumbar segment). Therefore, the SIJ is less stiff when subjected to compressive and torsional loads compared with the lumbar motion segments, making it susceptible to activities requiring forward flexion, twisting, and lifting (68, 69).

In addition, the SIJ's strategic location makes it susceptible to large downward shear loads ranging from 300 to 1750 N during daily activities (47). Gunterberg et al. reported that SIJs in cadaver specimens had a mean downward shear strength of 4865 N (70). The SIJ withstands such forces by nature of its architecture and surrounding soft tissues. The flat orientation of the joint surfaces enables the SIJ to transfer great moments of force, but it is extremely vulnerable to shearing forces resulting from loads and moments occurring in a direction parallel to the joint surface (24, 71, 72). This vulnerability to shear may predispose the SIJ to sublux superiorly; however, this is prevented by a hypothesized "self-bracing mechanism" (24, 41, 61). This mechanism is facilitated by the following SIJ characteristics:

1. The archlike architecture of the pelvis.
2. The joint's longitudinal dimension is twice that of the transverse, thus providing favorable resistance against bending moments along this plane.
3. Grooves and ridges of the joint surfaces form a resistance to sliding.
4. The higher friction coefficients in the joint because of the rough-textured surfaces.
5. The corkscrew appearance of the joint created by the different wedge angles in transverse cross sections at the cranial and caudal ends of the joint.
6. The ligaments.
7. The muscles.

Snijders et al. have postulated that the ligaments and muscles that cross the SIJ play a key role in compressing the joint surfaces (73). When loaded, the interosseous and the sacrotuberous ligaments diminish the total range of ventral rotation (nutation) while the long dorsal sacroiliac ligament tensed during counter nutation. Interestingly, connections between the long dorsal sacroiliac and sacrotuberous ligaments and the muscles of the pelvis and lower limb may act to control excessive slackening of these ligaments (74).

Wilder et al. have suggested that in order for movement to take place in the SIJ, the joint must sufficiently separate to allow them to move over their irregular surface (23). To do this, energy is required to overcome the internal resistance of the ligaments. The energy absorbed by the ligaments suggests that the SIJ may function as a shock-absorbing structure.

Ro (unpublished data), after careful analysis of the anatomy and structure of the SIJ, contends that joint gapping is integral in determining its characteristic movement and function. The

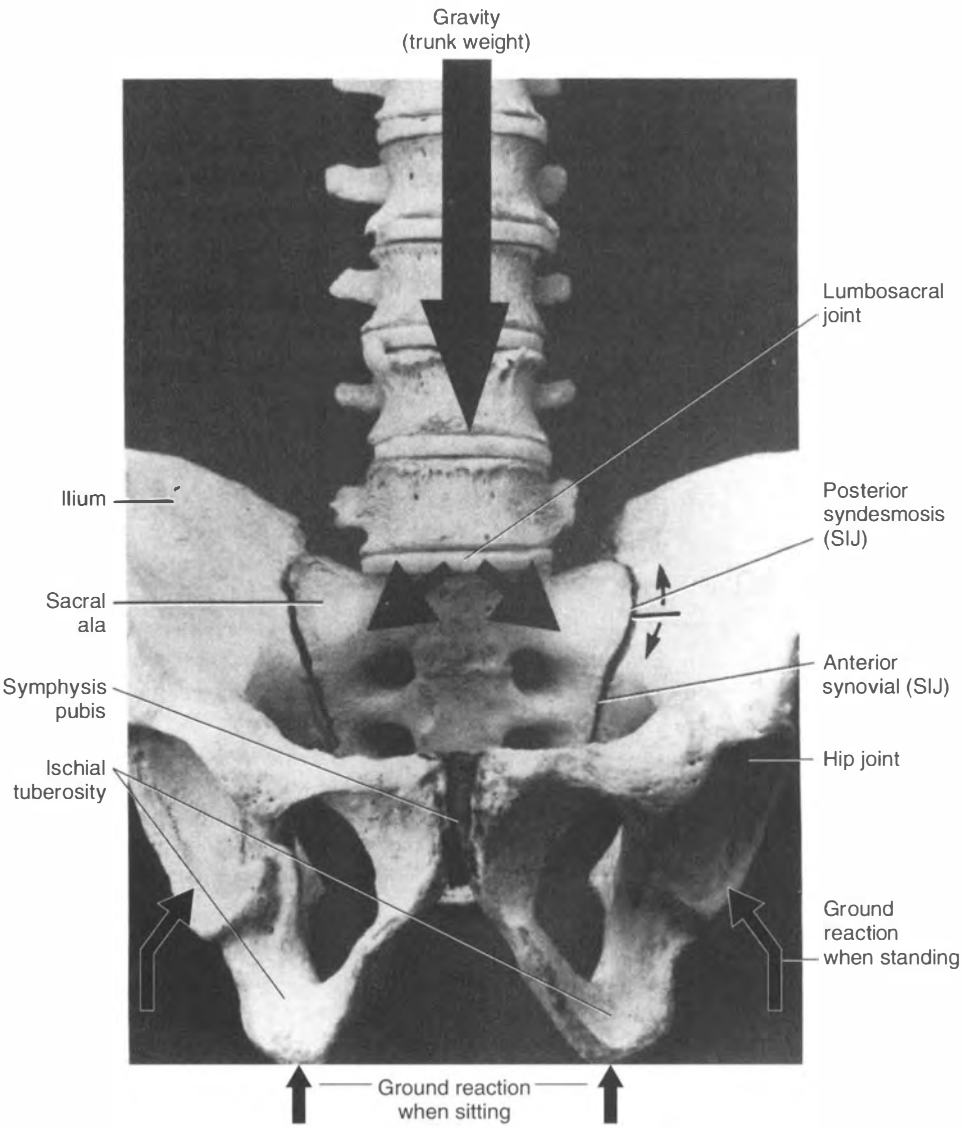

Figure 5.14. A schematic representation of the various forces that can impact on the sacroiliac joint.

concept of SIJ gapping is relatively new and not often mentioned, although some do allude to its importance in movement (23, 75). Others feel that gapping of the posterior–superior part of the joint could be more important than the rotation of the synovial part (76). This gapping motion combined with the rotation of the synovial part make it extremely difficult to measure the SIJ motion because of the resultant complicated three-dimensional motion.

The iliac sulcus between the iliac tuberosity and auricular surface and the sacral crest (medial rim of the inferior limb) are interlocking. The highest point of the iliac tuberosity interlock is with the middle sacral fossa. This is also the axial articulation for rotation of the auricular surfaces, the ridge in the groove. Inspecting these surfaces, one can find the traces of iliac tuberosity pivot in the middle sacral fossa (26, 65). Ro (unpublished data) found that this interlocking disfigured the left side iliac tuberosity by widening the sulcus. This occurred only on the left side and was present in about 75% of the left iliac tuberosities examined, and it seemed never to occur on the right side. The reason for this is not clear, but it may be caused by a short leg on the left side. Janse (33) found that body weight was carried by the short leg, although this has been disputed. One thing that is clear is that the sacral auricular crest pushed the iliac tuberosity upward (Fig 5.15). Some believe that early degeneration and the eventual ankylosis of the SIJ is caused by aging or the need for further stabilization; Ro believes that the disuse degeneration (disuse atrophy) is a consequence of the structures not being used and that the process can be altered by exercise.

Vlemming et al. believe that four muscles play a key role in stabilizing the SIJ (74). These are the erector spinae, gluteus maximus, latissimus dorsi, and biceps femoris. The sacral attachment of the erector spinae has been purported to pull the

sacrum forward to facilitate nutation, whereas the iliac attachments pull the posterior side of the iliac bones together, countering nutation. The dual functions of this muscle results in a compression of the cranial and widening of the caudal aspects of the joint, the latter being countered by the sacrotuberous ligament. The gluteus maximus muscle acts to compress the SIJ. The latissimus dorsi muscle, via the thoracolumbar fascia, has been found to act with the contralateral gluteus maximus muscle, to compress the SIJ. The thoracolumbar fascia can also be affected by its connection with the erector spinae. The long head of the biceps femoris by nature of its attachment to the ischial tuberosity and slips to the sacrotuberous ligament plays a role in preventing the sacrum from tilting forward, especially when the body is in a stooped position. The abdominal muscles have also been felt to play a role in stabilizing the pelvis. Therefore, the simultaneous activity of the hamstrings, gluteus maximus, erector spinae, biceps femoris, abdominal muscles, and the pelvic ligaments work together to provide further support and ensure the stability to the SIJ when subjected to loads.

Respiration has also been found to aid SIJ motion (28). During inspiration, the rectus abdominus muscle and the pelvic diaphragm relax causing the pelvic ring to tilt anteriorly (77). This is countered by the contraction of the erector spinae muscle's attachment to the sacrum. During expiration, the erector spinae muscle relaxes, while the rectus abdominus, because of its attachments along the superior pubic arch, pulls up on the pubic bone tilting the pelvis posteriorly (61, 78, 79).

The influence of the forces exerted on the SIJ in sitting may be responsible for the cause of joint dysfunctions and degeneration and perhaps may be more important as society moves more to the sitting rather than standing postures. In sitting the ground reaction forces from the ischial tuberosity act directly on the SIJ rather than being dissipated by the foot, knee, and hip as in standing. Sitting also causes the ilium to move posterior and superior and the ischial tuberosities closer together, thereby causing gapping of the posterior superior portion of the SIJ via the tension produced by the interosseous ligaments (80). Unfortunately, many authors neglect analysis of the SIJ in the important seated position.

The biomechanics of walking is also complicated. It involves flexion/extension, abduction/adduction of thigh, and flexion/extension, lateral bending, and rotation of the vertebral column. These motions are balanced and adjusted by SIJ motion. When the SIJ is dysfunctional, walking may become stiff and awkward (34, 81). Herzog and Conway noted changes in selected parameters of the vertical ground reaction force of a specific patient with SIJ syndrome following treatment (82). However, they caution that force recordings only provide information about the movement of the center of mass, but such movements have limited resolution and questionable accuracy.

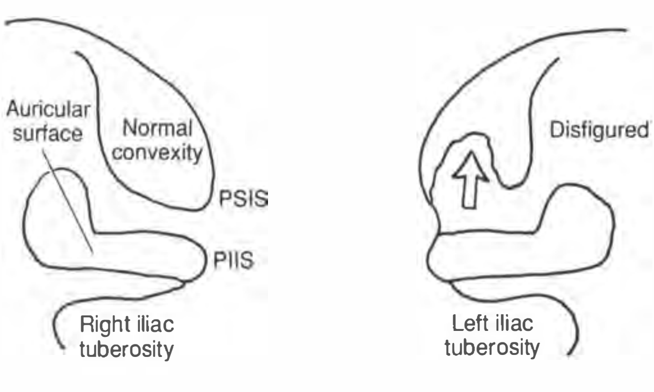

Figure 5.15. Disfigurement of iliac tuberosity.

Clinical Considerations

The prevalence of SIJ pain in the general population is unknown. Davis and Lentle used bone scan imaging to assess women presenting with low back pain and reported that 44% had SIJ involvement (83). They concluded that sacroiliac disease was a common cause of low back pain in women. In a retrospective study, Bernard and Cassidy estimated that 22.5% of patients presenting with low back pain had a form of SIJ syndrome (46). Schwarzer et al. used joint blocks and arthrography to investigate SIJ pain and found that the prevalence of SIJ pain ranged from 13 to 30% if the ablation of pain postinjection was the criterion, or 9 to 21% if pain relief plus evidence of capsular disruption was the criterion (84). Maigne et al., using a double anesthetic block technique, found that 18.5 % of their patient sample had pain of SIJ origin (85).

Mierau et al. also found that a significant portion of primary and secondary school children in the city of Saskatoon reported a history of low back pain and had evidence suggesting a sacroiliac joint dysfunction (86). Grieve (17) reported that Levitt found that more than 40% of school children between the ages of 6 and 7 had some degree of pelvic torsion but no (SIJ) symptoms.

Assuming adequate reliability and validity of the testing procedures used in the aforementioned studies, findings may imply that the spine and its related soft tissues can adequately compensate for altered function. It appears that the pelvic structures are sensitive and may be influenced by change (e.g., notable pelvic torsion may result from the removal of a small piece of bone for grafting) (17, 34, 53, 87–90). However, considering the complexity and limited knowledge of the pelvic and spinal mechanisms, it is not clear how the body compensates for such altered function (50–53).

Pathogenesis of SIJ Pain

The SIJ is subjected to many factors, ranging from trauma to infection, that may render the patient symptomatic. Although the SIJ's role as a causative factor in the genesis of low back and leg pain is becoming increasingly accepted, the underlying mechanisms are speculative at best. It has been suggested that pain about the SIJ is most commonly mechanical in nature, the most common cause being altered mobility (69). Altered mobility has also been defined as a fixation, dysfunction, subluxation, hypomobility, hypermobility, or instability. Unfortunately, some confusion exists regarding how too much or too little movement in the SIJ effects patient symptoms (69, 91).

Mechanical Causes

Recent attempts to understand the function of the SIJ have led to the hypothesis that explains how the SIJ may become dysfunctional and thus symptomatic. Vleeming et al. have done considerable research in trying to elucidate the normal and abnormal behavior of the SIJ. Their theories rest on the assumption that the integrity of the SIJ is maintained by "form closure" (i.e., the inherent anatomic characteristics of the joint) and "force closure" (i.e., the force created by the structures in and about the joint) (74) (Fig. 5.16). Together, form and force closure provide the basis for the "self-bracing mechanism" of the SIJ. If closure is affected or altered then the SIJ loses its stability and is predisposed to increase shear forces through the joint, leading to ligamentous and cartilaginous injury. Abnormal forces may be of sufficient magnitude that they overcome the stiffness of the surrounding soft tissue and bony structures of the SIJ, resulting in potential structural injury. Such injury may result in either hypo (fixation) or hypermobility or instability of the joint, possibly leading to pain.

Theoretically, a fixated joint can be caused by the altered position of the joint surfaces. For example, an abnormal load may force the joint surfaces apart and cause movement such that the ridge and depression are no longer complementary. If the joint surfaces fail to slide back "into position," a blocked or fixated joint will result (72), and it will be maintained by the compressive and elastic forces of the ligaments and muscles (92). This failure to return to normal position may be caused by external forces overcoming the internal protective resistance of the SIJ and its structures. Such forces may be created by repetitive stresses from running, dancing, gymnastics, skating, kicking, and jumping, to name but a few (64, 69, 93).

For instance, an example of how the aforementioned forces may impact on the SIJ and lead to symptoms has been described by Fortin (93). Applying the information reviewed above to the jump–landing mechanics of a competitive freestyle figure skater, he provided a realistic model for the cause of SIJ syndromes. Repetitive impact loading through one lower extremity on jump–landing creates tremendous shear force across the SIJ. Innominate shear dysfunction, sacral torsion, and disruption of the weak anterior sacroiliac ligamentous complex can be inevitable if muscle imbalances and missed landings (resulting in striking the buttocks directly on the ice) occur (93). Depending on the extent of the resultant injury, the SIJ may become fixated or hypermobile.

Hypermobility of the SIJ can result from repetitive injury (as described above) or from physiologically induced changes. Physiologically, the female SIJ increases its motion and decreases its stability during pregnancy, which may predispose the SIJ to possible ligamentous and joint injuries because of increased mechanical loading and lead to a hypermobile or unstable joint and subsequently to pain (62, 94). A pelvic or trochanteric belt worn below the SIJ, at or just above the greater trochanters, may provide external "force closure" to decrease the shear force and help reduce symptoms (73, 62).

Many theories attempt to explain the cause of SIJ syndrome (17, 95–98) and its consequence (99–102). The scenario described above can be replicated for many athletic and everyday activities; it can be further complicated by the impact of asymmetrical ground reaction or compressive forces produced by running on uneven surfaces, lift-off imbalance, or weak lower limb and spinal muscles. Each of these activities produces imbalanced, unilateral loads that may render the "self-locking mecha-

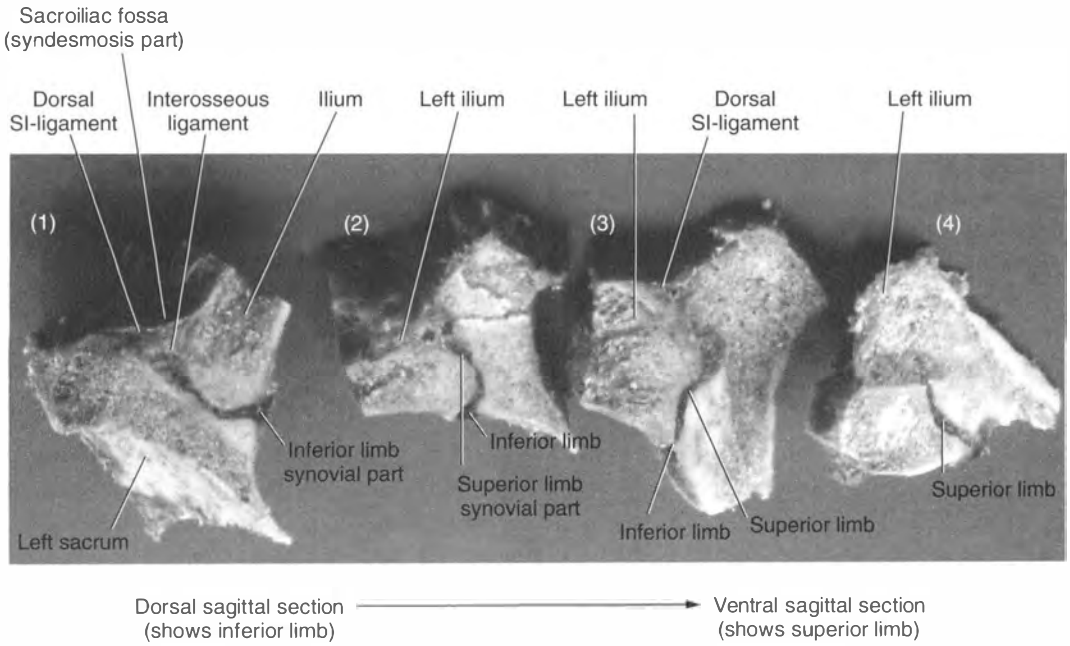

Figure 5.16. Serial anatomic sections of the sacroiliac joint illustrating its unique structure and the ligamentous structures that provide the basis for the "self-bracing mechanism" hypothesis.

nism" of the SIJ incompetent. However, much of this is conjecture and requires considerable more research to be validated.

Inflammatory Causes

Many inflammatory disorders affect the SIJ (103–116). Most of these are considered to be seronegative spondyloarthropathies. Spondyloarthropathies comprise a heterogeneous group of disorders that share common clinical and genetic features. They include ankylosing spondylitis, Reiter's syndrome, psoriatic arthritis, arthritis of inflammatory bowel disease, reactive arthritis, juvenile chronic arthritis, and others collectively listed as "undifferentiated spondyloarthropathies" (103). They invariably affect the lower half to two thirds of the synovial area of the SIJ. The pattern of joint involvement helps to differentiate the condition (e.g., bilateral and symmetric in ankylosing spondylitis and inflammatory bowel disease compared with asymmetric and unilateral in Reiter's and psoriatic arthritis).

Osteitis condensans ilii is characterized by osteosclerosis along the upper one third of the ilium near the SIJ at about the level of the PSIS. Its cause is unknown, but it appears to be seen more frequently in women, especially postpartum (117, 118). Degeneration of the cartilage begins on the iliac side because it is relatively underdeveloped compared with that of the sacrum (20, 119). The joint may become widened or narrowed, with cystic or erosive changes that may lead to fibrous or bony ankylosis (119). This bony and cartilaginous activity can be detected using scintigraphic procedures (120–132).

Infections within the SIJ are most frequently caused by staphylococcal bacteria, but have been caused by tuberculosis, gonococcal, and typhoid bacilli infections (133). Radiographs and bone scans are useful in identifying the presence of an inflammatory response. However, diagnosis is made through culturing of organisms obtained via joint aspiration and hematology.

Presenting Complaint

Patients presenting with SIJ syndrome typically complain of a dull to sharp pain that is localized to an area about the PSIS. The pain may be referred to the groin, buttock, or posterior thigh and may occasionally extend below the knee. The onset of the symptoms has been reported to be unknown or to be attributed to minor trauma in 58% and to compensable injuries in 42% of the patients studied by Bernard and Cassidy (46). They found an average duration of the complaint was 11 months. The symptoms can be aggravated by bending, sitting, lifting, rapid, forceful movements, turning over in bed, and difficulty with rising from a seated position. Relief may be noted with rest, standing, or walking. Patients rarely described any associated neurologic symptoms (10, 46, 93).

Pain distribution in SIJ syndrome has been complicated by the overlap of pain patterns created by other surrounding structures. Disc, facet, and muscle-related conditions can have a clinical presentation similar to that of SIJ. Fortin et al. attempted to map the pain emanating from the SIJ and determine if the SIJ had a characteristic pain pattern (134). Using provocative injections on asymptomatic subjects, they mapped an area of hyperesthesia in the buttock localized to 10 cm distal to and 3 cm lateral to the PSIS (Fig 5.17). In a subsequent study, they successfully screened SIJ syndrome patients from a group of low back pain patients, suggesting that pain maps can be successfully used to make the diagnosis (135).

However, similar conclusions regarding the pain maps of SIJ patients were not made by Dreyfuss et al. (135). They found varied and broad patterns of pain referral encompassing the en-

Figure 5.17. The sacroiliac joint pain referral map produced by injection. (Adapted from Fortin JD, Dwyer AP, West S, et al. Sacroiliac joint: pain referral maps on applying a new injection/arthrography technique. Parts I and II. Spine 1994;19(13):1475–1489.)

tire leg in patients with and without SIJ pain. They also found most of the characteristic features of the history in patients with SIJ pain were neither sensitive nor specific, except for the relieving factor of standing, which had high specificity but suffered from a low sampling rate.

Physical Examination

On examination, patients may present with a limp or difficulty weightbearing on the painful side. Postural inspection may reveal torsion and lateral deviation of the pelvis, unleveling of the iliac crests, and flattening of the buttocks on the side of joint restriction (136). Range of motion of the lumbar spine may be painfully limited in flexion or extension. On palpation, point tenderness is found in and about the PSIS. Tender or trigger points may be found in the gluteal muscles and lumbar paraspinal musculature. The straight leg raising test may be decreased owing to back pain or tight hamstrings, without evidence of nerve root tension signs. Lavignolle et al. demonstrated the influence of the straight leg raising test, noting that distinct movement at the SIJ was seen at 60° (75). The patient may report paresthesia or a subjective decrease in sensation to light touch, but have no neurologic deficits. Weakness noted on examination is typically the result of pain or muscle imbalances as opposed to hard neurologic findings.

Orthopaedic tests can also be used to provoke SIJ pain and rule out the involvement of surrounding structures as possible sources of pain. The common orthopaedic tests used to assess the SIJ are the Yeoman, Gaenslen, Patrick, and SIJ shear test. In Yeoman's test, the SIJ is stressed by producing rotation of the ilium by the extension of the leg, while pressure is placed over the ipsilateral joint of patient who is lying prone. In Gaenslen's test, the patient is lying supine as the contralateral hip is maximally flexed while the ipsilateral hip is extended. Patrick's test stresses the SIJ and the hip by placing the hip in a position of flexion, abduction, and external rotation. In the SIJ shear test, the patient lies prone while a thrust is applied over the posterior iliac wing in an inferior direction with the palm of the doctor's hand (46). In all these tests, reproduction or aggravation of the SIJ pain is sought. Care must be taken to rule out pathology from spinal, hip, or knee joints, or from muscles that may be stressed during the testing process, which could lead to an incorrect diagnosis.

However, because of the unusual location and oblique three-dimensional orientation of the SIJ, direct examination (i.e., palpation) is impossible. In addition, because the SIJ has normally wide-ranging anatomic and movement variations, it is difficult to ascertain what is normal and what is abnormal. Tests assessing the mobility of the SIJ include motion palpation tests (e.g., the Gillet tests) and tests assessing joint play (136a). The procedures used in motion palpation of the SIJ have been previously described by others (46, 136–138). Tests have been developed to assess the movement of the SIJ and its surrounding structures. Tests subjectively assess the qualitative and quantitative nature of the movement between the ilia and sacrum as well as the influence of the surrounding structures. Results are used in diagnosing and in making decisions regarding which manipulative procedure is to be used.

Researchers have investigated the reliability of many tests used in the physical examination of the SIJ. Potter and Rothstein (76) found that of the 13 SIJ tests assessed only two, iliac gapping and compression tests, had acceptable levels of reliability. Interestingly, these two tests relied solely on the patient's pain response to the therapist's action, rather than an assessment of joint motion. Dreyfuss et al. reported 20% of asymptomatic individuals had a positive finding with several of the SIJ tests assessed, and they concluded that asymmetry of SIJ motion caused by relative hypomobility can occur in asymptomatic subjects (135).

Laslett and Williams also assessed the reliability of selected pain provocation tests for SIJ pathology (139). They found moderate to almost perfect agreement with five of the seven tests selected and performed on patients with low back pain. However, sensitivity and specificity values could not be calculated. Mierau found that a positive Gaenslen's test for SIJ pain predicted an abnormal-appearing SIJ radiograph (132). Similar conclusions could not be reached with the Patrick or Yeoman tests. Dreyfuss et al. also assessed the value of physical examinations on the diagnosis of SIJ pain (140). Using anesthetic blocks as the "gold standard," they concluded that no single one of the 12 SIJ tests nor a combination of them demonstrated worthwhile diagnostic value. The 12 tests included three pain drawings, pointing to the painful area, sitting with partial buttock elevation from a chair on the affected side, Gillet test, thigh thrust, Patrick's test, Gaenslen's test, midline sacral thrust, sacral sulcus tenderness, and joint play. Similar conclusions were made by Maigne et al., who questioned the accuracy of the presumed SIJ provocation tests (85). Paydar et al. assessed the intra and interexaminer reliability of several SIJ tests (iliac crest height, tenderness of PSIS, sitting flexion test) and found only tenderness over the PSIS had acceptable agreement beyond chance (141).

Unfortunately, motion palpation tests fared no better than the physical examination tests described above. Mior and McGregor also reported slight to fair agreement between and within examiners of the Gillet test for SIJ dysfunction (142). In another study, Decina and Mior used symptomatic, asymptomatic, and ankylosis spondylotic patients to assess the reliability and validity of the Gillet test in detecting motion of the SIJ (143). The findings revealed poor to slight agreement beyond chance. Examiners could not detect which of the patients had fused joints, thus questioning the validity of the test. On the contrary, Herzog et al. reported that the reliability of the Gillet test was statistically significant; however, they reported only percent agreement (143a).

Considerable discrepancy exists between researchers and clinicians regarding the clinical utility of diagnostic findings and tests in accurately diagnosing SIJ pain. Increasing evidence indicates that the SIJ plays a role in the genesis of low back pain but the mechanism underlying this explanation and the methods and information used to detect them are still uncertain. Although empirical evidence abounds, much remains to be

learned and researched. At this time, the diagnosis of SIJ syndrome may be one of exclusion, and perhaps it is contingent on successful intervention.

Imaging

Several imaging techniques can be used to assess the integrity of the SIJ. Choice depends on the suggested diagnosis and the availability of the imaging modality. Plain film radiographs provide visualization of the alignment, cortical outline of the bony structures, joint spacing, and surrounding soft tissues. Plain film is limited, making early detection of sacroiliitis difficult. Further, understanding the normal aging pattern in the joint will prevent making a pathologic diagnosis. For example, the iliac cartilage degenerates earlier than the sacral cartilage. After 30 years of age, radiographic changes include narrowed joint space, subchondral sclerosis, erosion, ankylosis, osteophyte formation, subchondral cyst, and joint asymmetry (119). After 40 years of age, these findings (except erosion and ankylosis) may not be considered pathologic or may warrant other imaging modalities.

Early degenerative and inflammatory bony changes can be detected more effectively with scintigraphy using technetium phosphate compared with computed tomography (120–126, 132). As ankylosis progresses, radioactivity progressively falls making this procedure less effective. It should be noted that nonsteroidal anti-inflammatory drugs also diminish SIJ uptake in scintigraphic procedures. The procedure is not useful in assessing the immature SIJ (127–132).

Stress views of the SIJ may be warranted if hypermobility or instability is suspected. Relative movement of the SIJ and pubic symphysis is assessed with the patient standing and bearing weight first on one leg and then the other (Champerlain's views). The radiographs are assessed not only for pathology but also for the degree of vertical displacement of the pubis (62) (see above for measures).

PAIN MANAGEMENT

Management of SIJ pain is obviously dependent on the diagnosis and on correcting the causative factor(s). Most mechanical SIJ pain can be effectively managed by a variety of conservative measures. These measures are directed at restoring the movement of the joint; eliminating or decreasing the pain; correcting imbalances in length and strength of the muscles of the trunk, pelvis, and lower limbs; educating the patient to avoid or modify causative factors; using supportive devices; and providing rehabilitation protocols when necessary. Manipulation and muscle strengthening exercises provide satisfactory outcomes to SIJ pain of mechanical origin (46, 144, 145).

In a prospective trial, 90% of patients who were diagnosed with chronic SIJ syndrome responded favorably to daily side posture high-velocity manipulative–adjustive treatments over a 2- to 3-week period (69). Cox reported similar positive outcomes in his study (146). Dontigny reported that manual correction of the SIJ provided relief to 90% of low back patients (81). However, attention must be paid to both the joint dysfunction and the surrounding soft tissues.

Surrounding soft tissue injuries may perpetuate the joint dysfunction or, in some cases, mimic an SIJ syndrome (Fig. 5.18). In patients suspected of presenting with myofascial pain syndromes, reproduction of symptoms by provocative trigger point examination helps make the diagnosis and develop the appropriate treatment plan (35, 147, 148). Many approaches can be employed in the management of soft tissue injuries, and knowledge and skill in applying them is essential. Some of the approaches include stripping or transverse friction massage, spray and stretch techniques using vapocoolant sprays, counterstrain technique, muscle energy techniques, and proprioceptive neuromuscular facilitation, to name but a few.

In some cases, conservative care may not be effective in managing the patient's pain. In such cases understanding the underlying causative factors is important. For example, patients presenting with acute inflammatory conditions (e.g., ankylosing spondylitis) should not undergo high-velocity thrust manipulations or adjustments to the involved joint as these can aggravate the symptoms and prolong recovery. In this situation, the adjustment is an absolute contraindication (149). In other cases, intra-articular injections have been helpful (150). In cases of joint hypermobility, a sacroiliac or trochanteric support may be helpful (93). More severe injuries (e.g., pelvic fractures) may require surgical stabilization. (151–153).

In summary, the clinician should first consider the diagnosis and then develop a plan of management that includes the expected outcome after a certain number of visits. Patients with noncomplicated SIJ dysfunctions typically respond favorably after seven to nine visits of side posture manipulation. If a patient fails to show evidence of improvement, further investigation may be necessary or an altered approach to management should be considered.

Manipulative–Adjustive Procedures

Many manipulative–adjustive procedures have been described for the management of SIJ syndrome. They revolve around contacts on either the innominate or the sacrum. The decision as to which should be used has been based on the theories set forth by proponents of motion palpation or those using various radiographic mensuration techniques. Recent research suggests that different loads are transmitted through the pelvis depending on the manipulative thrust selected and the position of the patient (145). This may play a role in deciding which technique is selected based on the patient's tolerance to the applied external loads and to the individual's ability to be properly positioned for the thrust.

Two of the most common manipulative procedures used in the management of fixations (subluxations) of the SIJ are for corrections of an anterior and posterior innominate. These are described and illustrated below. These procedures can be modified by altering the hand contact position (e.g., correcting dysfunctions of the sacrum are made by contacting the sacrum and applying force opposite to the direction of the lesion) (136).

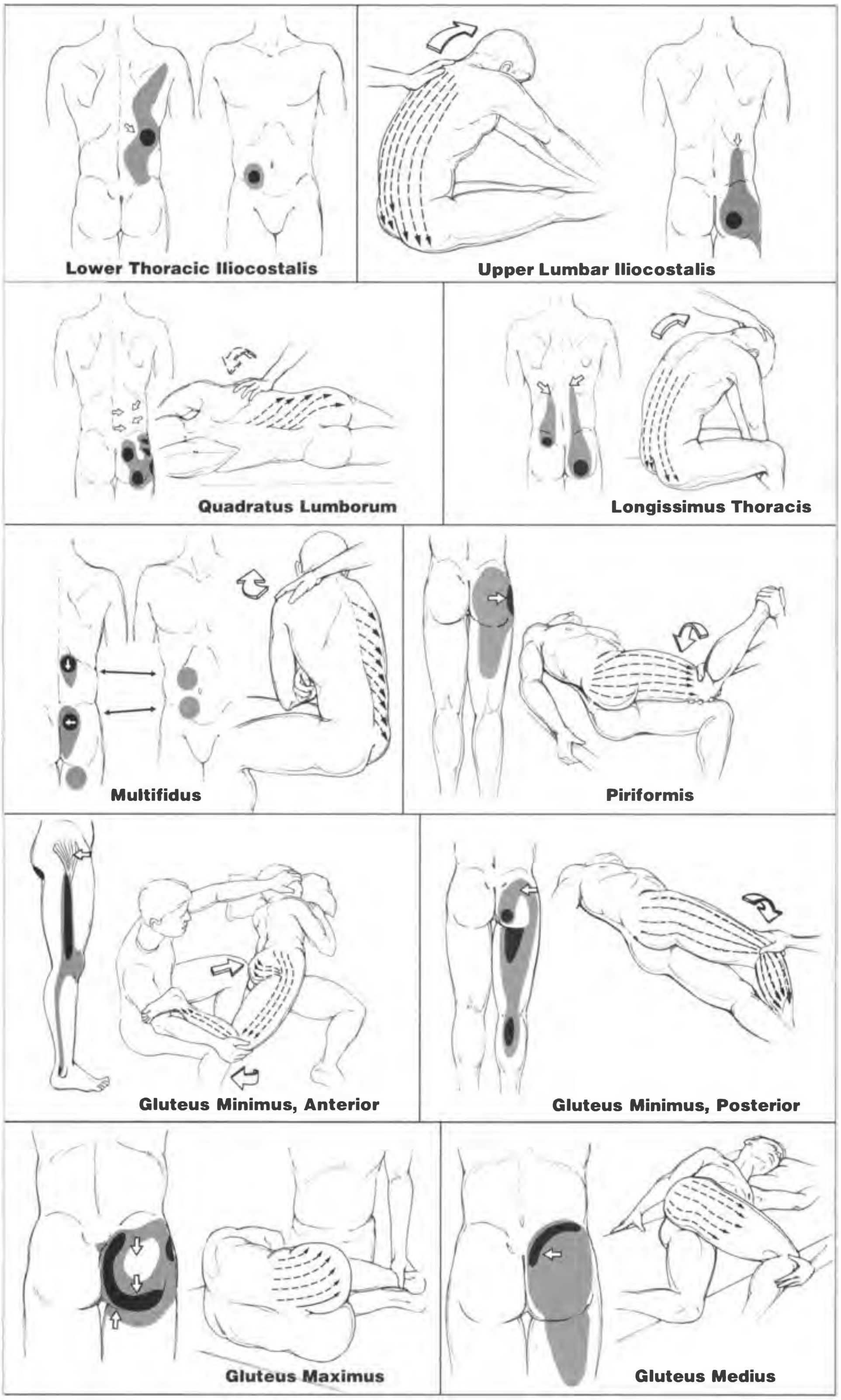

Figure 5.18. Illustration of the trigger points (Tp) and the referral pattern of the various muscles involved in pelvic movement and which may mimic pain about the sacroiliac joint. The location of the Tps (*short straight white arrows*) and referral patterns (*stipples*), stretch positions and spray patterns (*dashed arrows*). (Reprinted with permission of Simons DG. Myofascial pain syndromes due to trigger points. In: Goodgold J, ed. Rehabilitation Medicine. St. Louis: CV Mosby, 1988:686–723.)

Anterior Innominate

In the anterior innominate procedure the intent is to move the upper innominate posteriorly by using both the leg and the ischial tuberosity as levers. The patient is laterally recumbent with the involved side up and the up knee is flexed. The doctor's cephalad hand contacts the shoulder to stabilize the upper body to limit lumbar rotation, while the caudad hand contacts the ischial tuberosity. The thrust is made in an anterior cephalad direction on the ischium while the patient's knee is further flexed (Fig. 5.19).

Posterior Innominate

The patient is laterally recumbent with the involved side up, and the up knee flexed for the posterior innominate procedure. The doctor's cephalad hand is place upon the patient's shoulder to stabilize the upper body and limit lumbar rotation. The caudad hand contacts the PSIS and thrusts in an anterior direction, while the doctor distracts the up leg down by dropping the body (Fig. 5.20).

Figure 5.20. High-velocity posterior superior iliac spine (PSIS) manipulative procedure for a posterior innominate.

Flexion-Distraction Procedures

The basic procedure in flexion-distraction therapy to manage SIJ syndrome is to use the anatomic position of the innominates as a guide in selecting the proper technique. The position of the innominate is determined by inspection and examination. For example, in a posterior innominate the leg will appear short and the ilium will seem high and posterior on the involved side; spasm or trigger points will be found in select gluteal and lower limb muscles, and an apparent enlargement of the obturator foramen and lessening of the pelvic outlet vertical height on radiographs will be seen.

Figure 5.19. High-velocity ischial manipulative procedure for an anterior innominate.

Anterior Innominate

A block is placed under the ASIS and traction is applied to the innominate. If both innominates are anterior, a Dutchman's roll can be placed under the ilia and both ilia can be distracted simultaneously. A hand contact is placed on the L5 spinous process in a cephalad direction and held for 20 seconds while pumping the caudal section of the table up and down 2 inches. This 20-second distraction is repeated three times (Figs. 5.21 and 5.22).

Posterior Innominate

A block is placed under the acetabulum and traction is applied to the innominate. A hand contact is placed on the L5 spinous process in a cephalad direction and held for 20 seconds while pumping the caudal section of the table up and down 2 inches. This 20-second distraction is repeated three times (Fig. 5.23).

Mobilization Procedures

In patients who present with a hypermobile SIJ or in acute pain, it may be beneficial to first mobilize the joint. The patient can be positioned in a supine or lateral recumbent position, while the doctor contacts either the ischium and ASIS (to induce posterior ilium rotation) or the PSIS and AIIS (to induce anterior rotation). Repetitive low-amplitude, successive pressure is applied in an attempt to produce movement in the joint. Shallow, controlled thrusts may also be used to patient tolerance. Using mobilization techniques while the patient produces a muscle contraction (e.g., modification of proprioceptive neuromuscular facilitation technique) may also be helpful in facilitating the joint movement.

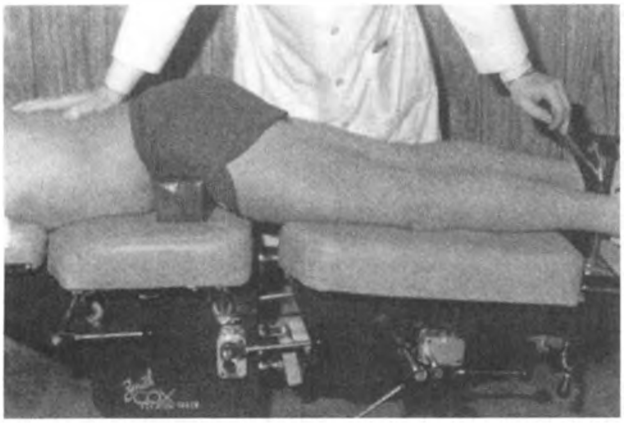

Figure 5.21. Flexion-distraction procedure for a unilateral anterior innominate.

Figure 5.22. Flexion-distraction procedure for bilateral anterior innominate using a dutchman roll.

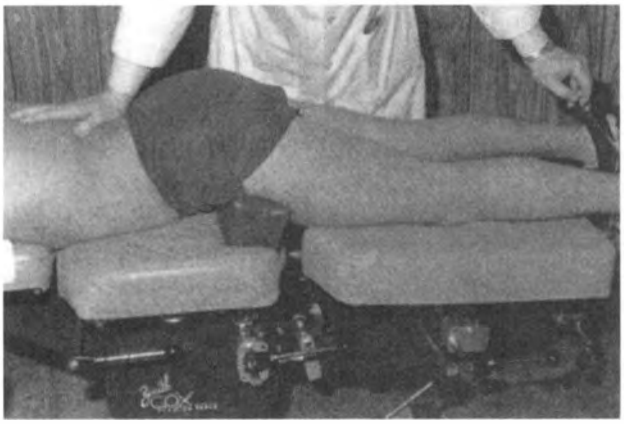

Figure 5.23. Flexion-distraction procedure for a unilateral or bilateral posterior innominate.

Exercise Procedures

As discussed, the biomechanical modeling of the joint's behavior places a significant role on the appropriate function and balance of the trunk, pelvis, and lower limb muscles. Understanding the biomechanical behavior of a joint ensures that attention is paid to structures other than the joint itself. Kuchera stresses that attention must be paid to the soft tissues by facilitating muscle re-education and postural balancing (154). In his opinion, the most common cause of somatic dysfunction is a faulty movement pattern resulting from muscle imbalance and postural overstrain. The clinician should carefully assess the muscles and provide appropriate range of motion stretching and strengthening exercises. For example, clinical experience suggests that treating a posterior innominate should include balancing the gluteal muscles by relaxing the gluteus maximus and strengthening the gluteus medius and minimus; strengthening the quadriceps and stretching the hamstrings; and strengthening the internal and external abdominal muscles. The abdominal muscles may play a role because they have been shown to be moderately to very actively involved during unconstrained erect standing (155).

However, clinicians should avoid the "cook book" approach to any exercise procedure or protocol. Instead, they should assess and provide each patient with an individualized exercise program that addresses the tight and weak muscles, especially those found to play a role in stabilizing the SIJ (i.e., gluteus maximus, biceps femoris, piriformis, and psoas) (64, 95).

Table 5.1

The Functional Restoration Program

Phase	Goal	Activity
Phase 1	Decrease pain and inflammation	Ice and modalities, nonsteroidal anti-inflammatory drugs Postural education and muscle therapy
Phase 2	Restore range of motion	Manipulation/mobilization Flexibility and muscle balancing, gait Dissociative movements (beginning) Elementary stabilization
Phase 3	Improve strength and stability	Intermediate/advanced stabilization Proprioceptive retraining Dissociative movements (intermediate/advanced) Plyometrics, resistive exercises and weights
Phase 4	Return to work or play	Task- or work-specific activities

Modified from Fortin JD. Sacroiliac joint dysfunction: a new perspective. Journal of Back Musculoskeletal Rehabilitation 1993;9(2):407–418.

Rehabilitation

Functional restoration programs should be implemented as soon as possible and designed with the individual patient's need

in mind. A program for an elite athlete should be considerably different from that for a sedentary office worker. The fundamental components of the program are similar, however, and have been summarized by Fortin (95). Fortin divides the functional restoration program into four phases, each designed to attain a specific goal (Table 5.1). The goal with any program is to facilitate the patient's return to activities of daily living and to try to avoid, or limit, the opportunities for symptom recurrences.

CONCLUSION

In the opening quote to this chapter, Osler reminds us that while finding answers to questions we have empirically accepted, we will undoubtedly discover that we actually have only few answers. This is especially true of current understanding of the SIJ. This chapter presented information related to the anatomy, biomechanics, clinical presentation, and management of related conditions of the SIJ, although it is not a comprehensive treatment.

In this chapter we have presented the view that the SIJ plays a role in the genesis of low back pain. Its diagnosis is dependent on the integration of the history and physical examination. It also necessitates that the clinician rule out other more common and sinister conditions. Conservative management has been found effective, with invasive interventions required only in severe traumatic conditions. The nature of the mechanics of the SIJ requires that attention also be paid to the surrounding soft tissues. Further research is necessary to confirm and add to the current level of knowledge of this important, yet enigmatic joint.

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CHAE SONG RO, M.D., Ph.D

Chae Song Ro, MD, PhD, passed away during the writing of The Sacroiliac Joint chapter of this text. He had contributed to the same chapter in the 5th edition of this textbook as well.

Dr. Ro was a very special person, beloved and respected by friends, family, students, and those who worked with him. He was Korean by nationality, graduated from medical school at Kyung-Sung University (which later became Seoul University), served in the Korean Navy during the Korean War, and became a diplomate of the Board of Psychiatry (Korea). He served as the chief of the neuropsychiatry section of the Korean Naval Hospital from 1958 to 1961. He taught at Seoul University and Kyung-Hee University and earned his PhD from Seoul University in 1968. He practiced at the Ro Mental Hospital in Seoul, Korea until migrating to the United States in 1978. In 1981 he began teaching at the National College of Chiropractic, in the Department of Anatomy.

Dr. Ro's training as a medical doctor and a PhD brought a great deal of understanding, knowledge, and clinical importance to his teachings. He was known for his beautiful drawings, which are handsomely presented in this book. Very active in research and scholarship, Dr. Ro collaborated with William Bachop, PhD, professor of anatomy at National College, to determine the normal position and relationship of the anterior primary division, fibers from the sympathetic chain contributing to the recurrent meningeal nerve, spinal ramus of the segmental artery, and transforaminal ligaments within the intervertebral foramina.

Gregory Cramer, DC, PhD, director of research at National College, states that Dr. Ro was the world's leading authority on the anatomy of the sacroiliac joint. His writings have been read by thousands of chiropractors and have helped them understand this important region while being used to develop the best treatment protocols for countless patients with pain arising from the sacroiliac joint. His conclusions were based on thorough review and interpretation of the literature and methodical observation from meticulous dissections.

In addition to his successful teaching and writing, Dr. Ro's most distinctive characteristics were his sincere and warm smile and his humble, gentle, yet confident demeanor. Active in his church and community life, in his family life, as a practicing physician, as a teacher, and as a scholar, Dr. Ro will be remembered most of all as a man of true grace.

My greatest remembrance of Dr. Ro was seeing him dissecting in the laboratory at National College one day, and upon asking what he was studying, he replied "I am dissecting out the sensory nerve supply of the lumbar spine and intervertebral disc." This happened 10 years ago, the time when the pain sensitivity of the disc was being actively studied. Dr. Ro, true to his life, was a leader in that important work. Dr. Ro is appreciated and remembered for his contribution to this chapter and it stands as a tribute to his life.

I wish to thank Gregory D. Cramer, DC, PhD, for his help in preparation of this memorial to Dr. Ro.

James M. Cox, DC, DACBR

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Transitional Segment

James M. Cox, DC, DACBR

chapter 6

Genius may be described as the spirit of discovery . . . It is the eye of intellect, and the wing of thought . . . It is always in advance of its time . . . (the) pioneer for the generation it precedes.

—Simms

A chiropractic multicenter observational pilot study on the examination procedures, diagnosis, and treatment of low back pain revealed that the condition requiring the greatest number of days and visits to attain maximal relief was the transitional segment (1). The same conclusion was reached by Schwerdtner (2) in discussing the causes of relapse of low back pain following chiropractic treatment. In a study of 165 patients with therapy-resistant recurrent pain after manipulative treatment, he cited three factors that particularly contributed to the relapse:

1. Inadequate stability.
2. Inadequate muscular balance of the lumbar-pelvic-hip region.
3. Inadequate attention to the acquired peculiarity of lumbosacral transitional anomalies.

Schwerdtner states that the transitional vertebra is recognized as a factor predisposing to low back pain. He also states that rotation manipulation is contraindicated in asymmetric disturbances of the lumbosacral transitional area. The best treatment is traction manipulation in the neutral position together with gentle muscle technique.

Bressler and Deltoff suggest, on the basis of clinical inference, that lumbosacral anomalies may predispose to aberrant sacroiliac mechanisms, which ultimately will lead to inefficient biomechanical adaptations and symptomatic manifestations (3).

One hundred twenty patients with hemisacralization of the L5 vertebra associated with low back pain underwent clinical, conventional radiograph, and computed tomography (CT) examinations. More than 50% of the patients had scoliosis. One third of the patients had disc lesions. Sacroiliac joint sclerosis was noted in two thirds of the cases. Almost all patients had an articulation between the hemisacralized side of L5 and the sacrum, which usually showed degenerative changes. Clinical symptoms were correlated to these CT and radiographic findings (4).

Figures 6.1 and 6.2 reveal a classic bilateral pseudosacralization of L5. Four lumbar vertebrae are seen above this level, with degenerative disc disease at the L3–L4 and L4–L5 segments. Traction spurs of the anterior vertebral body plates at these two levels, coupled with the marked loss of the disc space and some evidence of vacuum phenomenon, indicate longstanding degenerative change. Based on the work of Cailliet (5), approximately 70 to 75% of lumbar flexion occurs at L5–S1, with 20 to 25% observed at L4–L5, and only 5 to 10% at L1–L3 (Fig. 6.3). Because the thoracic spine does not participate in flexion-extension movements (most lumbar flexion occurs at L5–S1), it is estimated that 75% of the 219° range of total spinal flexion-extension movement occurs at the lumbosacral joint (6). Degeneration of the L5–S1 disc places the paramount responsibility for motion on the cephalad disc levels. These discs often are seen to degenerate in a domino effect, beginning at L4–L5 and moving upward.

Except for facet tropism, a change in the number of mobile vertebrae in the lumbar spine is the most significant congenital vertebral anomaly that can cause low back pain. Lumbarization of the first sacral vertebra (giving the individual, in effect, six lumbar vertebrae) increases the lever arm of the lumbar spine and causes greater stress on the lumbar spine and the lumbosacral joint. In contrast, sacralization of the fifth lumbar vertebra (reducing the number of mobile vertebrae in the lumbar region to four) is unlikely to cause symptoms when the entire vertebra is solidly incorporated into the sacrum. Occasionally, only one transverse process articulates with the

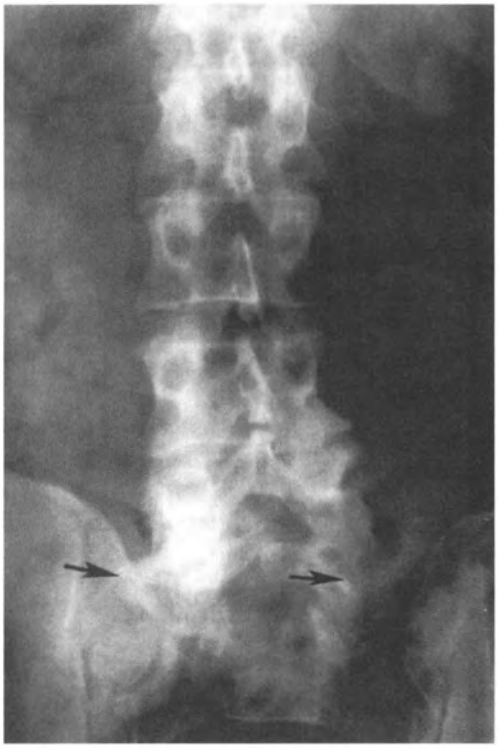


Figure 6.1. The *arrows* point to the overdeveloped, spatulated transverse processes of L5 that form the pseudoarticulations with the sacrum. Note the four lumbar segments above.

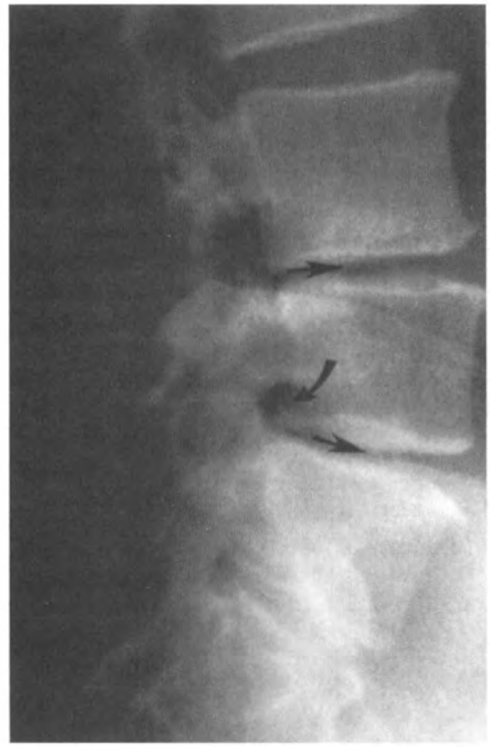
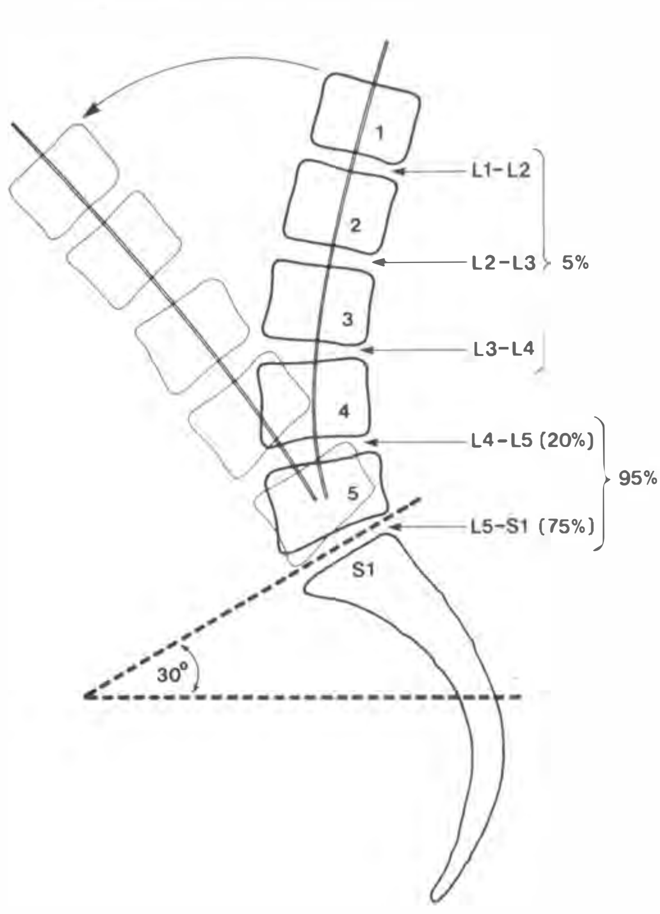


Figure 6.2. Lateral view of the patient in Figure 6.1 shows degenerative changes of the L3–L4 and L4–L5 discs (*straight arrows*) with traction spurs of the anterior and lateral body plates. Note the narrowing of the L4–L5 intervertebral foramen (*curved arrow*).

Figure 6.3. Segmental site and degree of lumbar spine flexion. The degree of flexion, noted at each segment of the lumbar spine as a percentage of total lumbar flexion, is indicated. The major portion of flexion (75%) occurs at the lumbosacral joint, 20% of flexion may occur at the L4–L5 interspace, and the remaining 5% is distributed between L1–L4. The forward-flexed diagram indicates the mere reversal past lordosis of total flexion of the lumbar curve. The lumbosacral angle is computed as the angle from a base parallel to horizontal and the hypotenuse drawn parallel to superior level of the sacral bone. The optimal physiologic lumbosacral angle is about 30°. (Reprinted with permission from Weinstein PR, Ehni G, Wilson CB. Lumbar Spondylolysis: Diagnosis, Management, and Surgical Treatment. St. Louis: Mosby Book, 1977:14. Originally based on Cailliet R. Low Back Pain Syndrome. 2nd ed. Philadelphia: FA Davis, 1968.)



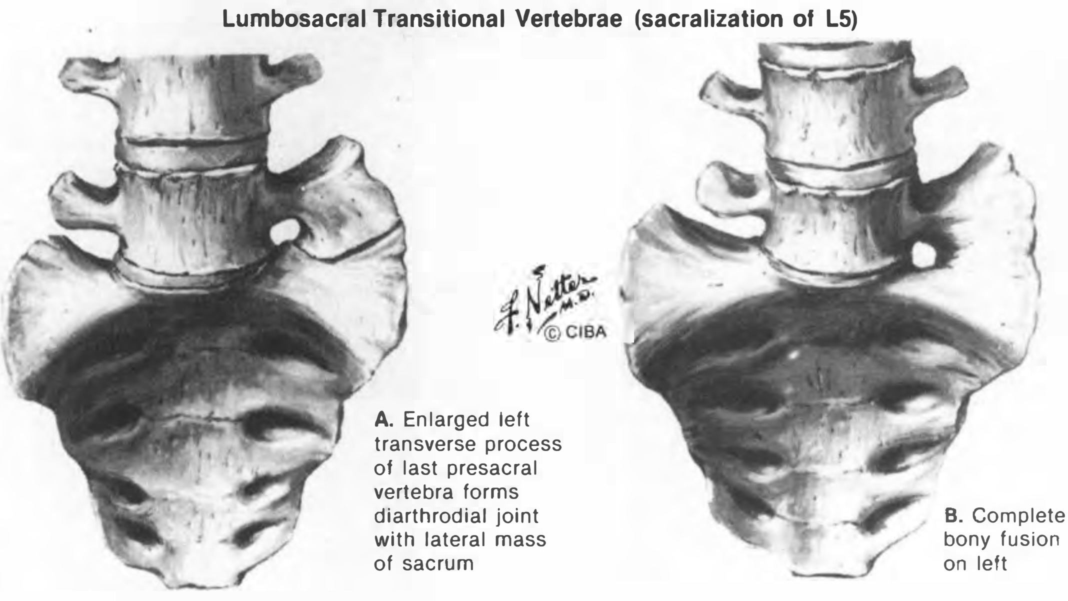


Figure 6.4. Illustration of lumbosacral transitional vertebrae (sacralization of L5) by Frank H. Netter, MD. (Reprinted with permission from Keim HA, Kirkaldy-Willis WH. Clinical Symposia. Ciba Found Symp 1980;32(6):9, 1980. Copyright, 1980. [Novartis. Reprinted with permission from Clinical Symposia, 32/6, illustrated by Frank H. Netter, MD.] All rights reserved.)

sacrum, altering spinal mechanics and resulting in severe instability and stress (7).

Unilateral lumbar vertebra sacralization or sacral vertebra lumbarization produces a condition known as Bertolotti's syndrome (Fig. 6.4), which has been diagnosed with increasing frequency in the past 10 years. Unilateral contact places unusual stress on the spine, and the resulting torque movements often cause herniation of the disc one level above the sacralization or lumbarization. Herniation, in turn, produces symptoms of nerve root entrapment. In the patient with Bertolotti's syndrome, surgery to decompress the herniated disc should always include spinal fusion to weld the affected vertebrae together so that further torque stresses are eliminated.

Wigh (8) found that none of 42 patients with a transitional segment who underwent disc surgery had any sign of disc protrusion at the level of the transitional segment. The disc at the transitional segment was hypoplastic, with the stress placed on the segment directly above it. Therefore, in an L5 transitional segment, the L4–L5 disc is under stress, and the level of protrusion will be found here, whether or not contained.

CLASSIFICATION OF LUMBOSACRAL TRANSITIONAL VERTEBRAE

Morphologic and clinical characteristics with respect to herniated nucleus pulposus were used to develop a classification of lumbosacral transitional vertebrae (9) (Fig. 6.5).

Type I. Dysplastic transverse process. (*A*) Unilateral; (*B*) bilateral. A large triangular-shaped transverse process, measuring at least 19 mm in width is seen.

Type II. Incomplete lumbarization or sacralization. (*A*) Unilateral; (*B*) bilateral. A large transverse process forms a pseudoarticulation between the transverse process and the sacrum. This appears to be a diarthrodial joint.

Type III. Complete lumbarization or sacralization. (*A*) Unilateral; (*B*) bilateral. A true bony union exists between the spatulated transverse process and the sacrum.

Type IV. Mixed. The patients who fall into this category exhibit type II (pseudoarticulation) on one side and type III (bone fusion) on the other.

The terms "lumbarization" and "sacralization" were not used because the total number of vertebrae in the patients' spines could not be determined.

The sex distribution of lumbosacral anomaly showed a greater incidence in men (71.5%) than in women (28.5%).

Conclusion

Type II unilateral or bilateral pseudoarticulation between the transverse processes and sacrum has the highest incidence of disc herniation at the disc above the transitional segment. Types I, III, and IV do not produce any higher incidence of disc herniation above the transitional segment.

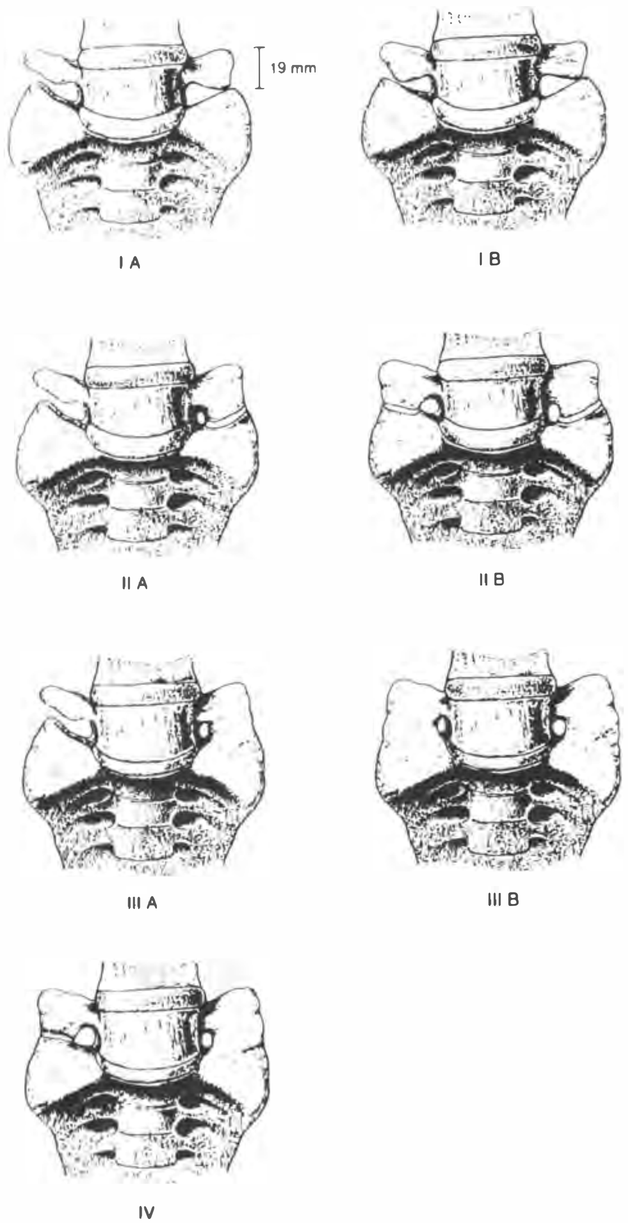


Figure 6.5. Classification of lumbosacral transitional vertebrae according to radiomorphologic and clinical relevance with respect to lumbar disc herniation. (Reprinted with permission from Castellvi AE, Goldstein LA, Chan DPK. Lumbosacral transitional vertebrae and their relationship with lumbar extradural defects. Spine 1984;9(5):493–495.)

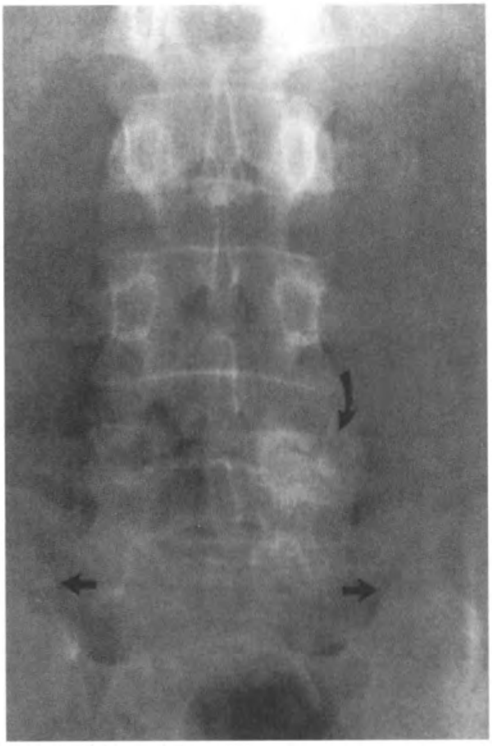


Figure 6.6. Note the bilateral sacralization of the fifth lumbar vertebral transverse processes (*straight arrows*). Also, the fourth lumbar right superior articular facet hypertrophy is shown by the *curved arrow*.

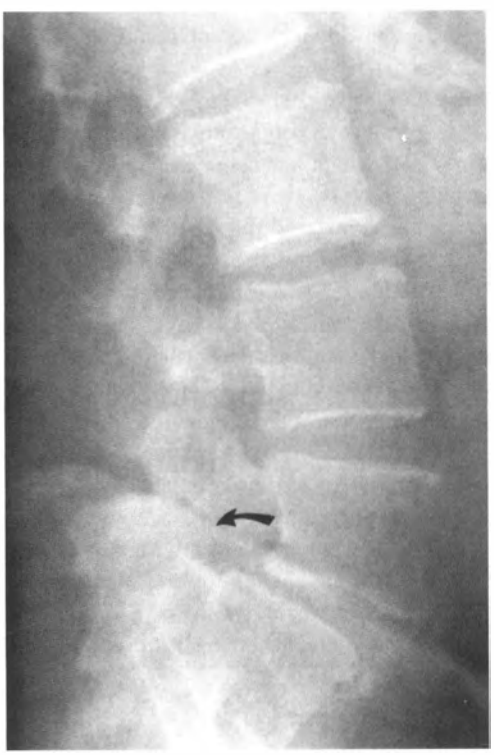


Figure 6.7. L4 is a true spondylolisthesis with the *arrow* denoting the pars interarticularis fracture.

Cases 1 and 2

A 40-year-old white chiropractor (case 1) had low back pain for several years, but in the last 4 years it had been getting progressively worse, to the point that at times his right leg gave out from under him when he was walking. He stated that both legs felt asleep.

Examination failed to reveal any motor or sensory change of the lower extremities. The straight leg raises were negative except for some shortness of the hamstring muscles. The deep reflexes were +2 bilaterally. The gluteus maximus and hamstring muscles were grade 5 of 5 strengths.

Radiographic examination (Figs. 6.6 and 6.7) showed one of the most interesting studies I have ever seen. Figure 6.6 revealed a transitional fifth lumbar segment, namely a sacralized fifth lumbar vertebra. Note the hypertrophy of the right superior L4 articular facet. The pars interarticularis fracture is visualized on lateral projection.

Here, we see a forward slippage of the fourth lumbar vertebral body on a transitional fifth lumbar segment. This is truly a Bertolotti's syndrome with actively degenerating disc tissue and annular fiber stress of the L4–L5 level.

Figures 6.8–6.10 (case 2) reveal a transitional L5 segment and true spondylolisthesis at L4. Cases 1 and 2 represent the only two cases I have seen in clinical practice of a transitional segment with spondylolisthesis at the segment above. Case 2 is courtesy of Alice Wright, DC, Hatfield, Pennsylvania.

In the treatment of transitional L5 segment, a Dutchman flexion roll is placed under the L5 segment as shown in Figure 6.11. The contact is placed on the spinous process of L4 while flexion distraction is applied with the caudal section of the table. A description of technique application to patients with and without

Figure 6.9. L4 is anterior on L5. Note the step defect (*arrow*) of the L5 anterior superior vertebral plate due to failure of epiphyseal development.

Figure 6.8. Both L5 transverse processes are overdeveloped and spatulated, forming pseudoarticulations with the sacrum (*arrows*). (This case is courtesy of Alice Wright, DC, Hatfield, PA.)

Figure 6.10. The *arrow* denotes the pars interarticularis fracture at L4.

Figure 6.11. Distraction is applied to a patient with a transitional segment of L5. A flexion Dutchman roll is placed under the L5 level, and the thenar contact of the doctor's right hand contacts the spinous process of L4, the vertebra above the transitional segment. A downward tractive force is applied to the caudal section of the table, while the spinous process is vectored cephalad. This force is applied until the doctor feels the space between the spinous processes under the contact hand become taut or begin to separate. This application of tractive force is applied repeatedly at about five or six such applications per 20-second period of time until such time as the desired motion is sensed by the contact hand. We then place the segments through the other ranges of motion as shown in Figure 6.12. The above technique is applied to the transitional segment, without spondylolisthesis above the level of transitional segment, as shown in Figure 6.2, in which the patient has a degenerative disc and loss of the intervertebral foramen vertical diameter at L4–L5, forming a facet syndrome at the L4–L5 level. If a spondylolisthesis is present above the transitional segment, the same procedure of care is followed *except* that the contact hand will be on the spinous process of the vertebra above the spondylolisthetic segment. Figure 6.9 shows this type of case.

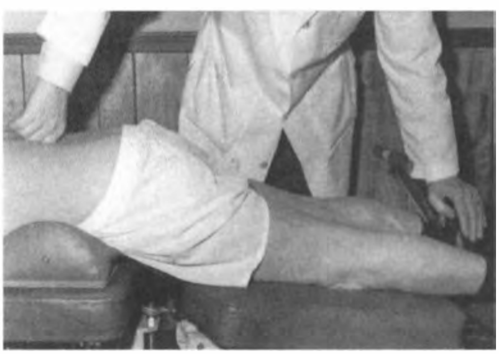


Figure 6.12. Lateral flexion is applied to the facet joints following the application of flexion distraction as shown in Figure 6.11. The spinous process is held firmly between the index finger and thumb as lateral flexion is applied with the caudal section of the Zenith-Cox instrument. The contact of the spinous process is done to ensure that the joints being adjusted are those directly caudal to the spinous contact. That is, if the fourth lumbar spinous process is contacted, only the facet joints between L4 and L5 are mobilized. *Remember*, in the absence of sciatica, the facets are moved through their full physiologic ranges of motion. If sciatica is present, *only flexion is performed until 50% relief of the leg pain is attained.*

Figure 6.13. Acupressure points are treated with deep compression before and after the application of manipulative procedures to the vertebrae found at lesion. These acupressure points are outlined in Chapter 12, *Care of the Intervertebral Disc Patient*.

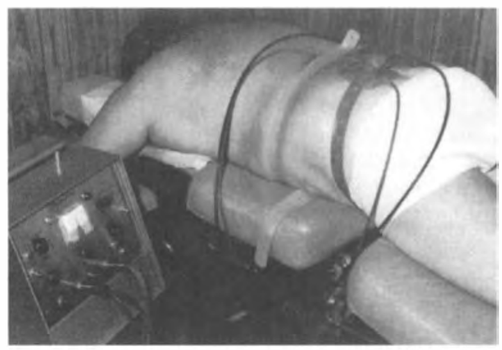


Figure 6.14. Tetanizing current is applied to the paravertebral muscles following manipulation, and sometimes prior to manipulation if muscle spasm is great.

Figure 6.15. A lumbar support orthosis is worn for stabilization in patients with great pain, especially if instability is found in the segment above the transitional segment.

spondylolisthesis above the level of transitional segment is shown in the legend of Figure 6.11.

Figure 6.12 shows the spinous process contact made while the lumbar segments are individually placed in lateral flexion. A flexion roll is left in place to maintain slight flexion of the lumbar spine to prevent further stenosis of the spondylolisthetic slip.

Figure 6.13 shows goading of the acupressure points B22 through B49 prior to and following flexion-distraction manipulation.

Figure 6.14 shows the application of tetanizing current to the paravertebral muscles following spinal manipulation. In addition, if the pain is severe, we apply alternating hot and cold packs as well; heat is applied for 10 minutes and cold for 5 minutes.

Figure 6.15 shows a belt placed on such patients for stabilization. This belt contains a memory foam insert that molds snugly against the lumbar spine regardless of the contour. Transitional segments do well with this type of support while healing takes place. We start the patients on exercises to regain abdominal strength and stretch the hamstring muscles; in the case of spondylolisthesis in the transitional segment, we also prescribe knee-chest exercises. Such exercises would be Cox exercises 2, 5, and 9, as shown in Chapter 12, *Care of the Intervertebral Disc Patient.*

Case 3

A 32-year-old white man, 71 inches tall, weighing 182 pounds, was seen for low back pain and numbness in the right foot. This pain had started 2 years previously with low back and complete right lower extremity pain and the patient had been treated by a chiropractor, who had relieved the leg pain, but not the numbness in the right foot. He then developed a severe antalgic posture approximately 1 month prior to seeing us. He also developed complete low back and right lower extremity pain but no testicle pain. He lost approximately 40 pounds. It was suggested that he have a laminectomy.

Examination revealed flexion at 30° and extension at 10°, both of which were extremely painful. Straight leg raising was positive on the right at 40°, creating both low back and leg pain, whereas the left leg created a well leg-raising sign at 80° with right low back pain. The right L5–S1 dermatomes were hypesthetic to pinwheel examination. This patient could heel and toe walk normally; however, the right gluteus maximus muscle was approximately grade 4 of 5 strength compared with the left. Circulation in the lower extremities was within normal limits.

Radiographic examination (Figs. 6.16 and 6.17) revealed a degenerative spondylolisthesis of L5 on S1, and the partes interarticularis are bilaterally intact. Anterolateral lipping and spurring are noted at the body plates of L4–L5. The transverse process of L5 is spatulated on the right side. The facets at L4–L5 appear to be tropic. Review of a myelogram taken by the surgeon revealed a large, central-type disc prolapse at the L4–L5 level and a smaller L5 right discal protrusion. Figure 6.18, an oblique view, reveals no pars interarticularis defect.

Treatment was instituted with the usual rule that, if at least 50% improvement was not obtained within 3 to 4 weeks of treatment, surgery would be recommended. Of course, with any increased motor weakness or cauda equina symptoms, surgery would be recommended earlier. The results of flexion-distraction manipulation were that, 6 days following the onset of treatment, the patient stated that he was "remarkably free of pain." He was started on Nautilus exercise consisting of extension. This patient had extremely short hamstring muscles, and stretching with proprioceptive neuromuscular facilitation was instituted. Acupressure points B22 to B54 were treated.

This is the only case of transitional segmentation with spondylolisthesis at the same level that I have ever seen.

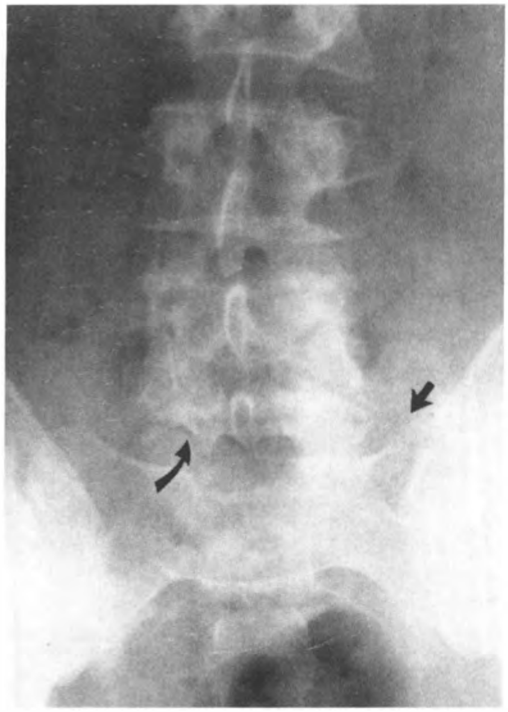


Figure 6.16. The posteroanterior x-ray study reveals the right transverse process of L5 to be spatulated *(straight arrow)*, whereas the left superior sacral facet forms an articulation with the lamina of L5 *(curved arrow)*.

Figure 6.17. A lateral projection view of the patient in Figure 6.16 shows that L5 is anterior on the sacrum, a pseudospondylolisthesis at the level of a transitional segment. This is an unusual condition, because pseudospondylolisthesis occurs 90% of the time at the L4 segment.

Figure 6.18. Oblique view shows an intact pars interarticularis at L5, the level of forward slippage shown in Figure 6.17.

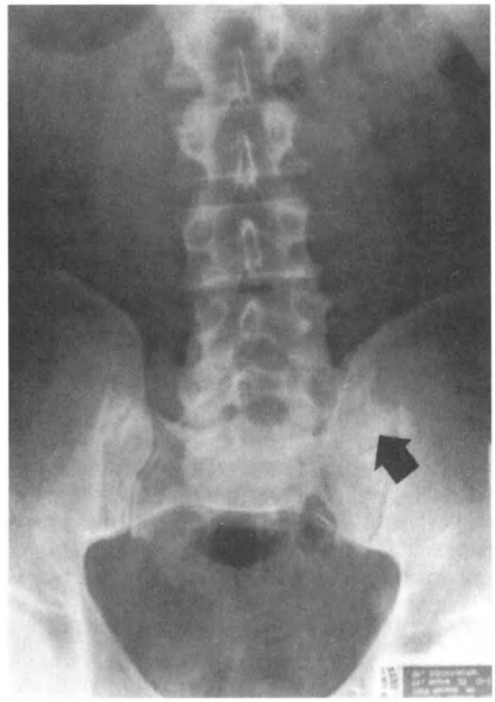


Figure 6.19. Left lateral flexion of the lumbar spine is seen, with left lateral flexion subluxation of L4 on L5 noted. The right transverse process of L5 is spatulated, forming a pseudoarticulation with the sacrum (*arrow*).

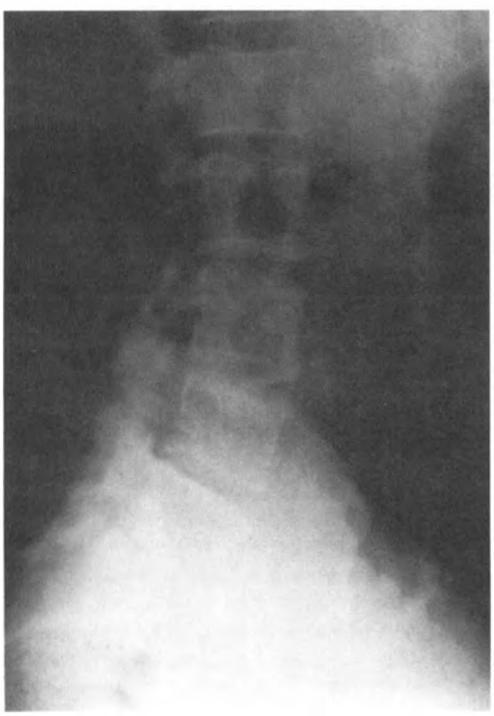


Figure 6.20. Note the rudimentary disc at L5–S1, the level of the transitional segment. The L4–L5 disc is degenerated as seen typically in Bertolotti's syndrome.

Figure 6.21. T2-weighted sagittal image showing the large L4–L5 disc protrusion and the T12–L1 degenerative disc disease and small disc protrusion. This is the presurgical magnetic resonance image.

Case 4

A 40-year-old man had right L5 dermatome pain lasting for approximately 1 month. He had seen a neurosurgeon and had a myelogram, and surgery was recommended.

Examination findings included straight leg raising positive on the right at approximately 20°. The deep reflexes were active and +2 equal bilaterally with no signs of motor weakness noted.

Radiographic examination (Figs. 6.19 and 6.20) revealed a levorotatory list of the lumbar spine with L4 in left lateral flexion subluxation on L5 and a transitional segment of L5 on the sacrum. This transitional segment has a large spatulated transverse process forming a pseudoarthrosis on the right side with the sacrum. Note the rudimentary disc at the L5–S1 level.

The diagnosis here is a Bertolotti's syndrome, meaning the combination of a transitional segment with an intervertebral disc protrusion above. To reiterate, this means that all the flexion and extension movement must now occur at the L4–L5 disc, although normally 75% of such movement occurs at the L5–S1 disc, which is now rudimentary, partially fused at the sacrum, and incapable of motion. This will lead to early degenerative change at the L4–L5 disc, eventually causing protrusion and compression of the L5 nerve root. This case represents a right L4 lateral disc protrusion.

Treatment consisted of 3 weeks of daily flexion distraction, which resulted in progressive relief of the leg pain. It is to be remembered that transitional segment requires more days and more visits than any other condition to achieve relief (1).

Case 5

A white 42-year-old woman complained of bilateral hip pain and left lower extremity pain into the plantar surface of the foot, described as a numb feeling. A year previously the L4–L5 disc had been surgically removed and Figures 6.21 and 6.22 show the large left central disc herniation, which contacts the thecal sac and fills the lateral recess.

Figures 6.23–6.25 are the anteroposterior, lateral, and tilt plain radiographs showing the degenerative L4–L5 disc disease (*arrows*), the right sacralization of the L5 transverse process, and retrolisthesis of L4 on L5, which exceeds 3 mm, confirming instability of L4 on L5. Discogenic spondylosis of the thoracolumbar spine is noted with T12–L1 degenerative disc disease and posterior disc protrusion (*see arrowhead* on Figure 6.21).

This is an excellent case of Bertolotti's syndrome—unilateral L5 transverse process spatulization and L4–L5 disc degeneration and herniation.

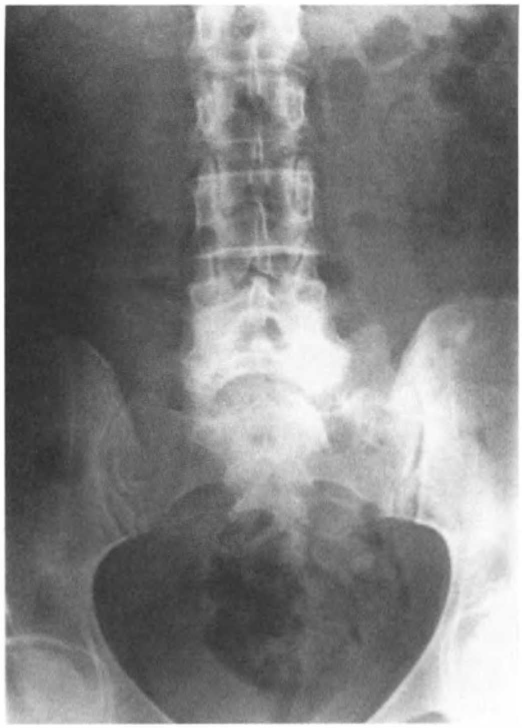


Figure 6.23. Anteroposterior lumbar spine and pelvic radiograph showing the right sacralized transverse process of L5 and sclerotic change of the right sacroiliac joint.

Figure 6.24. Lateral plain x-ray film shows the retrolisthesis subluxation of L4 on L5, which exceeds 3 mm and the degenerative L4–L5 disc (*see arrow*) and rudimentary L5–S1 disc space at the level of transitional vertebra.

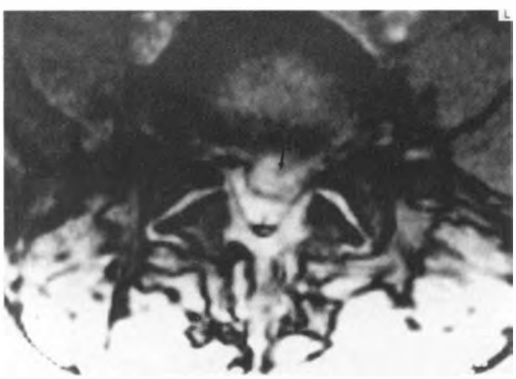


Figure 6.22. Axial section at the L4–L5
tral disc herniation contacting the thecal sac and occupying the left lateral recess. This is the presurgical magnetic resonance image.

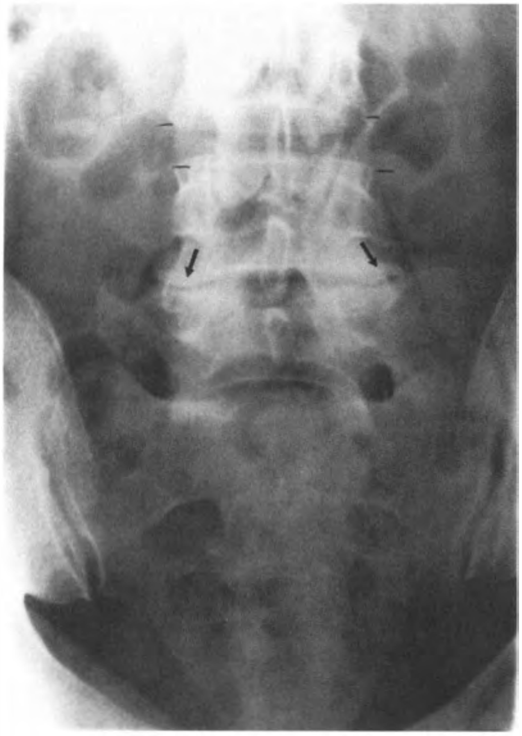


Figure 6.25. Tilt view of L5–S1 shows the spatulated L5 right transverse process fused to the sacrum.

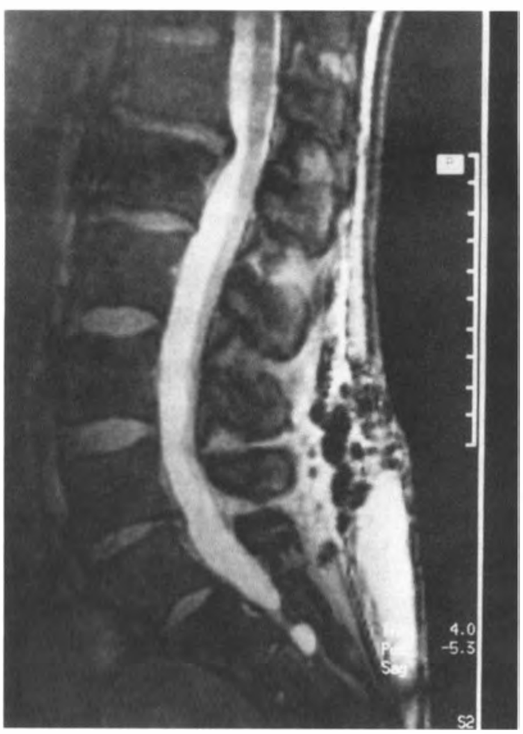


Figure 6.26. Repeat magnetic resonance image 1 year postsurgery shows reduction of the L4–L5 disc protrusion shown in Figure 6.21 and 6.22. Note the remaining T12–L1 disc degeneration and herniation.

Figure 6.27. The L4–L5 disc herniation is reduced compared with Figure 6.21 in this enhanced magnetic resonance imaging study. Note the retrolisthesis of L4 on L5 and the hyperintense area behind the L5 vertebral body, representing possible venoadipose tissue or fat placed in the area at the time of surgery to prevent scar tissue.

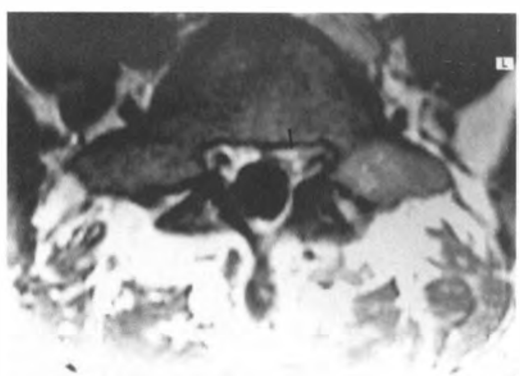


Figure 6.28. Note the hyperintense area in the left central vertebral canal at the site of previous surgery (*arrow*). This represents probable scar tissue in this gadolinium-enhanced magnetic resonance image.

Repeat magnetic resonance imaging (MRI) was performed 1 year postsurgery because of continued left leg pain of the same neural distribution. Figure 6.26 shows that the large L4–L5 disc herniation has been markedly reduced postsurgically. Figures 6.27 and 6.28 are postgadolinium-enhanced sagittal and axial T1-weighted MRI studies showing enhancement of scar tissue within the left central vertebral canal at the site of previous surgical disc removal. The scar is seen to contact the left L5 nerve root and thecal sac.

Treatment recommended was intramuscular injection of anesthetic followed by distraction manipulation and back bracing to support the unstable L4 vertbral segment. Stabilization home exercises of hamstring stretching, abdominal tightening, extensor muscle strengthening of the lumbar spine, abductor and adductor muscle exercises, wobble board, low back wellness training, Nautilus extension exercise on achieving 50% pain relief, and wearing a back support for 2 months was suggested. If 50% relief was not attained within 2 months of such care, surgical fusion would be considered, but it was not strongly recommended because of thecal sac scar tissue and nerve root chemical irritation and the decreased chance that surgery would benefit the patient greatly because of possible increased scar formation. The patient has not yet consented to any form of care and is depressed over the problem. Counseling is recommended.

Case 6

A 33-year-old man was seen for low back pain subsequent to playing softball. He later bent forward, coughed, and felt severe sharp pain in the lumbosacral spine. He took muscle relaxants but continued to have pain and stiffness.

Imaging studies are shown in Figures 6.29 through 6.35. These were taken 2 years previously to the current injury, when this man had left fifth lumbar nerve root dysesthesia of the lower extremity

Figure 6.30. The L5–S1 disc is rudimentary, as is typical of the transitional segment (*arrow*).

Figure 6.29. The spatulated transverse process (*arrow*) forms a pseudoarthrosis with the sacrum.

Figure 6.31. Oblique view shows the rudimentary facet joint formation (*arrow*) compared with the normal levels above.

Figure 6.32. Lateral projection of the myelographic examination shows the filling defect (*arrow*) behind the L4–L5 disc space due to discal protrusion compressing the dye-filled subarachnoid space.

Figure 6.34. Oblique myelographic study shows the L4–L5 discal defect (*arrow*).

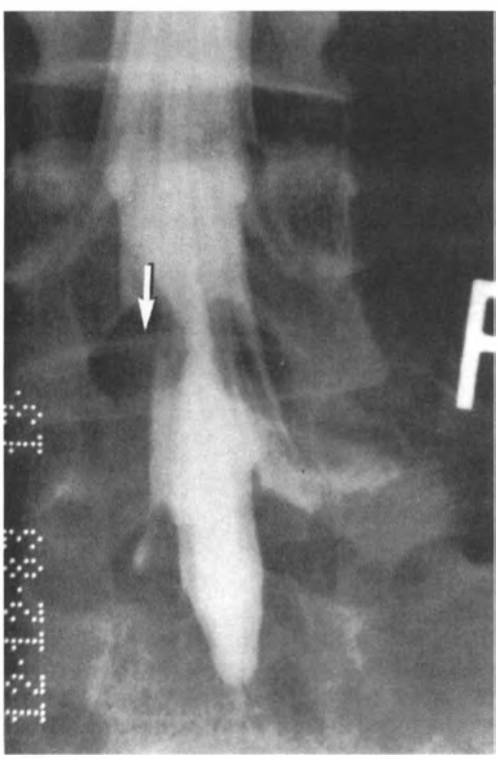


Figure 6.33. Posterior-anterior myelographic study shows the filling defect at L4–L5 (*arrow*).

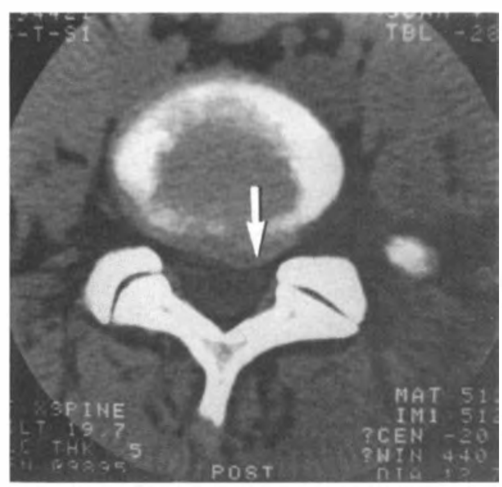


Figure 6.35. The computed tomography scan shows the large, left central discal protrusion that stenoses the lateral recess and enters the intervertebral canal (*arrow*).

and had undergone surgery to correct it. Figure 6.29 reveals the spatulated transverse process of L5 as it forms a pseudoarticulation with the sacrum. Figure 6.30 demonstrates the rudimentary L5-S1 disc that accompanies the transitional segment. Figure 6.31, an oblique view, further illustrates the underdeveloped facet joints at the transitional segment. Figure 6.32 reveals the discal protruding defect into the anterior dye-filled subarachnoid space. Figure 6.33, the posteroanterior study, shows the filling defect due to the L4–L5 protrusion. Figure 6.34, oblique myelographic study, also shows the defect. The computed tomography (CT) scan in Figure 6.35 shows a classic asymmetric left posterior protrusion of the left L4--L5 disc into the lateral recess and intervertebral canal.

This is an excellent representation of Bertolotti's syndrome with which to end this chapter. At the time we saw the patient, he had no leg pain. This patient did heavy manual labor entailing repetitive bending and lifting. He was placed on a strong regimen of abdominal, low back, and gluteus maximus muscle strengthening exercises. His hamstring muscles were especially contracted, and proprioceptive neuromuscular facilitation technique was used in lengthening them.

Flexion-distraction manipulation at the L4–L5 level was administered, along with range-of-motion palpation, and motion was restored to the entire lumbar spine with lateral flexion, circumduction, and progressive rotation into the upper lumbar levels. This patient made good progress in 1 month of care, at the end of which he stated he felt better than at any time since his surgery 2 years previously.

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Fibromyalgia

Lee J. Hazen, DC

chapter 7

The human body represents the actions of three laws, spiritual, mechanical and chemical, united as one triune. As long as there is perfect union of these three there is health."

—DD Palmer (AD 1910)

Fibromyalgia is defined as "nonarticular rheumatism with widespread and chronic musculoskeletal aching or stiffness associated with soft tissue tenderness at multiple, characteristic sites in the absence of an underlying cause" (1) (Fig. 7.1). Fibromyalgia syndrome (FMS) is also commonly associated with constitutional symptoms that include fatigue and morning stiffness, which may suggest a psychoneurophysiologic mechanism of dysfunction. Fibromyalgia can be further classified as idiopathic, post-traumatic, primary (without contributing disease), or secondary (as a result of a primary disease).

HISTORY

Fibromyalgia syndrome has historically been misnamed and misunderstood. Although FMS has been recognized as a specific clinical entity since the 1800s, it is likely to have existed in the distant past as one of the many musculoskeletal disorders that afflict mankind. Descriptions of the constellation of symptoms associated with FMS appear in the medical literature as far back as the writings of Hippocrates (460–377 BC) (2).

Since 1904 when Gowers coined this disorder "fibrositis" (3), it has had various sobriquets, including "fibromyositis," "myofascial pain syndrome," "psychogenic rheumatism," "generalized tension myalgia," "generalized nonarticular rheumatism," and "generalized soft tissue rheumatism" (4, 5). The term "fibromyalgia," first suggested by Hench (6) in 1976, has become the nomenclature of choice as it describes the essence of this painful syndrome (*fibro* = fiber, *myo* = muscle, *algos* = pain, *ia* = condition).

Fortunately, in 1990 the American College of Rheumatology endorsed the term "fibromyalgia" and dropped the distinction between primary fibromyalgia occurring in the absence of another condition and secondary (concomitant) fibromyalgia, which occurs with another disorder (1).

INCIDENCE

Fibromyalgia syndrome is estimated to affect approximately 3 to 6 million individuals, and it represents 20% of rheumatology and 5% of family practice office visits. It is estimated to be present in 5% of the general population (7). It is the third most prevalent rheumatologic disorder (after osteoarthritis and rheumatoid arthritis) (8).

Approximately 90% of FMS patients are women (usually white) between ages 40 and 60 years (mean age 49) (9–11). Studies show 28% of fibromyalgia patients indicated they were age 9 to 15 at onset (12), and estimates are 8 to 28% of school children may have FMS (13, 14). FMS may also affect the elderly (15, 16). Of the 10 to 20% of cases that affect males, the presentation is no different than that for women (17). An apparent familial tendency is seen, with 12% reporting symptomatic children and 25% reporting symptomatic parents. (18)

DIAGNOSIS

The current American College of Rheumatology classification criteria for the diagnosis of FMS are listed in Table 7.1.

The key diagnostic feature is tenderness found at specified sites (Fig. 7.1), but diagnosis must also include the following findings:

- Diffuse musculoskeletal pain for at least 3 months
- Stiffness that is worse in the morning
- Tenderness to digital palpation of at least 11 of 18 specific points

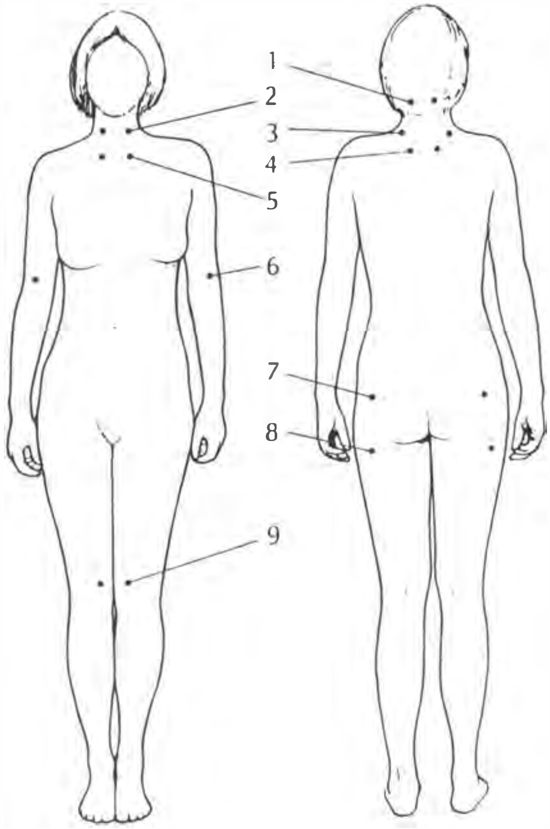

Figure 7.1. Tender points in patients with fibromyalgia.

Using these criteria, FMS can be diagnosed with a sensitivity of 88.4% and a specificity of 81.1% (1). In the American College of Rheumatology study, 56% of patients had all three symptoms, and 81% had two of the three.

The central feature in the diagnosis of fibromyalgia is generalized body pain—pain above and below the waist and on the right and left sides of the body. Although great variation is seen among individuals, an underlying uniformity is found in their pain pattern (19–22). Although fibromyalgia may be diagnosed by wide-spread pain and tenderness, the syndrome has a number of other characteristic symptoms (Table 7.2).

The key diagnostic feature of fibromyalgia is identification of tender points at specific anatomic locations (shown in Figure 7.1). These tender points can be assessed by palpation or spring gauge algometry (Pressure Threshold meter from Pain Diagnostics and Thermography, New York, NY). Using the algometer, 4 kg/cm^2 pressure is applied to each site; or manually until whitening of the examiner's fingernails (equivalent to 4 kg/cm^2) on the examined site. A site is considered tender when a pressure of less than 4 kg/cm^2 induces an uncomfortable sensation, with the patient responding to the examiner that pain was experienced (24). Pressure to the forehead is usually a good area from which to establish a control site as it is generally nontender. The existence of exaggerated tenderness at anatomically reproducible locations is essential to an accurate diagnosis. Some sites may be somewhat tender in normal individuals, so their location should be verified by the examiner. These tender points may be largely unknown to the patient, and often they are not even central to the main areas of complaint. These tender point sites are typically bilateral and fairly symmetrically involved but asymmetry is not uncommon (20).

Also, the symptoms of fibromyalgia can be aggravated or relieved by many factors (Table 7.3). Chronic muscle pain and exhaustion, with multiple somatic complaints, has often led to a diagnosis of hypochondriasis or hysteria in patients suffering from fibromyalgia. The uniform constellation of symptoms including tension headache, muscle aches, generalized stiffness, fatigue, and a high incidence of irritable bowel syndrome and sleep disorders in addition to tender points in characteristic locations makes fibromyalgia a readily definable syndrome within the spectrum of muscle pain syndromes.

Table 7.1

American College of Rheumatology Diagnostic Criteria for Fibromyalgia

History of Widespread Pain

Pain is considered widespread when all of the following are present:

- Pain in the left side of the body
- Pain in the right side of the body
- Pain above the waist
- Pain below the waist

In addition, axial skeleton pain (cervical spine, anterior chest, thoracic spine, or low back) must be present. Shoulder and buttock pain is considered as pain for each involved side. "Low back" pain is considered lower segment pain.

Pain on Digital Palpation in 11 of the 18 following sites of tender points

1. Occiput: bilateral, at the suboccipital muscle insertions.
2. Low cervical: bilateral, at the anterior aspects of the intertransverse spaces at C5–C7.
3. Supraspinatus: bilateral, at origins, above the scapular spine near the medial border.
4. Trapezius: bilateral, at the midpoint of the upper border.
5. Second rib: bilateral, at the second costochondral junctions, just lateral to the junctions on upper surfaces.
6. Lateral epicondyle: bilateral, 2 cm distal to the epicondyles.
7. Gluteal: bilateral, in upper outer quadrants of buttocks in anterior fold of muscle.
8. Greater trochanter: bilateral, posterior to the trochanteric prominence.
9. Knee: bilateral, at the medial fat pad proximal to the joint line.

Modified from Wolfe F, Smythe HA, Yunus MB, et al. The American College of Rheumatology 1990 Criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis Rheum 1990;33:160–172.

Table 7.2

Characteristic Symptoms of Fibromyalgia and Prevalence

Symptom	Prevalence (%)
Pain symptoms	
Widespread pain	98
Neck	85
Low back	79
Posterior thorax	72
15 or more painful sites	56
Dysmenorrhea	41
Headache	53
Other symptoms	
Fatigue	81
Morning stiffness > 15 min	77
Sleep disturbance	75
Paresthesias	63
Urinary urgency	26
Raynaud's phenomenon	17
Anxiety	48
Dry mouth	36
Prior depression	31
Irritable bowel syndrome	30

Modified from Wolfe F, Smythe HA, Yunus MB, et al. The American College of Rheumatology 1990 Criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis Rheum 1990;33:160–172.

Table 7.3

Factors Affecting Symptoms of Fibromyalgia

Aggravating Factors	Relieving Factors
Excessive physical activity	Restful sleep
Physical inactivity	Moderate activity
Anxiety or stress	Warm or dry weather
Cold or humid weather	Hot showers or baths
Nonrestorative sleep	
Physical or mental fatigue	

Modified from Wolfe F, Smythe HA, Yunus MB, et al. The American College of Rheumatology 1990 Criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis Rheum 1990;33:160–172; and Hench PK. Evaluation and differential diagnosis of fibromyalgia. Rheum Dis Clin North Am 1989;15(1):19–29.

DIFFERENTIAL DIAGNOSIS

Because of the nonspecific symptoms of pain, fatigue, and sleep disturbance associated with fibromyalgia, a plethora of musculoskeletal, systemic, and psychiatric diagnoses should be entertained. Multiple lists of potential differential diagnostic possibilities, which are often voluminous, have been published. However, the clinical use of such lists is limited, and a more practical differential list is suggested (Table 7.4).

Fibromyalgia syndrome has many symptoms in common with other well-recognized functional disorders (25–27). It may be helpful for the clinician to keep three broad categories in mind during the examination. First, rule out major medical disorders; thereafter, distinguish between primary fibromyalgia or fibromyalgia as a comorbid psychiatric disorder (the most common comorbid psychiatric disorder is depression), or finally a primary psychiatric disorder with symptoms that mimic fibromyalgia. This is best accomplished with a referral to a mental health specialist.

Although fibromyalgia occurs in association with other rheumatic disorders that must be included in the differential diagnosis, each disorder must be managed separately. It should be noted, however, that a diagnosis of fibromyalgia alone remains valid, regardless of any other diagnoses. Once other disorders have been discovered or ruled out, a treatment program can proceed.

Although musculoskeletal symptoms are universal, the wide range of symptoms reported by patients with fibromyalgia indicates that musculoskeletal pain is only one of a large number of diverse physical symptoms.

The overlap of symptoms of fibromyalgia with other conditions (fibromyalgia tends to be a diagnosis of exclusion) often lead to both extensive investigative costs and frustration for the patient. A careful and thorough history and physical examination are essential in helping direct the physician to the possibility of other underlying disease processes. Often, diagnostic uncertainty may cause "doctor shopping," useless diagnostic and invasive tests, and surgery, all of which accentuate the patient's worry and perpetuate the "sickness behavior" (12).

A through history, examination, and radiographs with a laboratory work-up to include a complete blood count (CBC), sedimentation (SED) rate, measurements of muscle enzymes and thyroid-stimulating hormone (TSH), rheumatoid factor, and antinuclear antibody determinations, should be sufficient both to rule out sinister pathology presenting as fibromyalgia and to secure the diagnosis of primary fibromyalgia (25).

Table 7.4

Practical Differential Diagnosis of Fibromyalgia

Myofascial pain syndrome
Chronic fatigue syndrome
Depression/anxiety
Multiple bursitis/tendinitis sites
Endocrine myopathies
Occult malignancy
Connective tissue diseases (rheumatoid arthritis, systemic lupus erythematosus, polymyalgia rheumatica, giant cell arteritis, polymyositis)

Modified from Hench PK. Evaluation and differential diagnosis of fibromyalgia. Rheum Dis Clin North Am 1989;15(1):19–29.

Many clinical and pathophysiologic similarities are found among fibromyalgia, myofascial pain syndrome (MPS), and chronic fatigue syndrome (CFS), including muscle changes, sleep disturbances, and depression. Yet, although these entities overlap, distinguishing features of each should be briefly reviewed

Myofascial Pain Syndrome

First, let us review myofascial pain syndrome (Table 7.5), a syndrome characterized by painful, tender areas in muscles on palpation in association with muscle twitch and a zone of referred pain (28). MPS is a nonchronic, localized disorder without systemic manifestations (25). Found in both sexes, it can occur at any age, with the peak prevalence between age 30 and 60 (23). Although MPS has been described as occurring in most muscles of the body, it most commonly affects the axial muscles involved in maintaining posture (29). In contrast to fibromyalgia, MPS presents with a more identifiable precipitating event, is more localized (particularly to a single body region), and features more prevalent trigger points with characteristic radiating patterns. This differentiates MPS from fibromyalgia, which involves multiple muscle groups and nonreferring tender points. Some helpful differential features are listed in Table 7.5. Differentiation becomes clouded when patients exhibit characteristics of both myofascial pain and fibromyalgia. Myofascial pain can lead to fibromyalgia, with unresolved localized muscle pain ultimately involving multiple muscle groups (30). Indeed, it has been suggested that muscle pain syndromes as a whole are neighboring stages of a biologic continuum of a single disorder.

Chronic Fatigue Syndrome

A second important disorder in the differential diagnosis of fibromyalgia is chronic fatigue syndrome. Criteria for the diagnosis of CFS remain controversial. Essential elements for the diagnosis of chronic fatigue syndrome include two major criteria:

1. Chronic, persistent, and disabling fatigue.
2. Exclusion of other conditions that may produce similar symptoms.

Minor criteria comprise a constellation of symptoms (26). The number and extent of dolorimetry scores of tender points is the most significant feature distinguishing fibromyalgia from CFS.

Fatigue, the hallmark of CFS, has an abrupt onset, occurring within hours or days. Chronic fatigue syndrome frequently follows a viruslike illness (31). Other symptoms observed in these patients include low-grade fever, pharyngitis, myalgia, arthralgia, sleep disturbance, visual problems, headache, malaise, and varying degrees of anxiety and depression. The illness can last from months to years but is not progressive; symptoms are most severe during the first year.

Diagnostic criteria proposed for CFS and fibromyalgia overlap in many areas. In one study, a comparison of 27 patients with CFS and 20 patients with fibromyalgia revealed that patients with CFS who had pain at the time of the study had tender point scores identical to those of patients with fibromyalgia (7). The authors also found that severe fatigue and/or sleep disturbance were present in more than 90% of patients in both groups. In the patients with fibromyalgia, 54% reported recurrent pharyngitis and 52% thought their symptoms began with a flulike illness, characteristics that are more typical of CFS than fibromyalgia. One explanation for these findings may be that patients with fibromyalgia are seldom questioned about fever, swollen lymph nodes, and sore throat, whereas patients with CFS are seldom examined for the presence of tender points (27–33).

Table 7.5

Features of Fibromyalgia and Myofascial Pain Syndrome

Feature	Myofascial Pain Syndrome	Fibromyalgia
Sex	Men 2:1	Women 10:1
Tender point pain	Referred trigger point	Local
Tender point distributions	Regional (usually axial)	Widespread
Tender point locations	Muscle belly	Muscle-tendon junctions
Stiffness	Regional	Widespread
Fatigue	Usually absent	Debilitating

Modified from Bennett RM. Confounding features of the fibromyalgia syndrome: Current perspective of differential diagnosis. Rheumatology 1989;16(Suppl 19):58–61.

CAUSE AND PATHOPHYSIOLOGY

The cause of fibromyalgia is unknown, although some authors note that patients often have an antecedent viral infection or traumatic event (32, 34–36). No evidence of an underlying cause or pathophysiologic basis for fibromyalgia currently exists, although a myriad of mechanisms have been proposed.

Included in the list of proposed mechanisms are lack of physical fitness (37, 38), sleep deprivation (39, 40), chronic muscle spasm with ischemia (41–45), disturbances in muscle microcirculation (41), adenosine monophosphate and creatine level imbalances (18), neurohormonal imbalances (46), as well as other chemical imbalances that include tryptophane-serotonin levels (47), levels of corticotropin (ACTH) (48), prostaglandin and catecholamine changes (49), or somatomedin C levels (50, 51). Stress and emotional disorders are also implicated, and they are almost invariably associated with the clinical picture (52–54). Also postulated as causes are viral infections (55–59), nutritional deficiencies (60–62), as well as the hypothesis of hyperpermeability of the intestinal mucosa leading to the systemic cascade of antigenic invasion

(63–68). Unfortunately, little scientific evidence exists to support any of these hypotheses.

MANAGEMENT AND TREATMENT

Treatment of fibromyalgia can be difficult for both the physician and the patient. A multifaceted treatment plan has shown the most promise for these patients. Treatments must be directed to stop the trend toward functional disability and chronic disease (25). This trend is reflected in a gradual increase in pain-related behaviors that correlate with an increase in physical disabilities and pain scores.

The key to management of fibromyalgia is a firm diagnosis, followed by assurance that the condition is benign, noncrippling, and may eventually remit (69). Patients must be encouraged to help themselves through positive environmental changes, and physicians must accept that they can only do so much to ameliorate these patients' conditions (70).

Because treatment protocol is so diverse, the chiropractor should focus on a team approach. This includes interdisciplinary relationships with other qualified health care practitioners (e.g., medical doctors, physical therapists, massage therapists, and psychotherapists).

Using a team approach for more than 3 years, Nies (71) found that 70% of patients will have significant improvement in pain symptoms and functional capacity if the syndrome is identified early and the patient is well motivated. Treatment must be directed toward decreasing functional disability and chronicity and gradually increasing functional capability. This will tend to reduce the pain-related behaviors and dependence mentality that accompany fibromyalgia.

Patient education is another key component in achieving satisfactory results with treatment. Patient education is important in assuring patients that they have a common, non–life-threatening condition and that little will be gained by seeing multiple physicians and undergoing repeated tests. The more that patients understand their condition, the better they will be able to help themselves. Individualized programs can be devised in which the patient and family members assume an active role in treatment. As always, the doctor is responsible for the patient's health care, and the patient is responsible for personal health.

Management programs for fibromyalgia sufferers must focus both on the modalities that reduce pain and on instruction in posture, ergonomic training for activities of daily living and the work place, chiropractic manipulation to restore proper biomechanics, stretching exercises and gradual intervention with aerobic exercise, initial pharmaceutic intervention if warranted for pain management and restorative sleep, as well as sleep hygiene instruction. The chiropractor treating fibromyalgia patients should ask the patient to modify activities of daily living, and help them recognize that temporary setbacks inevitably occur and are part of the course of this frustrating condition. Fortunately, chiropractic manipulation and palliative modalities such as heat, massage, trigger point therapy, and stretching exercises will provide temporary relief.

EXERCISE THERAPY

Evidence indicates that aerobic exercise has a protective role as well as a treatment role in preventing FMS. Therefore, *aerobic exercise is the cornerstone of therapy* (72, 73).

Virtually all patients with fibromyalgia experience some degree of pain following initial exercise and as a result are reluctant to continue an exercise program, thus leading to further deconditioning (77). Furthermore, unconditioned muscles are subject to postexercise muscle soreness, which includes muscle pain, stiffness, tenderness, and reduced strength 24 to 48 hours after exercise (74). This is particularly true of fibromyalgia patients as it is likely they are considerably deconditioned. Immediate postexercise effects may also reinforce the patient's belief that there is no way to control the disease, which in itself may perpetuate noncompliance with treatment regimens and encourage "doctor shopping." Also, patients may tend to be resistant to change because it implies change in lifestyle and activities of daily living, with some increase in discomfort and long-term effort. The chiropractor must inform the patient of the importance and difficulty of proceeding with an exercise regimen, encouraging gradual development of conditioning. Poorly conditioned muscles cannot be restored as quickly as conditioned muscles due because they have less glycogen storage and low adenosine triphosphate (ATP).

Research has indicated that more than 80% of patients with fibromyalgia are not physically fit (74). Study results have suggested a "detraining phenomenon," which can lead to habitual inactivity with a resultant common symptom complex that includes palpitations, tachycardia, dizziness, headache, paresthesias, breathlessness, chest pain, abdominal pain, dysphagia, muscle pain, tremor, excessive sweating, fatigue, weakness, tension, and anxiety (37, 75).

One study included 42 patients with fibromyalgia who were assigned to a 20-week program consisting of either cardiovascular fitness training or simple flexibility exercises (76). Blind assessments were made, and patients who received cardiovascular fitness training showed significantly improved cardiovascular fitness scores compared with those who received flexibility training. Analysis showed clinical and statistically significant improvement in pain threshold scores among patients in the cardiovascular training group. These patients also improved significantly in both patient and physician global assessment scores.

Another study evaluated patients with fibromyalgia for hypermobility of joints (74). The 210 patients who exercised during the study showed improvement, but patients with fibromyalgia who had articular hypermobility were more likely to exercise with greater improvement in symptoms.

Although it is understandable that fibromyalgia patients do not want to exercise because of fatigue and pain, if they do so, the prognosis is greatly improved.

CHIROPRACTIC CARE

It should be noted that gentle chiropractic manipulation (particularly distraction manipulation) is ideally suited to this pa-

tient population as it provides a stretching component to the soft tissues while relieving the intradiscal and facet joint pressures, which in turn reduces the necessary forces involved in providing a spinal adjustment. This form of chiropractic adjusting is better tolerated by the fibromyalgia patient. Manipulation of hypomobile segments is essential; however, the doctor is strictly cautioned to avoid inducing hypermobility to the intervertebral motion segments (3-joint complex) as this will aggravate the overall condition.

Chiropractic patients typically describe receiving a few hours of temporary relief after manipulation (1 to 2 hours), with a return of symptoms thereafter. This pattern, seen in the pain-spasm-pain cycle, often leads to increased dependance on the chiropractor, which is to be avoided.

Wolfe

had received chiropractic care reported moderate to great improvement. Chiropractic scored among the most effective

providing more improvement. This is noteworthy, as it shows chiropractic care to be more effective

PHARMACOLOGIC INTERVENTION

No single treatment method has been shown to be completely effective, and combinations of therapies are often used to relieve the symptoms of fibromyalgia. A number of pharmacologic agents have been used to treat FMS with mixed results.

Low-dose tricyclic antidepressants (Elavil, Endep, Flexeril) are widely used in the treatment of intractable pain disorders. These agents offer various benefits, including antidepressant effects, anti-inflammatory properties, effects on central skeletal muscle relaxation, and enhancement of pain-inhibiting factors through both serotonergic and noradrenergic pathways (79). The major pharmacologic action of tricyclic antidepressants appears to be facilitation of central monoamine transmission by inhibiting serotonin and norepinephrine uptake at the synapse, thereby potentiating neuronal activity (10, 80–85).

Low-dose tricyclic antidepressants have been effectively used at night to modulate sleep disturbance. These drugs apparently improve stage four sleep and probably increase the level of brain serotonin and other neurotransmitters. In addition to improving sleep, adequate analgesia must be provided.

Nonsteroidal anti-inflammatory drugs (NSAIDs) alone are often not sufficient to relieve the aching and discomfort. However, NSAIDs combined with amitriptyline has a synergistic effect, and has proved more effective (84). Both amitriptyline (Elavil) and cyclobenzaprine (Flexeril) have been used (79–86); however, it appears that in long-term therapy, some of the benefits of tricyclics may be lost, and the side effects are often daunting.

Other authors doubt the utility of pharmaceuticals in the treatment of fibromyalgia because of an overall poor performance and side effects showing no improvement in 56.6% of patients using amitriptyline and no improvement in 46.3% of patients using cyclobenzaprine (12).

Opioid analgesics are not indicated in the management of fibromyalgia. Nonopioid analgesics (acetaminophen) can be utilized as needed.

Medication should play only a minor role in the treatment of fibromyalgia because of the risk of dependency and the long-term ineffectiveness of these drugs (78).

These facts underscore the importance of combining conservative therapies

PSYCHOLOGICAL TREATMENT

Cognitive therapy can be effective in relieving the patient's depression, anxiety, anger, and so forth, as well as perhaps dealing more directly with stress reduction (71, 87) and ferreting out possible causative factors hidden in the patient's psyche. Changing the fibromyalgia patients' perspective of themselves and others can have a dramatic effect on their well being (88). Also, memory, comprehension, and concentration difficulties may be experienced by the patient (89). It should also be mentioned that post-traumatic fibromyalgia patients report significantly higher degrees of pain, disability, life interference, affective distress, and a lower level of activity than do idiopathic fibromyalgia patients (90).

MISCELLANEOUS THERAPIES

Several other therapies have recently shown promise in the treatment of fibromyalgia syndrome. Here are a few noteworthy examples.

Goldenberg et al. found 96% of fibromyalgia patients in a relaxation/stress reduction course felt the program valuable and 75% showed at least moderate clinical improvement (91).

Meditation-based stress reduction programs show 51% of fibromyalgia patients have moderate to marked improvement (92).

Electromyographic (EMG) biofeedback techniques show improvement in pain scores, morning stiffness, and number of tender points even at 6-month follow-up (93).

Electroacupuncture has also been shown to be effective in relieving the symptoms of fibromyalgia (94).

The use of malic acid with magnesium was beneficial in the treatment of fibromyalgia, as tested in a double-blinded study (95).

Vitamin E has long been suggested for rheumatic disorders as well (96).

Homeopathic tincture of Rhus toxicodendron (poison oak) used in a double-blind, placebo-controlled, cross-over designed trial showed improvement in pain and sleep patterns (97–99).

Other therapies include tender point injection therapy, counterirritant therapy, and the use of transcutaneous electrical stimulation (TENS), all of which have been disappointing in the treatment of FMS (100, 101).

The greater the level of active patient involvement the more likely the patient will experience a favorable clinical outcome. However, as with any group of chronic pain patients, patient compliance is a significant problem. It may be difficult to wean the patient from drugs, physical medicine modalities, or care-receiver/codependent behaviors. In a 1994 study, it was shown that 13% of patients refused to attend treatment ses-

sions and others were discharged because of repeated disruptive behavior (101).

The best recipe for relief of fibromyalgia would appear to be is a combination of patient education, rest, counseling, moderate aerobic exercise, stretching, nutritional intervention, and chiropractic care.

PROGNOSIS

The prognosis for patients with fibromyalgia remains unclear. We currently do not understand why certain patient's with fibromyalgia seem to recover or adapt and others do not. Whether these differences are physiologic or psychological in nature is unknown.

To date several studies on the prognosis of this curious syndrome are conflicting. The longest study conducted on fibromyalgia found that many of the symptoms of fibromyalgia persisted at 10 years of follow-up. But most patients in the study reported that their overall condition had improved, and more than half characterized themselves as being "well" or "very well" (102).

Another group of researchers in Australia reported that many patients with fibromyalgia actually recover; at a 2-year follow-up of 44 fibromyalgia patients, 47% no longer satisfied the criteria for fibromyalgia. This study also showed that simple exercises were more effective than drugs or physical therapies (103).

However, a 1994 study followed 176 post-traumatic fibromyalgia patients for 10 years. A dramatic reduction was found in the use of all forms of physical therapy, yet 85% of the patients continued to have significant symptoms and clinical evidence of fibromyalgia (104).

A 1993 study showed a bleak outlook for patients with fibromyalgia. Ledingham et al. followed 72 patients for 4 years. At follow-up 97% still had fibromyalgia symptoms and 60% felt worse (105).

Finally, another study from Sweden tracked 56 patients for 5 years and showed that only 20% of patients had any improvement with one half of the patients reporting a worsening of pain, fatigue, and sleep disturbances (41).

SUMMARY

Fibromyalgia is a common clinical entity seen in chiropractic offices today. Its diagnosis has been facilitated by the development of clinically useful criteria. Until more is understood about this syndrome, the empiric approach to treatment shows the greatest chance of relief. This approach is multidisciplinary and includes the use of an empowering paradigm wherein the patient is taught to control his/her condition through proactive rehabilitation. The prognosis remains unclear, perhaps because of the lack of this interdisciplinary approach being taken by more researchers. However, what does remain clear is that fibromyalgia patients may improve with reassurance that the condition can be helped with their cooperation, minimal acute phase modalities, short-term medication, nutritional supplementation, and chiropractic adjustments coupled with a strong rehabilitation approach. Chiropractic physicians are uniquely suited to address the management and treatment of this difficult syndrome, and they may have the means to afford the fibromyalgia sufferer the greatest relief of any health care provider.

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Biomechanics Research on Flexion-Distraction Procedure

MR Gudavalli, PhD

chapter 8

I'm a great believer in luck, and I find the harder I work the more I have of it.

—Thomas Jefferson

Low back pain (LBP) is a common condition in the United States; in fact, it is the second most common symptom causing a patient to seek medical care. At any given time, 6.8% of the population suffers from back pain. The direct and indirect costs of medical care for LBP are more than $50 billion per year (1). Deyo and Tsui-Wu (2) report that one third of the US population seek chiropractic care as a first-line treatment for LBP. Objectives of treatments for LBP are to reduce suffering, hasten recovery, and minimize recurrence or development of chronic disability. A number of treatment alternatives are available.

Typically, the physician must determine from which tissue the pain is emanating and why. The poorest prognosis exists for the patient when LBP is caused by disc herniation and when neurologic deficits are present. In these more difficult patients, several scenarios are possible. First, the condition may heal on its own during a period from 6 weeks to 1 year. Second, the patient can be prescribed orthotic supports. Third, the patient may require surgery. Fourth, the patient may seek a form of chiropractic treatment in an attempt to obtain relief. This last option is selected by 31% of US patients with LBP (2).

Manipulative or manual procedures have been used to treat spine-related disorders since antiquity (3). Chiropractic physicians deliver approximately 94% of all manipulative treatment administered in the United States (4), and numerous studies have shown that some forms of manipulation are therapeutically effective (5, 6). Further, manipulative therapy by chiropractic physicians is cost-effective. Recent studies comparing costs show that manipulative therapy by chiropractic physicians costs one tenth that of medical care (6).

When a patient seeks chiropractic treatment for low back pain, a commonly used technique is flexion-distraction. The first use of flexion-distraction in treating patients with low back pain is attributed to Stoddard (7), an osteopathic physician. He (7) suggests that another osteopathic physician, McManis, initially developed the procedure, but no citation indicates that the development was ever documented in a published work. Stoddard (7) also reported that the procedure was completely safe for treating mechanical and disc lesions in the lumbar spine, but data to support this contention are completely lacking. Beginning in 1974 Cox, a chiropractic physician, undertook a variety of modifications to the procedure. The history of these modifications and recommendations for the use of the procedure are described (8). Although safety and efficacy are undocumented, a survey conducted by the National Board of Chiropractic Examiners (9) in 1993 indicated that 52.7% of the chiropractors surveyed routinely employ flexion-distraction in the management of LBP. The modality as modified by Cox is specifically designed to treat LBP disorders in which disc herniations of the lumbar spine are a prominent feature.

RESEARCH ON FLEXION-DISTRACTION PROCEDURE

The federal government has funded The National College of Chiropractic in collaboration with Loyola University Medical School to conduct research on this therapeutic procedure using the flexion-distraction instrument. The technique is based on the hypothesis that vertebral decompressive displacements occur during the flexion-distraction procedure, and the neural foraminal elements can be decompressed by providing increased foramina space in the lumbar spine. The funded research study addresses the following research questions:

1. Does the flexion-distraction procedure create vertebral motions and increase the intervertebral foramina (IVF) space in the lumbar spine in vitro?

2. How reproducible are the biomechanical effects of the flexion-distraction therapy (i.e., what is the intra- and interclinician reliability of the biomechanical parameters) while administering this procedure in vitro?
3. What are the loads on the internal tissues of the spinal segments L4–L5 and L5–S1, and are the tissues at risk of injury during the flexion-distraction procedure in vitro?
4. Is there significant trunk muscle activity that may reduce the effectiveness of the procedure by affecting the transmission of loads and motion to the spinal segment in vivo?

These questions will be addressed using the following specific aims during the proposed 3-year program:

1. By conducting in vitro experiments with 10 unembalmed whole cadavers, we propose to quantify the following parameters during the flexion-distraction therapeutic procedure: (a) the three-dimensional motions at L4–L5 and L5–S1 segments, (b) dimensional changes of the IVF (height, width, and area) at L4–L5 and L5–S1 segments, and (c) the loads applied to the subject.
2. When three different chiropractic physicians are administering the therapeutic procedure three times on each of the 10 unembalmed whole cadavers, we will quantitatively describe the intra- and interclinician reliability of the biomechanical parameters measured in the first specific aim.
3. By means of a computer model developed by the investigators, we propose to estimate the internal loads on the disc and the ligaments of the L4–L5 and L5–S1 spinal motion segments under the loads applied during the in vitro flexion-distraction therapeutic procedure. We will compare these loads with the failure loads of these tissues available in the literature to assess the risk of injury to these tissues.
4. By means of in vivo experiments on 60 LBP patients, we will quantitatively describe the following biomechanical parameters: (*a*) loads applied to the subject during the therapeutic procedure and (*b*) the electromyographic (EMG) activity of the right and left erector spinae, right and left abdominal, and right and left oblique superficial muscles surrounding the lumbar spine.

These studies to measure the changes in the lumbar spinal canal dimensions (e.g., posterior disc height, posterior disc bulge, and dimensional changes in the IVF), as well as the vertebral displacements, will provide quantitative scientific data on the mechanism of action, as well as determine the biomechanical limits of the flexion-distraction procedure.

This information is vital for the following reasons:

1. Biomechanical data will elucidate the mechanism by which this treatment is hypothesized to provide relief. This information, in turn, will assist clinicians in making a decision regarding the appropriateness of this treatment for particular patients. Knowledge regarding the mechanism of action of flexion-distraction is essential for the design of an efficacy trial. Without such data, setting the inclusion and exclusion criteria for a randomized clinical trial of flexion-distraction becomes a matter of "crystal ball gazing" rather than a rational design decision.
2. Biomechanical data are important to determine the reproducibility of this procedure. Once known parameters of loads and so forth are known, clinicians can be trained to deliver the procedure using the biomechanical objective criteria as credentialing goals.
3. Biomechanical data can provide objective information to define the limits of safety of the treatment procedure. For example, the loads applied during the flexion-distraction procedure will be greatly affected by the degenerative condition of the disc and ligaments. Thus, the biomechanical data will aid in modifying the procedure to suit the degenerative conditions of the spine.
4. Biomechanical data can be used to see if the trunk musculature EMG activity can significantly alter the loads transferred to the spine during the flexion-distraction procedure, thus altering the biomechanical effectiveness of this procedure. The EMG data may aid in modifying this procedure for the patients with significant muscle activity. This chapter present the current basic research findings thus far in progress on the flexion-distraction procedure. The research was funded by the Bureau of Health Professions (BHPr), Health Resources and Services Administration (HRSA), Public Health Service (PHS), and Department of Health and Human Services (DHHS) to conduct biomechanical research in understanding the mechanism of action of this procedure. The chapter reports on the studies conducted so far. These include (*a*) the radiographic measurement of the motion of the lumbar vertebrae of a cadaver under flexion-distraction treatment (*b*) mobility studies of the flexion-distraction table during treatment of low back pain (LBP) patients, (*c*) EMG activity determination of the superficial muscles surrounding the lumbar spine on healthy volunteer subjects, (*d*) estimation of the loads on the ligaments and disc of the lumbar spine, and (*e*) the intradiscal pressure changes during the flexion-distraction procedure.

Radiographic Studies

This study was undertaken to determine whether or not vertebral motions occur during the flexion-distraction therapy. An unembalmed cadaver was procured from Demonstrators Association, Chicago, Illinois, and stored in a freezer at −20°C until use. The cadaver was that of a 57-year-old white man weighing 159 pounds, who had died of brain hemorrhage. The table was positioned in radiographic equipment to obtain lateral radiographs. A Plexiglas frame, with an embedded 5 × 5 mm grid made of lead balls, was placed on the table in the same position as the center of the spine to determine the magnification associated with the radiographs. This radiograph of the lead ball grid was used to determine the magnification, and a magnification factor of 1.6 was calculated from the ratio of the distance

between adjacent lead balls on the radiograph to the actual distance between the lead balls.

Prior to experimentation, the cadaver was thawed at room temperature. The cadaver was positioned on the table in a prone position similar to that used during treatment, and radiographs were taken in two positions of the treatment procedure: neutral and extreme. The posterior and anterior points of the end plates of L4, L5, and S1 were used as landmarks to measure the relative displacements between L4 and L5 and L5 and S1. The results from the radiographs indicate a flexion angle of 6° between L5–S1 and 3.5° between L4–L5, and increases of 3 mm posterior disc height for L5-S1 and 1.87 mm for L4–L5. These results indicate that vertebral motions occur and widen the spaces available in the posterior region for neural elements. However, further studies are not proposed in this direction because the three-dimensional displacements cannot be obtained from the lateral radiographs alone and the physician(even with the protection of lead apron) is vulnerable to radiation exposure. The preliminary study was performed to provide a rationale for the proposed studies of research design and methods.

Mobility Study of the Flexion-Distraction Table

Figure 8.1 is a photograph of the flexion-distraction table with the physician treating a patient with LBP. This study was undertaken to determine the ranges of the flexion-distraction table movements in clinical situations and to assess whether these ranges change as a function of patient population. These values will be used for the range of treatment while conducting in vitro experiments on cadavers. A study on the flexibility of the table was conducted on 30 patients who had been treated by James M. Cox, DC for LBP using the flexion-distraction therapy. The patients ranged in age between 15 and 76 years. Their weights ranged from 52 to 154 kg (115 to 340 pounds). The flexion angles of the caudal portion of the table were recorded by a digital goniometer with a resolution of 0.1° mounted on the side of the moving portion of the table. Recorded angles were from the beginning position to the intermediate therapy position and from the beginning position to the full extreme position of the therapy. The average angles were 3.4° to the intermediate therapy position and 6.6° to the extreme position. The difference in the angle from the intermediate position to the extreme position had a mean of 3.2° The maximal angle to the extreme position was 11°. No statistically significant correlations or differences were found with respect to the categories of age, sex, weight, and height of the patients.

EMG Activity of Lumbar Muscles During the Flexion-Distraction Procedure

This study was performed to identify the role of the musculature during flexion-distraction therapy. Six superficial muscles surrounding the lumbar spine, namely, left and right erector spinae, right and left abdominal, and the right and left oblique muscles were studied with surface EMG. The diameter of the contact area of the surface electrodes was 2 cm and a bipolar spacing of 6 cm was used. EMG signals were monitored by

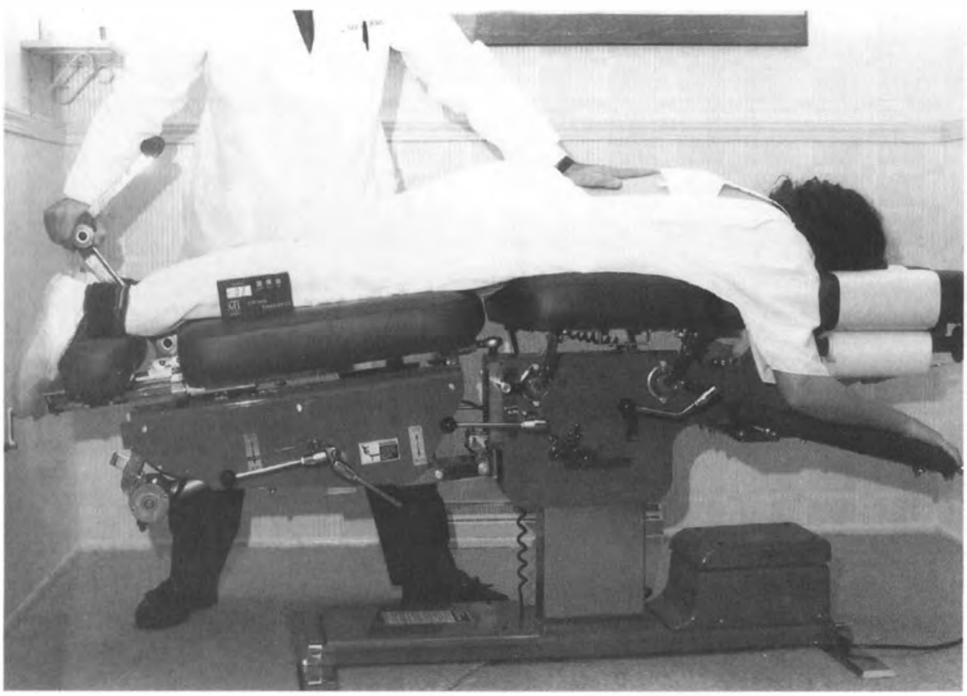

Figure 8.1. A photograph of the experiment showing the flexion-distraction table and an electronic goniometer.

means of a microcomputer, and the root mean square (RMS) values of the muscle activity were computed in micro volts. Figure 8.2 shows a subject mounted with surface electrodes and placed in prone position for treatment. To provide normalizing baseline data, the EMG activity was obtained when the subjects were exerting their maximal voluntary strengths. The subjects were two male and two female healthy volunteers (with no history of back pain within the past 1 year) with ages ranging from 25 to 55 years. The subjects were positioned in an Isostation B200 (Isotechnologies Inc., Hillsborough, NC), which allows the evaluation of low back strength. The subjects were asked to exert their maximal strength in flexion, extension, right and left lateral bending, and right and left rotation. EMG activity was recorded during the maximal voluntary exertion and with the maximal voluntary strengths (Fig. 8.3). The maximal voluntary strengths ranged from 30 to 66.7 N-M (22.2 to 49.2 ft-lb) in rotation, 69.8 to 114.3 N-M (51.5 to 84.3 ft-lb) in lateral bending, 64.2 to 109 N-M (47.4 to 80.5 ft-lb) in flexion and 96.2 to 131.6 N-M (71 to 97.1 ft-lb) in extension. The RMS values of the EMG activity under these maximal voluntary strength conditions varied from 39 to 247 μv for erector spinae, 73 to 142 μv for the abdominal muscles, and 61 to 208 μv for the oblique muscles.

The subjects were then placed on the flexion-distraction table and the EMG activities of these six muscles were recorded while the subject was both at rest with no treatment procedure and during the treatment procedure. The RMS values of the EMG activity indicate the activity of the muscles during treatment were one to five times the activity of the same muscles during rest. However, comparison of the EMG activity of the muscles while under treatment with the maximal EMG activity during voluntary contraction indicates the activity to be 2 to 12% of the maximal activity. Responses in patients with LBP may be higher or lower, suggesting that the activity of the muscles need to be monitored during flexion-distraction therapy. Consequently, in our proposed study we will monitor the patients. The option of using the needle electrodes was considered. However, this option was discarded because the same information can be obtained without the discomfort of the needles. Furthermore, it is possible that needle electrodes would interfere with therapy.

INTRADISCAL PRESSURE MEASUREMENTS

The flexion-distraction treatment is based on the hypothesis that the intradiscal pressure decreases during the procedure, which may provide an opportunity for the disc bulge to reduce. However, no data exist to support this hypothesis. This study measured the changes in the intradiscal pressures in the lumbar spine on unembalmed cadavers during the flexion-distraction procedure.

Materials and Methods

Two miniature pressure transducers (Model # SPR-524) were purchased from Millar Instruments, Houston, TX, for this study and calibrated with specially built devices that can be pressurized or create a vacuum. These devices are fitted with a calibrated reference pressure gauge (Model: ASHCROFT; range: 0 to 20,686 mm Hg; accuracy: 0.25%) or vacuum gauge (Model: DURO-UNITED; range: 0 to −762 mm Hg; accuracy: 2%). While monitoring the voltage from the pressure transducers, we varied the input pressures from −483 to 1062 mm Hg. Figure 8.4 shows the calibration curves for both the

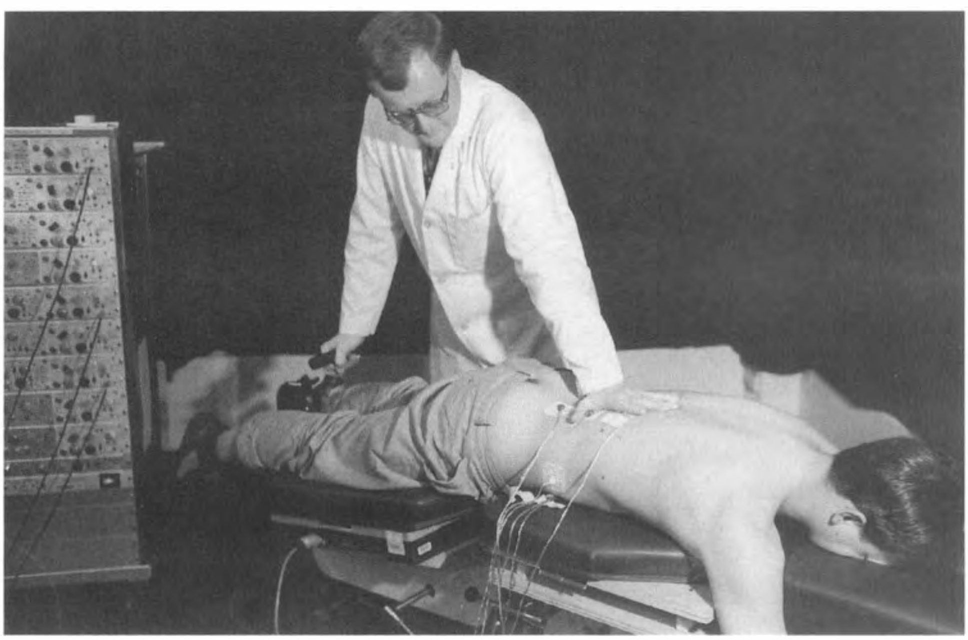

Figure 8.2. A subject on the flexion-distraction table with surface electrodes placed on the lumbar muscles.

Figure 8.3. The subject exerting maximal voluntary contraction in a B200 machine.

transducers used in this study. The calibration curves had straight line relationships with a linearity of 5% in the end range and a Pearson's correlation coefficient of 0.9997 for the entire range of measurement that is desired for this study.

We procured five unembalmed whole cadavers for this study (four male and one female; age range 43 to 75 years). The cadavers were frozen at $-20°$ C immediately after death and thawed at room temperature prior to experimentation. An anatomy consultant dissected some of the paraspinal musculature to permit accurate insertion of the needle and pressure transducer. We inserted a Touhy epidural needle with stylette (17 gauge) into the nucleus of the disc (either L2–L3, L3–L4, or L4–L5). (Figure 8.5 shows a pressure transducer and a needle used for the study.) We then removed the stylette and inserted the miniature pressure transducer so that the sensor was exposed to the nucleus. We connected the pressure transducer to a computer through a signal amplifier and analog-to-digital converter. Figure 8.6 shows the close-up view of the intradiscal pressure transducers mounted into the disc. We placed the cadavers in a prone position on the flexion-distraction table, similar to the positioning for a living patient. The treatment procedure consisted of five cycles of table motion in approximately 20 seconds. The discs were pressurized with water using a Cornwall continuous pipetting outfit (B-D # 3052) connected by flexible tubing to a second needle in the disc of interest. LUER-LOK stopcocks allowed air to be bled from the system before pressurizing.

An operator monitored the intradiscal pressures by means of the computer during the flexion-distraction procedure under two conditions: (*a*) the discs unpressurized and (*b*) the discs pressurized with water. The pressures were monitored during three separate trials with 30-minute intervals between each

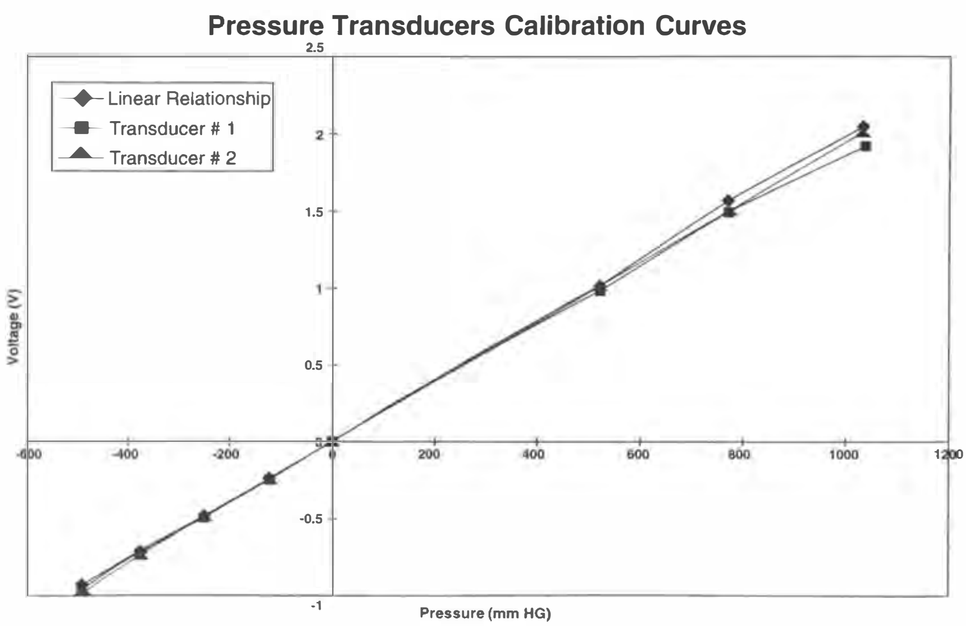

Figure 8.4. Graph showing the pressure transducers calibration.

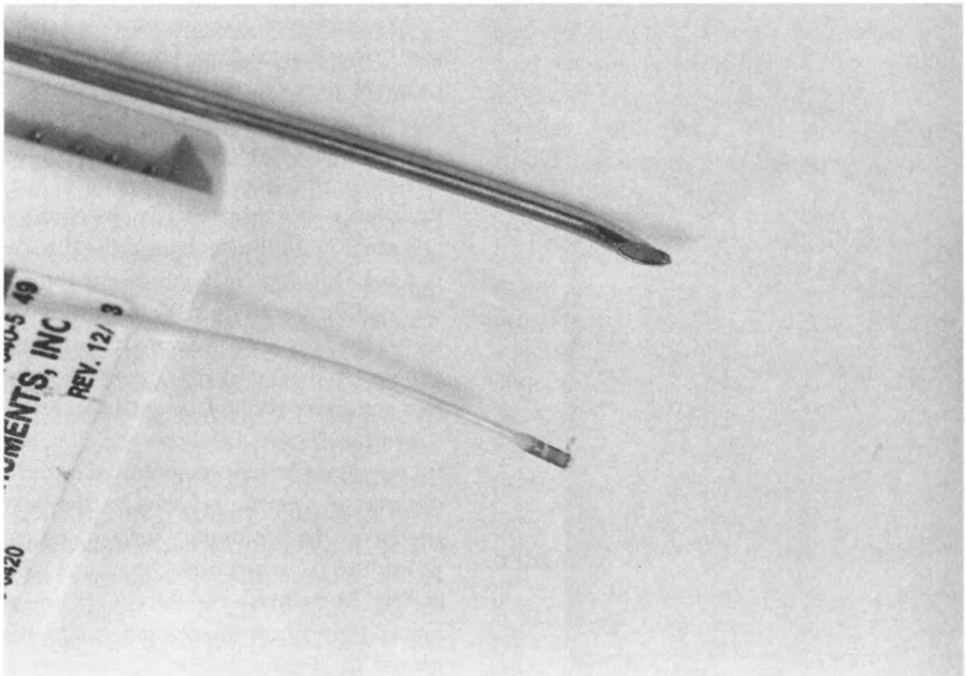

Figure 8.5. The pressure transducer and the needle.

Figure 8.6. The mounting of the pressure transducers in the cadaver.

trial. Mean values of the pressures before each cycle of the treatment procedure, pressures in the distracted position, and the changes in the pressures were computed for all 15 cycles of the three trials.

RESULTS

Figure 8.7 shows a typical plot of the change in the intradiscal pressure at an L4–L5 disc during five, 4-second applications of the flexion-distraction procedure. The same graph also shows the downward table motion. The downward table motion and the decreases in intradiscal pressure changes are in same time phase. The pressure returns to its original value during the upward movement of the table.

Tables 8.1 and 8.2 list the means and standard deviation values of the intradiscal pressures before the treatment cycle and in the distracted position. The flexion-distraction procedure significantly decreased the intradiscal pressure in both the unpressurized and pressurized discs. In the unpressurized discs, the disc pressure went into the negative range at the distracted position corresponding to the extreme downward motion of the table. The decrease in intradiscal pressure varied from 39 to 192 mm Hg among the four discs tested in unpressurized mode (mean: 88.6, standard deviation [SD]): 64.2). The decrease in intradiscal pressure was statistically significant ($p < 0.01$). The injection of water in the disc raised the initial disc pressure to a mean value of 456 mm Hg (SD 227) in the prone position. The decrease in pressure ranged from 117 to 720 mm Hg (mean: 330, SD 222) during the procedure and the decrease was statistically significant ($p < 0.01$).

Discussion and Conclusions

A significant decrease in intradiscal pressure during the flexion-distraction procedure for low back pain was observed. When

Table 8.1

Mean Intradiscal Pressures (mm Hg) During the Flexion-Distraction Procedure (Discs Not Pressurized)

Cadaver No.	Joint	Pressure in Initial Prone Position	Pressure in Distracted Position	Decrease in Pressure
3	L3–L4	27 (22)	−165 (37)	192 (37)
4	L2–L3	24 (7)	−15 (2)	39 (9)
5	L3–L4	13 (13)	−48 (2)	61 (12)
	L4–L5	−87 (37)	−150 (11)	63 (10)

For cadaver No. 5, two joints were monitored using two transducers. Only three cadavers were monitored without pressurization. The numbers in parentheses represent standard deviation values for 15 cycles.

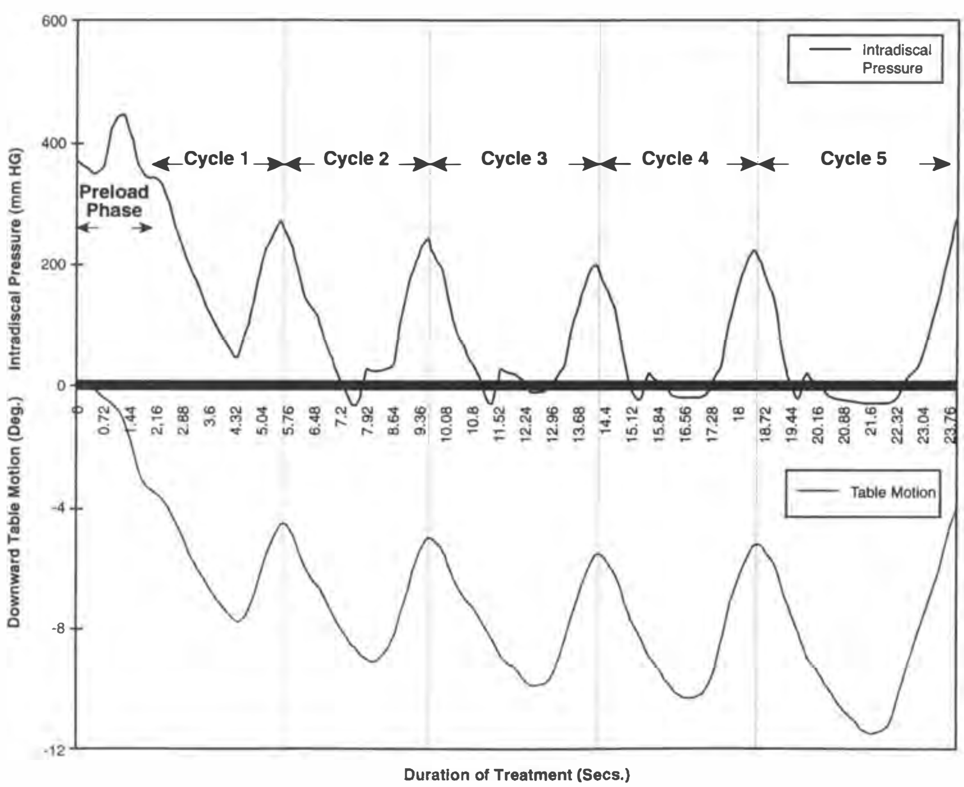

Figure 8.7. Graph showing the changes in the intradiscal pressure during table up-and-down motion.

Table 8.2

Mean Intradiscal Pressures (mm Hg) During the Flexion-Distraction Procedure (Discs Pressurized with Water)

Cadaver No.	Joint	Pressure in Initial Prone Position	Pressure in Distracted Position	Decrease in Pressure
1	L4–L5	417 (20)	144 (5)	271 (17)
2	L4–L5	823 (290)	103 (48)	720 (272)
3	L3–L4	279 (87)	−34 (34)	314 (83)
4	L2–L3	266 (49)	149 (10)	117 (45)
5	L3–L4	432 (37)	162 (36)	271 (28)
	L4–L5	519 (37)	232 (61)	287 (50)

For cadaver No. 5, two joints were monitored using two transducers. The numbers in parentheses represent standard deviation values for 15 cycles.

the discs were not pressurized, the pressures went below 0 mm Hg. When the discs were pressurized, the decrease in the intradiscal pressures was much larger, suggesting that in patients with higher intradiscal pressures, the decrease may be much higher during the treatment. The pressures returned to their original values when the spine was brought back to the initial prone position.

Cyriax (10), Quillette (11), and Kramer (12) hypothesized that as the vertebrae in the spine are distracted, a negative pressure develops in the disc, and sucks back a protrusion. Nachemson and Elfstrom (13) pioneered the measurement of intradiscal pressures during in vivo conditions of daily activities. Ramos and Martin (14) reported on the intradiscal pressure during a vertebral axial decompression (VAD) procedure on three patients measured intraoperatively. The results showed that the disc pressures reduced during the VAD therapy. They demonstrated that the disc pressures can go as low as −160 mm Hg. The results of the present study are in general agreement with the study reported by Ramos and Martin (14). Andersson et al. (15) reported the intradiscal pressures at L3–L4 disc on four volunteers during standing, lying, active traction, and passive traction. The findings showed an increase in disc pressure during both active and passive traction. The results from the present study do not agree with those results (15). A possible reason could be that the muscles of the in vivo subjects could have been contracting while under active and passive traction. Work is in progress to monitor the muscle activity during in vivo situations of treating patients using flexion-distraction procedure.

Estimation of the Loads on the Ligaments and Disc of the Lumbar Spine

The flexion-distraction procedure uses combined loads of traction and flexion to a particular motion segment. Both flexion and traction increase the loads on the posterior ligaments. This section presents quantitative data on the loads of the posterior ligaments of a lumbar motion segment (L4–L5) under loading conditions of traction and flexion. The analytic model of the lumbar motion segment was developed to estimate the ligament loads under the application of traction and flexion loads.

Model Development and Methodology

In our model the lumbar motion segments were idealized as a mechanical system of rigid bodies connected by means of springs and constrained by kinematic pairs. The vertebrae were idealized as rigid bodies. The posterior ligaments (yellow ligaments, interspinous ligament, supraspinous ligament, capsular ligaments, intertransverse ligaments) were modeled as simple linear elastic springs. The intervertebral disc, including the anterior and posterior longitudinal ligaments, was modeled as an elastic member capable of resisting bending, and shear and axial forces. The facet joints were modeled as two convex curved surfaces (one for the right and the other for the left articulating processes of the inferior moving vertebrae) that may come in contact with another two concave surfaces representing the superior articulating processes of the fixed vertebrae. These curved surfaces can be in contact with one another or can lose contact, thus representing the true behavior of facet joints. Figure 8.8 describes the model idealization and the forces acting on the motion segment, disc, ligaments, and facet joints. The geometric parameters and the elastic properties (16–20) from the existing literature were incorporated into the model. The methodology is based on the principle of static equilibrium in the displaced position caused by the external forces that has been used by Hong and Suh (21). External moments were applied in increments. The three-dimensional displacement matrix (consisting of three translations and three rotations) approach was used for the derivation of the equilibrium equations. The equilibrium conditions were applied in the displaced position of the moving vertebrae. The equilibrium equations have nonlinear relationships between the external forces and the displacements. The constraint equations of the facet joints were applied whenever facet joints come in contact with one another. These nonlinear simultaneous equations were then solved by means of the Newton-Raphson iterative procedure.

Input Data to the Model

An L4–L5 motion segment was subjected to flexion external moment load in increments of 530 N-mm so that the displacements in flexion reached 3°, 6°, and 12° along with traction loads of 222 N, 444 N, and 888 N. The stiffness properties of the intervertebral disc and the ligaments were varied to cover a range describing the individual variations available in the literature. Using these elastic properties available in the literature the responses of the motion segment were obtained under the combined loads of traction and flexion moment.

Ligament Loads

Figure 8.9 shows the results of the estimated loads on the ligaments under the combined loads of flexion and traction. Also

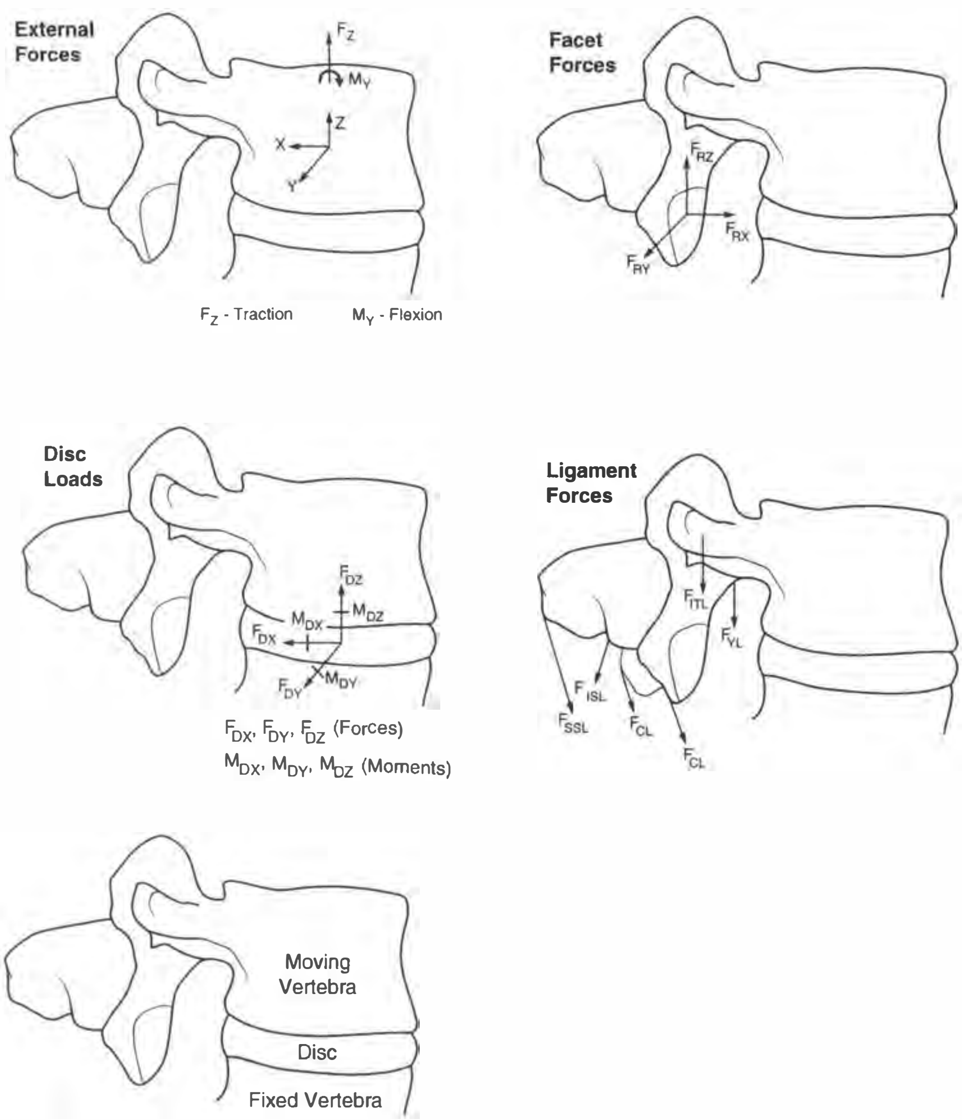

Figure 8.8. Schematic diagram showing the model and the forces.

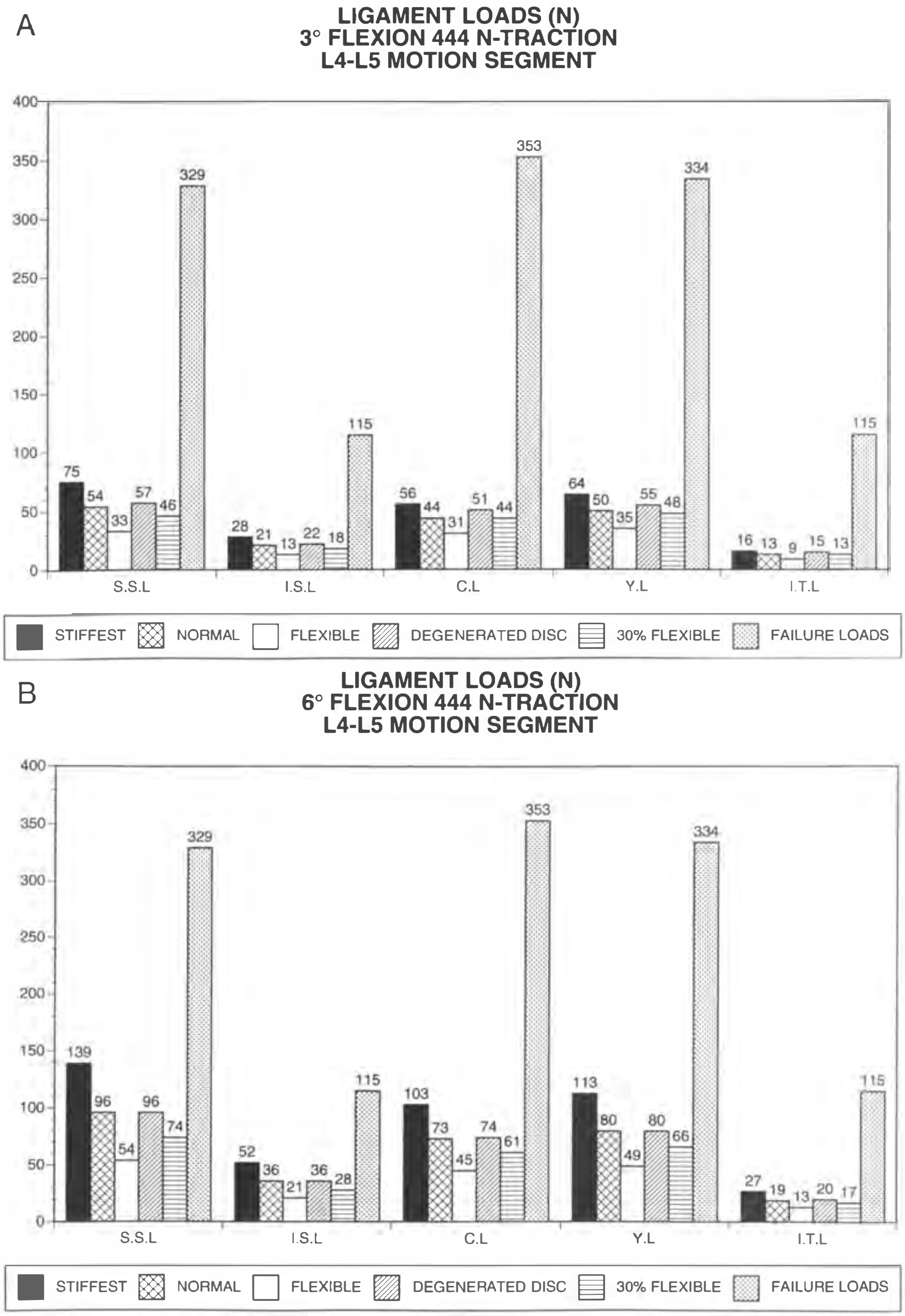

Figure 8.9. Ligament loads under different conditions of loading.

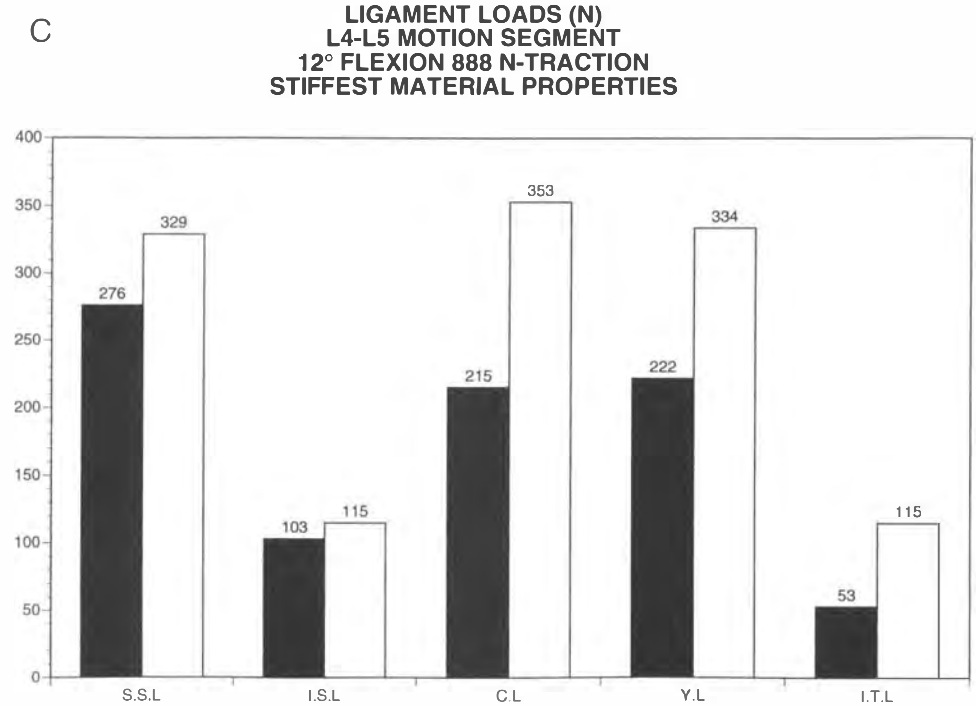

Figure 8.9.—*continued*

shown are the failure values reported by Myklebust et al.(22) for the various ligaments. The ligament loads were estimated for a variety of material properties of the motion segment such as stiffest condition, average conditions, highly flexible conditions, degenerated conditions. As can be seen from the graphs the loads on the ligaments are well below the failure loads under 3° of flexion and 222 N of traction as well as 6° of flexion and 444 N of traction loads for all types of material conditions. However, under loading conditions of 12° of flexion and 888 N of traction the ligament loads do approach the failure loads for the respective ligaments under stiffest material conditions. This suggests that caution has to be exercised while treating stiff patients with large table motions and traction loads.

Some of the research results presented in this chapter were presented at conferences Gudavalli et al. (23–25) and work is under progress to complete the data analysis and submit manuscripts.

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Biomechanics, Adjustment Procedures, Ancillary Therapies, and Clinical Outcomes of Cox Distraction Technique

James M. Cox, DC, DACBR

chapter 9

Accept the challenges, so that you may feel the exhilaration of victory.

—General George S. Patton

Fifty-three percent of chiropractic physicians use Cox distraction manipulation in patient care (1) and it is one of two "established" techniques in chiropractic (2). The biomechanics and effects of Cox distraction manipulation, the protocols of its proper implementation in clinical practice, and the outcome study of 1000 patients treated with it will be presented in this chapter. The outcome study shows:

- Fewer than 4% of low back or leg pain patients were candidates for surgery
- Fewer than 9% of low back patients reached the chronic stage of care
- Mean number of days to maximal improvement with chiropractic adjusting and care is 29
- Mean number of treatments to maximal improvement is 12

Cox flexion-distraction manipulation can be successfully used to treat back pain problems, from simple sprains or strain to serious disc herniations.

Spinal manipulation has been equated with the practice of chiropractic and 94% of manipulative therapy performed in the United States is performed by chiropractic doctors (3). Chiropractic represents the most rapidly growing segment of the professional health care services market (4). Cox distraction technique has been described in a reviewed text and in a number of well-respected, peer-reviewed journals, by doctors professing to use distractive procedures, and is the only procedure in which any statistical analysis has been done on clinical effects for various conditions (5).

DECISION-MAKING IN THE CARE OF THE LOW BACK PAIN PATIENT WITH AND WITHOUT SCIATICA

A One-Month Course of Manipulation Recommended

Following are some reports on clinical research in the treatment of low back pain. Spinal manipulation is appropriate for low back pain without indications of sciatica. An all-chiropractic panel states that "an adequate trial of spinal manipulation is a course of 12 manipulations given over a period of up to 4 weeks, after which, in the absence of documented improvement, spinal manipulation is no longer indicated" (6). Spinal manipulation is safe and effective for patients in the first month of acute low back pain symptoms without radiculopathy. For patients with symptoms lasting more than 1 month, manipulation is probably safe, but its efficacy is unproved. If manipulation has not resulted in symptomatic and functional improvement after 4 weeks, it should be stopped and the patient re-evaluated (7).

Chiropractic As an Alternative to Hospitalization

Nearly half of the hospitalizations in the United States for patients with nonspecific back pain and herniated discs were for diagnostic tests or pain control, which are safely performed in the outpatient setting. A need is seen for improved outpatient and home-based alternatives to hospitalization (8).

Two to Three Months of Conservative Care Before Surgery for Disc Herniation

Conservative care for 2 to 3 months is reasonable for disc herniation patients before surgical consideration is considered. Patients with radicular symptoms and signs caused by a herniated lumbar disc, but without definite indications for immediate surgery, should be observed for 2 to 3 months before a decision is made regarding surgery (9, 10).

Approximately 2% of all persons with low back pain undergo surgery for disc herniation (11). Surgical candidates include patients with cauda equina syndrome and those with neurologic abnormalities that suggest a herniation who have not responded to 3 to 4 weeks of conservative therapy or who exhibit progressive neurologic deficit (12).

Ninety percent of sciatica patients will get well with 4 months of energetic, non-operative, conservative care. Definite indications for surgery are cauda equina syndrome, intolerable pain, and progressive muscle weakness. Furthermore, the decision to continue the conservative regimen or to perform surgery should always be made with the patient (10).

Nonsurgical Care Provides Good Outcomes

Schvartzman et al. (13) comment that when a trial of conservative treatment fails in patients with herniated lumbar intervertebral discs (IVD), surgery is usually recommended. However, they state that surgical care is not more cost effective than non-surgical care, and it has no better outcome than continued conservative care. An initial 3 months of therapy is recommended, and, if the patient's condition does not deteriorate during that time, conservative measures should be continued. A patient not responding to the initial trials of conservative therapy has the option to undergo continued conservative treatment or to choose surgical intervention.

Continuing conservative treatment is usually safe when pain is the principal problem, and progressive neurologic, motor, or bowel and bladder dysfunction are not present. In comparisons of the efficacy of conservative therapy and surgery, no significant difference in recovery of function has been reported between patients whose herniated discs resolved spontaneously and those whose discs were surgically removed. Because only 5 to 10% of patients with radicular pain require surgery, surgery should be considered only if symptoms have not been significantly alleviated after 6 weeks of conservative therapy (14).

Between 2 and 10% of disc herniation patients may require surgery. Surgery is necessary when cauda equina symptoms are present or when there is progressive neurologic deficit or when the pain is intolerable. The decision to continue with the conservative regimen or go to surgery should be the choice of the patient.

CHIROPRACTIC DISTRACTION ADJUSTMENT—A POPULAR CONSERVATIVE TREATMENT REGIMEN

To be considered a "health care system" or "healing technique," an alternative method must claim to be curative; it must possess a systematized body of knowledge or theory and a technical intervention; and it must be executed by expert practitioners (15). More than 60% of all physicians referred patients to alternative providers at least once in the preceding year and 38% in the preceding month. Spinal manipulation is the most common referral condition to alternative providers (16).

Technique

Cox axial flexion distraction adjusting procedures, developed in 1973, were named "Cox distraction manipulation." The technique has advanced in use through research, clinical validation, and practitioner preference in treating many cases of low back pain of different causes. I never stated my procedures to be a singular treatment; rather, it is often combined with other forms of chiropractic treatment.

Why such a growth in this technique? *I think because it combines two biomechanical models—axial distraction and flexion—in the treatment of lumbar spine pain conditions.*

This technique is acknowledged to be a marriage of chiropractic and osteopathic biomechanical models of spinal manipulative adjustment. The work of John McManis, DO, developer of the McManis osteopathic table, was described by Stoddard (17).

> *"'McManis Technique' can be used in complete safety in all mechanical and disc lesions in the lumbar spine and is a movement used almost as a routine measure in the majority of cases with lumbar lesions."*

To ensure adequate venous drainage in the vertebral column, all the intervertebral joints should be freely moveable. Any restriction of movement (the most important quality of the osteopathic spinal lesion) in the spinal column is going to slow down the venous drainage in that area.

The purposes of traction are the adjustment of position, the freeing of longitudinal adhesions, the relief of nerve root pressure, the separation of apophyseal joints, and the obtaining of a circulatory effect to decongest the intervertebral foramen and reduce the hydrostatic pressure inside the disc.

Stoddard states principles of osteopathic technique as follows:

- Make a diagnosis
- Restore normal mobility
- Relax or stretch extraneous structures
- Restore mobility by passive movements to intrinsic structures by slow and rhythmic methods rather than sharp quick movements—long lever techniques
- Indirect specific adjustments to restore mobility

- A specific thrust to alter the relationship of one vertebra with the one above and below
- Use a minimum amount of force that is consistent with achieving the objective; undue force is negative

The osteopathic lesion is any structural disturbance with consequent functional deflection. An osteopathic lesion is any departure from the normal relation of skeletal units that affects function detrimentally by limiting articular motion.

Where the purpose of traction is the "adjustment of position," either the a herniated disc is repositioned or adjacent vertebral bodies realigned (17).

Note: Along the thinking of McManis and Stoddard, I treat all low back pain conditions with axial distraction adjustments. Intervertebral disc herniations represent only 5 to 10% of my patient load. The remaining 95% are patients with low back and thigh pain but *no sciatica*. These patients are treated with axial distraction adjustments, beginning with axial flexion distraction, followed by motion palpation of the facet joints through their normal ranges of motion, and restoration of physiologic ranges of motion. Therefore, to clarify my work: I treat all low back pain conditions with either manual or automated axial distraction, not just cases of intervertebral disc herniation.

DEFINITION OF COX FLEXION-DISTRACTION ADJUSTING

Cox flexion-distraction adjusting is a form of chiropractic adjustment of the intervertebral disc, posterior facet elements, and osseoligamentous canals that provides the following benefits:

1. Increase the IVD height to remove anular distortion within the pain-sensitive peripheral portion. The anulus fibrosus bulges into the concave side or the posterior lordotic curve of the lumbar spine, and distraction under slight traction reduces this protrusion (18–20).
2. Decrease intradiscal pressure by creating a centripetal force on the protruding nucleus pulposus to allow it to assume its more central position within the anulus fibrosus (21, 22).
3. Remove subluxation of the facet articulations and restore physiologic motion to the posterior elements of the vertebral motion segment.
4. Improve posture and locomotion while relieving pain, improving body function, and restoring a state of well-being.

Caution and knowledge must be applied in distraction techniques as certain traction techniques can actually cause an increase in intradiscal pressure (23), which would be undesirable in the treatment of low back pain associated with herniated discs and neurocompression. Sensitive to this point, I will next discuss the biomechanical differences in applying distraction in flexion and extension postures of the lumbar spine, and point out the advantages of flexion of the lumbar curvature when distraction is applied.

EFFECTS OF FLEXION-DISTRACTION ADJUSTMENTS

Positive Effects of Flexion Distraction

Figure 9.1 outlines the physiologic and therapeutic effects of applying flexion distraction adjustment to the lumbar spine. These effects are as follows:

1. The posterior disc space increases in height (22, 24–26).
2. Flexion decreases disc protrusion and reduces stenosis (22, 24, 25, 27–29). Note: Discs protrude and degenerate into the concavity of a curve, and into the side of extension, lordosis, or lateral flexion (19).
3. Flexion stretches the ligamentum flavum to reduce stenosis (24, 25).
4. Flexion opens the vertebral canal by 2 mm (16%) or 3.5 to 6 mm (28, 29).
5. Flexion increases metabolite transport into the disc (20).
6. Flexion opens the apophyseal joints and reduces posterior disc stress (20, 30).
7. The nucleus pulposus does not move on flexion (31). Intradiscal pressure drops under distraction (22) to below 100 mm Hg (21). On extension the nucleus or anulus is seen to protrude posteriorly into the vertebral canal (25, 27, 32, 33).
8. Intervertebral foraminal openings enlarge giving patency to the nerve or dorsal root ganglion (DRG) (30).

Extension-Distraction Effects

Figure 9.2 outlines the effects of applying extension-distraction adjustment to the lumbar spine. These effects are as follows:

1. The posterior disc space decreases in height (25).
2. Extension causes discs to protrude and produces stenosis (19, 25, 27–29).
3. Extension causes the ligamentum flavum to buckle into the

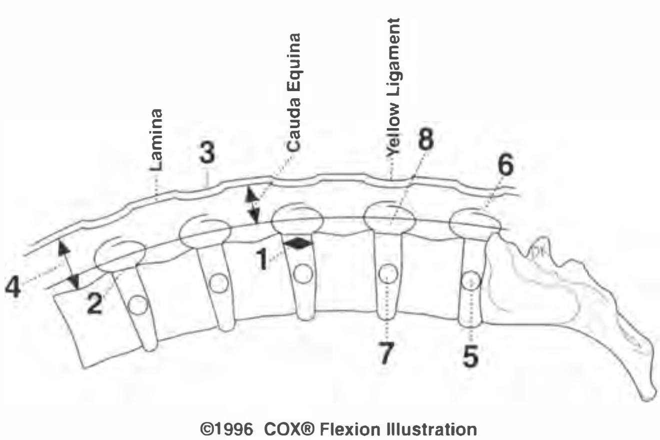


Figure 9.1. Flexion distraction positive effects on the intervertebral disc space height, intervertebral osseoligamentous canal diameter, and facet joint spacing and subluxation.

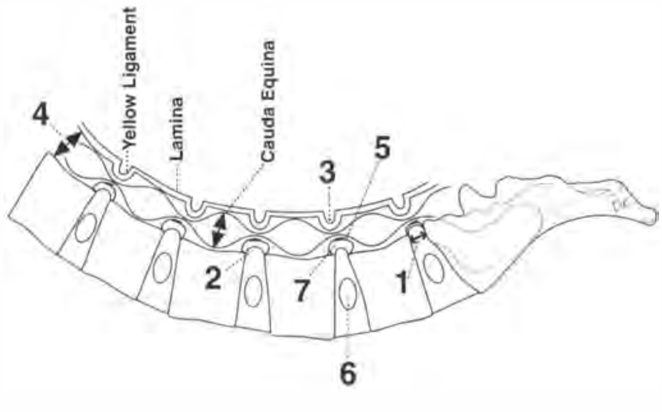


Figure 9.2. Extension distraction effects on the intervertebral disc space height, intervertebral osseoligamentous canal diameter, and facet joint spacing and subluxation.

vertebral canal causing stenosis and possibly cauda equina compression (25).

4. Extension causes the vertebral canal to close 2 mm (16%) or 3.5 to 6 mm from flexion causing stenosis (28, 29).
5. Extension closes the apophyseal joints and increases posterior disc stress (25, 30).
6. The intradiscal pressure is greater on extension. Nucleus pulposus and anulus fibrosus move posterior on extension (24, 25, 27, 30, 32, 34).
7. Extension causes the intervertebral foraminal openings to close, which causes stenosis to the nerve (30, 34).

FLEXION-DISTRACTION EFFECTS ON THE LUMBAR SPINAL CANAL AND INTERVERTEBRAL FORAMEN CAPACITY

Flexion Reduces Disc Protrusion

Figures 9.3 and 9.4 show that on flattening or flexion of the lumbar spine, the disc anulus fibrosus protrusion reduces; on extension, the anulus fibrosus bulges into the vertebral canal to cause spinal stenosis (24, 25).

At 6° to 8° degrees of flexion and extension the disc bulges anteriorly during flexion and posteriorly during extension, and toward the concavity of the spinal curve during lateral bending. *Discs protrude into the concavity of a curve* (35) (Fig. 9.5). When placed into a "U" shape, rat tail discs herniate and degenerate into the concavity of the curve (19).

Extension can cause posterior bulging of the lamellae in the posterior anulus (30). Avoid extension in distraction of back pain patients because of increased posterior disc protrusion.

Flexion reduces the posterior concavity of the lumbar spine and allows reduction of disc protrusion while spreading open the facet joints to increase the spinal canal openings. Extension increases the posterior concavity and accentuates the disc bulge, whereas it induces facet imbrication subluxation. Because the goal of distraction adjusting is to reduce disc bulge and stenosis, avoid extension and use flexion in disc bulge or stenosis.

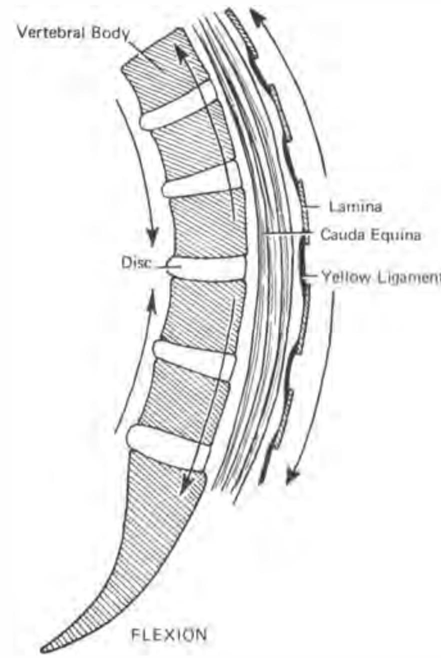


Figure 9.3. Increased spinal canal volume and decreased nerve root (cauda equina) bulk with flexion. (Reprinted with permission from Finneson BE. Low Back Pain, 2 nd ed. Philadelphia: JB Lippincott, 1980:432.)

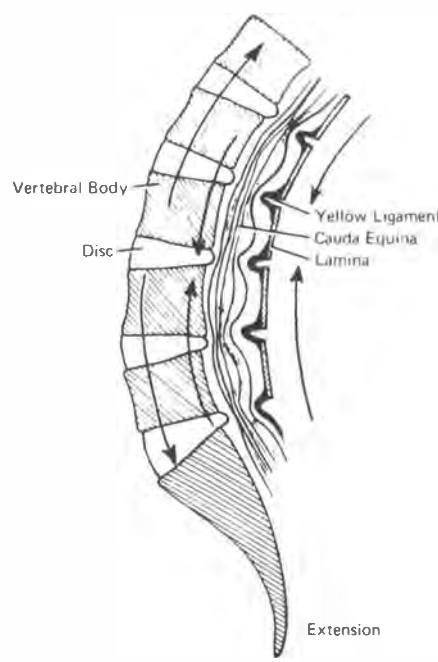


Figure 9.4. Decreased spinal canal volume and increased nerve root bulk with extension. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980;432.)

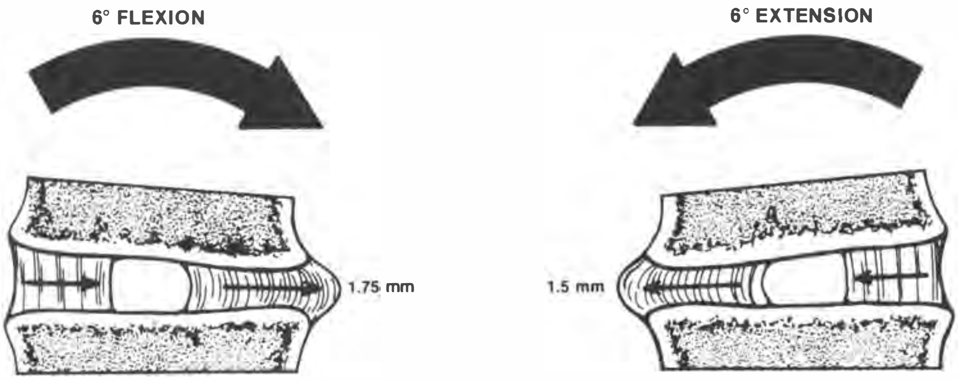


Figure 9.5. Disc protrusion with bending. Flexion as well as extension of the spine produces motion of the disc in the horizontal plane. In both cases of bending, bulging of the disc occurs on the concave side and contraction on the convex side. In a pathologic case, the expansion of the disc during physiologic bending may stretch or impinge the nerve root. (Based on the data of Brown T, Hanson R, Yorra A. Some mechanical tests on the lumbosacral spine with particular reference to the intervertbral discs. J Bone Joint Surg Am 1957;39A:1135.)

Magnetic resonance imaging (MRI) studies of the cervical spine in flexion show reduced disc herniation, whereas extension produced disc herniation (27).

Ligamentum Flavum

Flexion allows the ligamentum flavum to tauten and decrease its bulging into the vertebral canal, whereas extension causes it to bulge into the canal to create stenosis and further nerve root compression (24, 25) (Figs. 9.1 and 9.2).

Vertebral Canal Diameter Changes with Flexion

Flexion *Increases* the Spinal Canal Space

Schonstrom et al. (28) report that *flexion increases the sagittal diameter of the vertebral canal 16% or 2 mm* over extension. Computed tomography (CT) scan study of human lumbar spine specimens demonstrated a 40 mm^2 decrease in the cross-sectional area of the vertebral canal when the spine was moved from flexion to extension. Extension caused stenosis of the vertebral canal, a negative influence to nerve root compression.

Flexion *Increases* Spinal Capacity

Liyang et al. (29) report that the lumbar spinal capacity in flexion-extension lateral myelogram motion studies of ten cadavers showed a larger capacity of the dural sac in flexion overextension by 3.5 to 6 mm. This increased spinal capacity is highly significant, and it suggests that maintaining the flexed lumbar spine enlarges the spinal canal capacity and mitigates symptoms.

Flexion Reduces the "Pincer Effect" Narrowing of the Spinal Canal

Penning and Wilmink (30) show widening of the spinal canal with relief of pain in flexion whereas its narrowing in extension created a "pincer effect" of the canal.

Cervical Spine Foraminal Size Changes

Farmer and Wisneski (36) reported that cervical spine extension significantly increased nerve root pressure and radicular symptoms, whereas results with neck flexion were variable. A decrease is seen in foraminal size in extension. In flexion, Yoo et al. (37) reported that foraminal size increased 8 and 10% at 20° to 30°, respectively, and extension reduced the foramen diameter by 10 and 13% at 20° and 30° of extension.

A 15% reduced foraminal and spinal canal dimension was seen in extension. Nerve root compression in the foramen was 21% in the neutral posture, 15% in flexion, and 33% in extension (38). Extension loading of the lumbar spine produced the most cases of nerve root compression, whereas lateral flexion produces the fewest cases (39).

Flexion *Improves* Disc Metabolism

The disc receives nutrients from two sources: the blood vessels in the vertebral bodies and the tissue fluid surrounding the anulus fibrosus. Fluid flow into the disc depends on changing pressures within the disc structure. High pressure occurs in compression and weightbearing, and it forces fluid out of the disc, whereas low pressure, as in lying down, allows fluid to be sucked up by the disc, primarily the nucleus pulposus. Flexion improves the transport of metabolites in the intervertebral disc, reduces the stresses on the apophyseal joints and on the posterior half of the anulus fibrosus, and gives the spine a high compressive strength (20).

The erect upright posture allows diffusion more readily into the anterior anulus than the posterior anulus. The inner posterior anulus is the most critical area of the disc to be deprived of nutrients; flexion improves transport of metabolites into the inner posterior anulus (40). Improving the metabolic transport in the disc is of value as the glucose supply to the disc is barely adequate (41). Deficient metabolite transport has been linked with degenerative changes in the disc (42, 43).

Intradiscal Pressure Changes on Distraction

Ramos and Martin (21) report that the pressure in the nucleus pulposus of lumbar discs dropped to below −100 mm Hg when axial distraction decompression was administered. Contrast this to the intradiscal pressures reported by Nachemson (40), which ranged from 25 mm lying prone to 275 mm sitting flexed. Extension exercise of the lumbar spine created 180 mm of intradiscal pressure, and hip flexion caused 150 mm of pressure (40). I correlate these findings clinically to maintaining low intradiscal pressure by lying prone and applying axial distraction to provide further lowering of intradiscal pressure, which creates centripetal force within the nucleus to retract the bulging nuclear material from the anular area of the disc. Onel et al. (24), discussed later, and Burton (22) (Fig. 9.6) describe the influence of negative intradiscal pressure in disc herniation reduction.

NUCLEUS PULPOSUS AND ANULUS FIBROSUS MOVEMENT DURING FLEXION AND EXTENSION

Management of patients with low back pain is often based on theorized positional changes of the nucleus pulposus during spinal extension and flexion. I feel the anulus fibrosus is the primary part of the disc that protrudes in disc degeneration, not the nucleus pulposus. Certainly, with nucleus pulposus degeneration and dehydration and attending loss of intradiscal pressure, the nucleus pulposus allows greater tendency for the anulus fibrosus to splay out or protrude into the peripheral area of the disc. This increased anulus fibrosus laxity and motion can be compared to a tire inner tube that has been partially deflated—it has greater propensity to flatten and bulge out.

Certainly, in patients in whom the nucleus is herniating through a radial tear in the anular fibers, the doctor is sensitive to movement of the nucleus pulposus when performing axial distraction, flexion, and extension. The patient with sciatica and nerve root compression caused by a herniated nucleus pulposus through a contained or noncontained anulus fibrosus has an abnormal disc. This disc will in no way perform as a normal turgid and contained nucleus pulposus.

Because of the degenerative changes of the nucleus pulposus that are inevitable with aging, any discussion of nucleus pulposus movement on flexion, extension, lateral flexion, or rotation must compare normal versus degenerated nucleus pulposus.

Opinions Regarding Nucleus Pulposus Shift on Motion

Nucleus pulposus movement is not affected by flexion. Extension causes posterior nuclear shift.

Vanharanta et al. (31) with CT or discography showed no notable change in the location of the nucleus with respect to the anulus on flexion and extension motion. Flexion did not increase nuclear shift posteriorly. Vanharanta et al. challenge the suggestion that the disc nucleus moves anteriorly in extension and posteriorly in flexion.

Gill et al. (32) reported on 103 cadaver discography studies

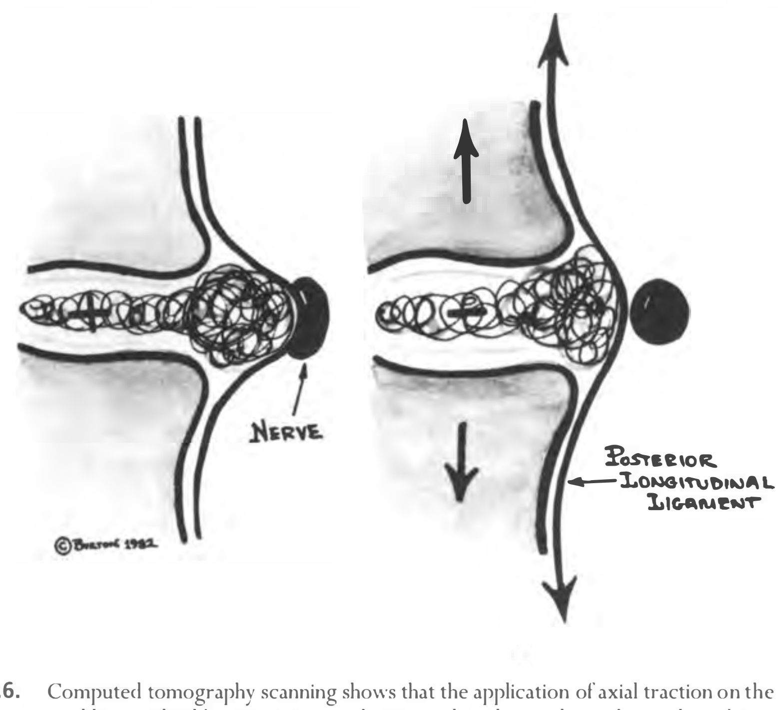


Figure 9.6. Computed tomography scanning shows that the application of axial traction on the vertebrae, anulus fibrosus, and longitudinal ligaments causes the protruding disc to diminish in volume but rarely to return to its normal state. The clinical problem relates to distention of anular and liagamentous dorsal ramus nerve fibers and spinal nerve compression. It is believed, on the basis of biomechanical calculation, that significant intradiscal negative pressures may be produced. The intermittent reduction appears to allow reparative processes to re-establish support. (Reprinted with permission from Burton CV. Gravity lumbar Reduction. In: Kirkaldy-Willis WH, ed. Managing Low Back Pain. New York: Churchill Livingstone, 1983:350.)

and showed the major effect of repeated extension moments on cadaveric lumbar spine motion segments appeared to lie in forcing dye from the nucleus pulposus into the spinal epidural space in many abnormal discs. This would not be a desirable effect in manipulation, and it is good reason to avoid extension in acute back pain disc protrusion patients.

Roaf (44) reported that the nucleus pulposus does not change shape or position during flexion or extension.

From these three studies two facts evolved:

1. The nucleus pulposus does not move anterior in flexion or extension.
2. The nucleus pulposus moves posterior in extension motion of the lumbar spine.

Movement of Normal and Abnormal Nucleus Pulposus

Seroussi et al. (33) placed metal beads throughout the disc, followed by flexion and extension compression studies of the disc. This showed that on extension the beads in the center of the disc moved in an anterior-superior direction, whereas the beads closer to the periphery of the disc moved posteriorly. On flexion, the beads in the center of the disc moved posteriorly, whereas the beads closer to the periphery of the disc moved anteriorly.

Beattie et al. (45) studied 20 healthy young women with lumbar spine MRI while they were supine with their hips and knees flexed (flexed position) and supine with a lumbar roll under the low back (extended position). The distance of the posterior margin of the nucleus pulposus to the posterior margins of the adjacent vertebral bodies was greater in the extended position compared with the flexed position *in healthy discs*. No difference was seen in the anterior distance. Of the 20 subjects 8 had at least one degenerative disc in the lower lumbar spine. The degenerative disc nucleus pulposus did not move the same as normal discs. Degenerative discs deform differently from nondegenerative discs.

Schnebel et al. (46, 47) used discography to study position change in vivo of the nucleus pulposus during flexion and extension. Results suggested that, in normal discs, the nuclear material moves anteriorly with extension and posteriorly with flexion. Schnebel et al. also used a digitizing technique to measure the position change of the nucleus pulposus from discograms obtained from subjects with low back pain. These subjects were studied in a flexed position (knees to chest) followed by an extended position (press-up extension). A significant difference was reported in the posterior distance of L3–4, L4–5, and L5–S1 between flexion and extension for normal nucleus pulposus.

Beattie et al.'s (45) study results suggest that the nucleus pulposus deforms and may possibly move within the intervertebral disc (IVD). Dietrich et al. (48) reported lateral shift of nuclear material when small loads, similar to those of daily life, were applied to the spine. They reported that traction reduced herniation 40%.

Fennell et al. (49) studied nuclear movement on flexion and extension of one normal and two patients with low back pain histories. In the normal patient, flexion tended to be accompanied by posteriorly directed migration of the nucleus pulposus. Extension tended to be accompanied by an anteriorly directed migration. Only L4–L5 levels were studied as it was technically impossible to study L5–S1. The two patients with low back pain histories showed that the anterior margin of the disc moved anteriorly during flexion; therefore, the authors state that the nucleus spread during flexion instead of migrating posteriorly. The final assessment of this paper shows what others have found—in abnormal discs the nuclear movement on flexion and extension is unpredictable. In two of three of these test subjects with low back pain histories, the nucleus did not move anteriorly on extension, but it did move anteriorly on flexion (49).

Abnormal Discs Show Little Difference in Position

Nuclear Motion Is Posterior in Extension in Abnormal Discs

In those subjects with an abnormal disc (a decreased nucleus pulposus signal on MRI with an irregular outline of the transition between the nucleus pulposus and anulus fibrosus on T2-weighted images), little difference was found in the shape and location of the nucleus pulposus between positions. Similar observations were reported by Schnebel et al. (47, 48) and Urban and McMullin (50).

In four of the eight subjects with degenerative discs, the nucleus pulposus of the involved segment was observed to "bulge" posteriorly in the extended position (45).

The concept that a motion segment with a degenerative nucleus pulposus may not move in the same manner as a one with a normal nucleus pulposus may be important clinically. We treat patients who have low back or leg pain, and their pain indicates probable abnormal disc morphology. Because movement of the nucleus pulposus appears to differ between normal and abnormal IVDs, Beattie et al. (45) question whether nuclear movement can be used to justify the McKenzie approach when treating individuals with degenerative disc disease. In addition to degenerative disc disease, other disorders such as herniated discs, bony abnormalities, and neuromuscular impairment can influence the displacement of the nucleus pulposus as a function of position.

Summary

Beattie et al. (45) state that an abnormal nucleus pulposus in the motion segments of L3–L4 to L5–S1 may not move in the same manner as a normal nucleus pulposus.

I feel that with degeneration, the nucleus pulposus ceases to be the primary factor in back mechanics. The anulus fibrosus protrudes in all directions, but its posterior protrusion is especially harmful because of the cauda equina, nerve roots, and dorsal root ganglion lying in close proximity, which are subject to compression or chemical inflammation. Kokubun et al. (51) found that herniated disc fragments at surgery contain particles

of cartilaginous end plate with nucleus pulposus and anulus fibrosus. Harada and Nakahara (52) found that fragments of cartilaginous end plate more often contained anulus fibrosus than nucleus pulposus. Tanaka et al. (53) stressed that the inner fibers of the anulus fibrosus pull the cartilaginous end plate causing it to herniate with its fragment.

DISC STRESSES UNDER FLEXION AND EXTENSION

High Nucleus Pulposus Intradiscal Pressure Effect on Disc Bulging

Compression loading of lumbar motion segments in backward bending (extension) can cause an anterior disc prolapse if sudden force is applied, and a cyclic compressive force can increase the posterior bulging of the anular lamellae (34). Adams et al. (34) further equate standing posture as extension, and state that any action increasing the standing lordosis causes the limit of extension to be approached. Extension causes posterior anular bulging by the combined increased intradiscal pressure in the nucleus pulposus (54) and the compression load on the posterior anulus (55). The compression causes the lamellae of the anulus to buckle, whereas the increased intradiscal pressure causes the lamellae to radiate outward. Brinckmann and Horst (56) emphasize that extension places a high compressive force on the posterior lamellae, and also stresses the disc to cause herniation because high intradiscal pressure alone cannot cause the disc to bulge. In fact, increasing nuclear pressure by fluid injection reduces rather than increases disc bulging.

Disc Strength in Flexion

Bogduk (57) states that, during flexion, the lumbar spine appears to be well protected against injury by the posterior ligaments, intervertebral discs, and back muscles. A normal healthy IVD is designed to sustain heavy loads in flexion, and it is not susceptible to rupture. Normal discs suffer acute herniations only with severe hyperflexion injuries involving forces and ranges of motion well outside those within a normal activities of daily living.

Adams (58) advocates heavy lifting be done in the flexed position rather than in the lordotic lumbar spine posture. He states the lordotic posture exposes the posterior structure of the spine to excessively high stress levels. A bent posture for lifting does not greatly raise intradiscal pressure, and, at high load levels, the anterior anulus appears to "stress shield" the nucleus from damage. Adams feels workers can lift safely in postures that are within the normal range of flexion.

Adams shows that the lordotic extended posture exposes the posterior area of the spine to excessively high stress levels, and that flexed postures transmit stress through the anterior anulus and low stress through the posterior anulus. The anterior anulus is the thickest and stiffest part of the anulus—usually the last part of the disc to degenerate (20).

Any disadvantage to flattened posture based on increased intradiscal pressure can be minimized by realizing that this pressure increase is only noted at low loads of compression where little likelihood exists of mechanical damage because the forces are less than those required to cause disc failure. Flexed postures increase the compressive strength of the lumbar spine (20). People who squat or sit with their spines in a flexed posture have less disc disease and degeneration, making it unlikely that the flexed sitting posture could be damaging to the disc (59).

Lordosis or extension, coupled with the compressive forces of loading the lumbar spine, produces high stresses on the apophyseal facet joints.

Bartelink (60) finds that the increased intra-abdominal pressure with flexion protects the lumbar spine against high compressive loads. Another advantage of flattening the lumbar lordosis during distraction is that the increased intra-abdominal pressure protects the spine from compressive loading forces.

Lumbar Lordosis in Chronic Low Back Pain Patients

Christie et al. (61) reported that, in standing, patients with low back pain show increased lumbar lordosis compared with controls. If extension or lordosis were the perfect neutral posture, pain would be relieved by it.

Schnebel et al. (47) point out that extension reduced the compressive force and tension on the nerve root, whereas flexion increased the tension. Brieg's work was cited as showing this phenomenon. I feel that extension is of benefit after a disc protrusion is reduced. In using distraction adjusting, a balance must be reached between adding nerve root compression with flexion (thus the need to limit flexion motion in disc herniation cases) and causing increased disc protrusion by extending the lumbar spine. Tolerance testing of the patient prior to distraction adjusting is mandatory to prevent iatrogenesis in the sciatica patient.

It is important to study these concepts of lifting and intradiscal pressure changes within the disc because we do place the lumbar spine into a flattened and often a flexed posture in axial distraction of spines with herniated discs as well as in those with spinal stenosis caused by degenerative disc disease, degenerative spondylolisthesis, true spondylolisthesis, facet syndrome, and scoliosis. The research cited above assures us that, along with tolerance testing performed prior to distraction adjustment of the lumbar spine, we are within safe parameters of spine tolerance.

BIOMECHANICS OF FLEXION-DISTRACTION ADJUSTMENT

Principles of Axial Flexion-Distraction Adjustments

Onel et al. (24) reported that 78.5% of medial, 66.6% of posterolateral, and 57% of lateral herniated discs retracted under 45 kg of distraction in 28 of 30 patients studied.

Figure 9.7 shows the patient lying in the CT scanner with pelvic traction applied to the lumbar spine, which is in a flat lumbar curve. A CT scan is made of the patient's spine before and during distraction. Results of these tests were reported on 10 patients.

Figure 9.8 shows 1 of the 10 patients in Onel et al.'s study prior to distraction being administered. Note the left lateral disc herniation creating stenosis within the lateral recess and osseoligamentous canal. Also note that the facet joints are imbricated causing narrowing of the nerve canals bilaterally (*see curved arrow*).

Figure 9.7. Shown here is positioning of the patient in the gantry of the computed tomography (CT) scanner during the CT investigation before and during distraction administration. The pelvic belt is attached to a traction device with flexion of the hips and knees to ensure lumbar lordosis flattening during distraction. (Reprinted with permission from Onel D, Tuzlaci M, Sari H, et al. Computed tomographic investigation of the effect of traction on lumbar disc herniation. Spine 1989;14(1):82–90. Copyright 1989, Lippincott-Raven.)

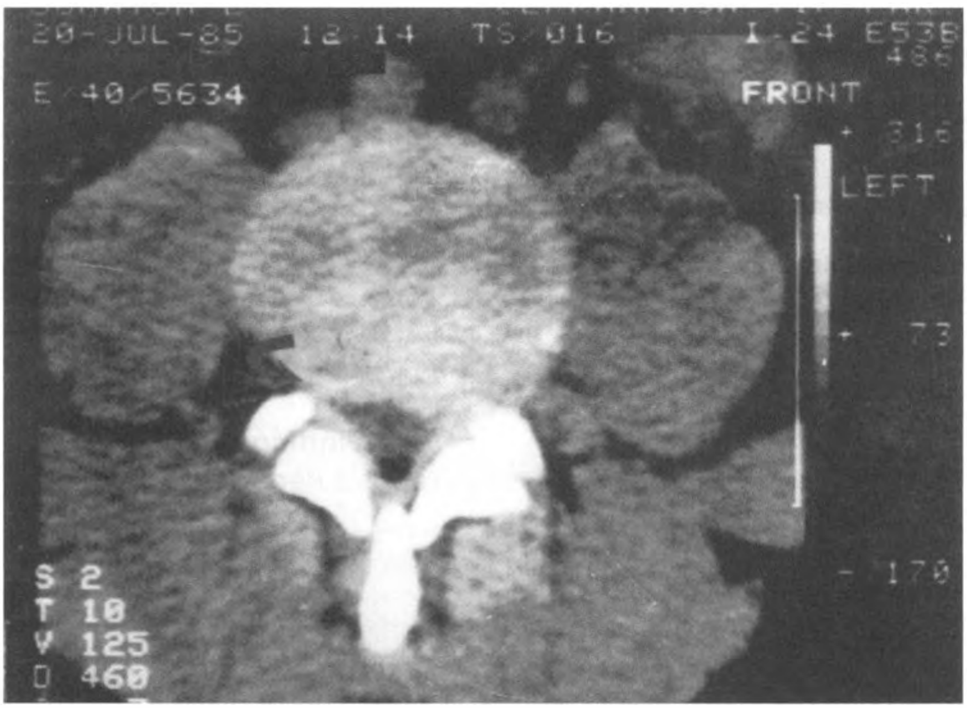


Figure 9.8. Here is the computed tomography scan prior to distraction, showing the medial disc prolapse and left lateral prolapsus at L4-L5 accompanied by invasion of the neural foramen by herniated nuclear material (*HNP*). This author would also note the sagittal narrowing of the osseoligamentous canals by facet imbrication subluxation, which contributes to lateral recess and foraminal stenosis. (Reprinted with permission from Onel D, Tuzlaci M, Sari H, et al. Computed tomographic investigation of the effect of traction on lumbar disc herniation. Spine 1989;14(1):82–90. Copyright 1989, Lippincott-Raven.)

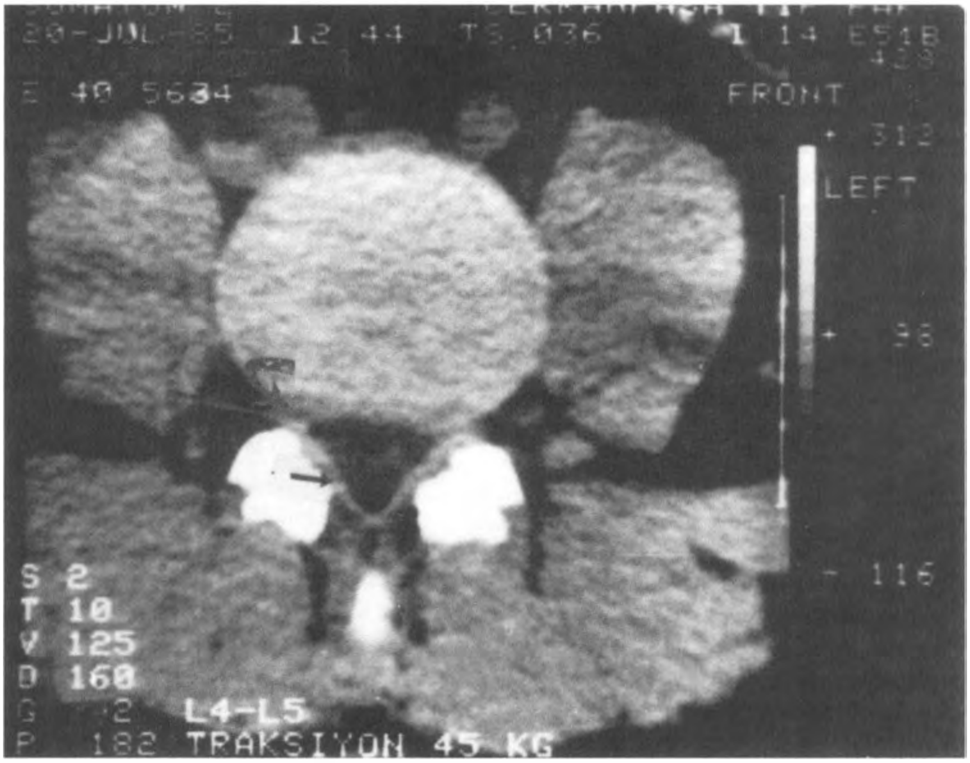


Figure 9.9. Here is the computed tomography scan during distraction using 45 kg of force. Regression of the herniated nuclear material (*HNP*) from the discal space and withdrawal from the neural foramina is seen. I would add that the osseoligamentous canals show increased sagittal diameter during distraction, thus further reduction of canal stenosis. (Reprinted with permission from Onel D, Tuzlaci M, Sari H, et al. Computed tomographic investigation of the effect of traction on lumbar disc herniation. Spine 1989;14(1): 82–90. Copyright 1989, Lippincott-Raven.)

Figure 9.9 is the CT scan of the same patient in Figure 9.8 during application of 45 kg of distraction force. Note the reduction of the disc herniation and opening of the lateral recesses as the facet joints are distracted in an axial plane (*curved arrow*). Also note the ligamentum flavum is tautened to afford a greater sagittal diameter of the spinal canal (*straight arrow*).

Onel et al. (24) state the following about distraction of the lumbar discs:

1. Static lumbar traction opens the disc and apophyseal joint spaces, reduces the herniated nucleus pulposus, and opens the anatomic structures of the lumbar spine.
2. The widened disc space causes intradiscal pressure to drop and probably creates a negative intradiscal pressure that draws the herniated disc material back into place.
3. The anterior and posterior ligaments are stretched under distraction. The posterior longitudinal ligament is stretched, and it may "push back" the herniated disc toward the disc space. Therefore, the herniated nucleus pulposus is reduced by the combination of the lowered intradiscal pressure drawing the nucleus pulposus back into the disc space and retraction of the posterior longitudinal ligament pushing the disc back.
4. The interspinous spaces are seen to increase during distraction with the ligamentum flavum becoming thinner. It is felt that facet joints separate and the posterior longitudinal ligament stretches.

Onel's work stimulates clinical confidence as I apply distraction adjustments to my patients. The knowledge that the spinal vertebral and osseoligamentous canals are opened to relieve stenosis and nerve root compression is a positive concept for chiropractic adjustment.

Other Findings on Disc and Stenosis Reduction

Komori et al. (62) recently showed reduction of an extraforaminal disc herniation following conservative care (Fig. 9.10). They feel migrating fragments of disc have the greatest tendency to disappear, whereas protruded discs show little change on follow-up MRI. This case is shown as an example that disc herniation can and does reduce under conservative care, which includes procedures described in this text.

A colleague sent me a report of a patient with an L5–S1 large left paracentral disc herniation. The patient had left first sacral dermatome pain with an absent ankle reflex and plantar weakness of the foot at the ankle. The patient had been

told to have surgery by both a chiropractor and the surgeon to whom the chiropractor had referred him; he was told surgery was the only option. Distraction adjusting, however, totally relieved the patient of both objective and subjective symptoms and signs. This case draws attention to facts about disc herniations and their clinical presentation, which are discussed below.

Benefits of Distraction Manipulation

Autotraction, a treatment for low back syndrome of benign cause, uses a specially designed traction table divided into two movable sections. While lying on the table, the pelvis is secured and the patient controls the traction forces by grasping and pulling the bars at the head of the table. Figure 9.11 shows how the traction forces are applied.

In comparing autotraction with conventional passive traction, auto traction showed a 75% favorable response (30 of 40 patients) versus 22% (6 of 27 patients) for passive traction. After 3 months, 19 of the 30 responders to auto traction (63%) reported continued improvement. In these patients, pain ratings remained stable and the disability scores decreased to 0 to 23% of the pretreatment level.

Response to autotraction did not seem to be caused by a placebo effect. In some cases, normalization of objective neurologic signs did accompany pain relief. The success rate of passive traction (22%) was much below the 35 to 55% rate of success that has been attributed to placebo treatments for pain (63).

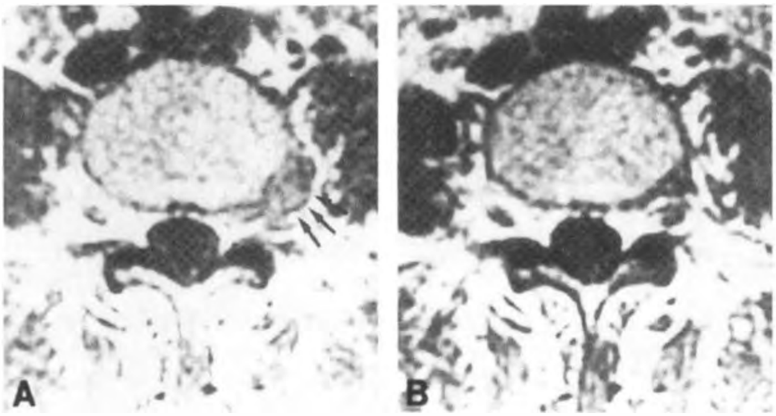


Figure 9.10. T1-weighted axial views of a 54-year-old woman's L4–L5 disc. **A.** March 29, 1993. **B.** August 30, 1993. The patient was suffering from severe anterior lower leg pain. Extraforaminal disc herniation (*black arrows*) was observed at L4–L5 disc in the initial magnetic resonance imaging (*MRI*) examination (**A**). Conservative measures including L4 radicular block resulted in failure, and operative treatment was planned. Remarkable improvement of her symptoms occurred after L4–L5 discography; thus, operative treatment was canceled and conservative treatment was continued. In the follow-up MRI (**B**), the herniated mass showed marked decrease in size, and the left L4 dorsal root ganglion (*DRG*) was easily recognizable. (Reprinted with permission from Komori H. The natural history of herinated nucleus pulposus with radiculopathy. Spine 1996;21(2):225.)

AUTOTRACTION VS PASSIVE LUMBAR TRACTION, Tesio

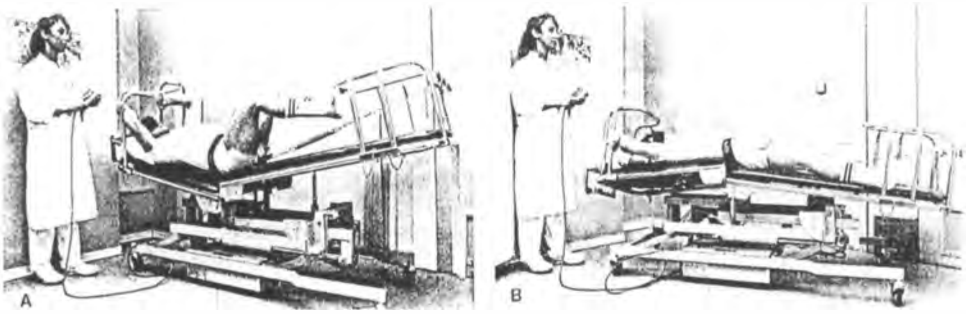


Figure 9.11. Autotraction treatment for low back pain. Patient pulls with the upper limbs while lying on a specially designed traction table. The treatment starts with the patient in the least painful position (**A**) and the goal is to reach, painlessly, the former painful position(s) (**B**). (Reprinted with permission of Tesio L, Merlo A. Autotraction versus passive traction: an open controlled study in lumbar disc herniation. Arch Phys Med Rehabil 1993;(Aug.):871–876. Copyright 1993, WB Saunders.)

Intermittent Distraction

Intermittent traction appears to be associated with less post-traction discomfort (15%) than does static traction (30%), and it likely produces intervertebral joint distraction equivalent to that of static traction (64).

Centralization Phenomenon

The centralization phenomenon is when the most distal symptom is relocated to a more proximal (i.e., more central) location (65). In clinical practice, Cox flexion-distraction practitioners scrutinize this phenomenon as care progresses. It signifies lessening of nerve root irritation by mechanical or chemical factors, and it is a sign of healing and improvement. The opposite effect, or lateralization and extension of pain into the extremity, or worsening of the extremity pain compared with the spine pain, is a morbid sign of increasing nerve root irritation by chemical or mechanical cause.

NO CORRELATION BETWEEN SIZE AND SYMPTOMS OF DISC HERNIATION

Modic et al. (66) find no correlation of pain and disability with disc size, behavior, or type. Matsubara et al. (67), reporting on 32 conservatively treated herniated lumbar disc patients, found repeated MRI studies done acute, 6, and 12 months later to show the following reductions of the cross-sectional area of the spinal canal and size of the herniation: The original MRI showed the spinal canal occupied by the herniated disc to be 32%, 29% 6 months later, and 25% 1 year later. The size of the disc herniation was decreased 20% in 34% of patients, 10 to 20% in 28% of the patients, and unchanged in 38% of patients at 1 year. *Symptoms and signs did not correlate with the degree of reduction of the disc herniation.*

Imaging Does Not Determine Treatment

Gonski (68) reports that imaging does not decide treatment. Clinical findings, not MRI evidence of disc herniation determine whether surgery is indicated.

Boos et al. (69) found 76% of asymptomatic patients showed a disc herniation in the lumbar spine on MRI examination, whereas 96% of patients with low back and/or sciatic pain showed MRI evidence of a disc herniation. The significant fact about disc herniation is whether it chemically or mechanically irritates the nerve root or dorsal root ganglion. If it does, sciatica is possible; if it does not, sciatica is not present.

Disc and Stenosis Reduction on Distraction

Decompression of the foraminal space was statistically significant after 5 to 10 mm of distraction (70). Quellette (71) states that distraction at one half to two thirds of body weight reduces herniated discs. He states distraction allows the vertebrae to separate, thus creating a negative pressure in the intervertebral joint. The nucleus, which is infiltrated in the fissure, is drawn by suction into its proper place. Stephens and O'Brien (72) reported a 20% increase in the cross-sectional area of the lumbar intervertebral foramina with distraction. Awad (73) reported that distraction widened the L1–L2 posterior intervertebral disc space 1.1 mm, L2–L3 2.0 mm, L3–L4 2.8 mm, L4–L5 2.3 mm, and L5–S1 0.8 mm. Gillstrom et al. (74) found good clinical results after autotraction in 25 patients with lumbar and sciatic pain.

Teplick and Haskin (75) encouraged the study of results of disc herniation reduction without surgery after seeing 11 patients with confirmed herniated lumbar discs get well without surgery.

Weisel et al. (76) reported that, in a blinded study, three neuroradiologists found 36% of 52 asymptomatic patients had disc disease, including herniation, on CT scan. The question is raised: How much disc protrusion is needed to create symptoms, or more importantly, *how much reduction of the disc protrusion is needed to relieve leg pain symptoms even though the disc protrusion remains* (74–76).

Cox and Aspegren (77) reported a case of intervertebral disc herniation that was reduced by 14% (a measurement introduced in the paper to determine discal reduction) with complete relief of the low back and sciatic pain.

Disc Herniation Treatment

Kessler (78) defines the treatment of acute stage disc herniation to be low intradiscal pressure and specific segmental distraction. Burton (22) reported 74% success using chest harness gravity traction for disc herniation patients. Burton found that rarely is the disc completely returned to its normal intervertebral location, but it is reduced sufficiently to decompress the nerve root and allow healing of the anular tear (Fig. 9.6).

Possible Mechanism of Distraction Benefits

Cyriax (79) states that as the vertebrae in the spine are distracted, a negative pressure develops in the disc and sucks back a protrusion. Cailliett (80) ascribes the effects of distraction to flattening of the lumbar lordosis. Wyke (81) suggests that the stretch imposed by traction influences the mechanoreceptors of the disc, ligaments, and apophyseal joints.

Increased Disc Space Is the Goal of Distraction

DeSeze and Levernieux (82) reported a distraction of 1.5 mm. per disc space after lumbar traction, and Mathews and Yates (83) reported a vertebral distraction of 2 mm per disc space after traction.

A paper translated from Chinese, "Treatment of Lumbar Disc Protrusion by Automatic Chiropractic Traction Instrument," reports 73% of 400 lumbar disc protrusion cases were completely cured of pain.

Neugebauer (84) reported 99% relief in treating 30,000 patients with disc protrusion over a 14-year period. He felt that distraction allowed the disc to be reduced, the intervertebral foramen to be increased in size to allow the nerve root to escape compression, and the posterior longitudinal ligament stretched to bring the disc back to its normal position.

Tien-You (85) states that manipulation is the key to the treatment of patients with protruding disc, and that a specific feature of the nucleus pulposus is the strong elasticity of the disc. This elasticity is used during manipulative reduction to change the shape of the space between the affected vertebrae and to produce a retractile force by which the prolapsed nucleus is pulled back to its original position.

Kramer (86) discusses the most important factor in traction, the reduction of intradiscal pressure, which facilitates normalization of dislocated disc fragments. Postimaging studies may show no change in the disc protrusion, but apparently only a few millimeters of pressure decrease can change the displaced disc fragment and the pain receptors.

Canal Size Difference Determines Pain

A 2-mm difference in canal size is all that is needed to determine whether a person will have back pain. Such narrowing of the vertebral canal can be caused by stenosis from disc protrusion, facet hypertrophy, ligamentum flavum hypertrophy, or a combination of such forces (87). Yefu et al. (88) documented 1455 cases of lumbar disc protrusion that were reduced by traction and manipulative reduction.

Double Crush Syndrome Treated with Distraction Adjustment

A 63-year-old man suffered from right anterior leg numbness and recurrent lower back pain for 36 months. A clinical diagnosis of double crush syndrome was made after appropriate testing. The patient was diagnosed with nerve root compression at the right L4 and L5 levels and infrapatellar saphenous nerve compression at the deep fascia just below the tibial tuberosity.

Lumbar flexion-distraction protocol was employed for a 6-week treatment plan with two visits per week. Follow-up at 6, 8, 12, 26, and 52 weeks revealed complete resolution of the right anterior leg numbness and reduced occurrence of low back pain (89).

TREATMENT OF PELVIC DYSFUNCTION WITH FLEXION-DISTRACTION MANIPULATION

Mechanically induced pelvic pain and organic dysfunction syndrome is characterized by various disturbances in pelvic organ function, and it has been successfully managed by chiropractic manipulative procedures. Treatment protocols outlining the application of distractive decompressive manipulation of the lumbar spine in the management of these cases have been developed. Their incorporation requires the identification of patients who present with symptoms of bladder, bowel, gynecologic, and sexual dysfunction secondary to the impairment of lower sacral nerve root function as a result of a mechanical disorder of the low back. It is not within the sphere of this paper to cover this important topic (90).

Chronic pelvic pain is noncyclic pain in the pelvis that has persisted for 3 months or longer. Palmer College Center for Chiropractic Research reported on 19 female patients with chronic pelvic pain treated with flexion-distraction manipulation (personal communication). This report showed positive short-term effects on symptomatology, disability, and chronic pelvic pain with 50% reduced analgesic use, decreased pain intensity or complete remission of pain, feelings of a "better mood," decreased menstrual cramping, and pain-free coitus.

MECHANORECEPTOR ACTIVATION WITH AXIAL DISTRACTION ADJUSTING

An exciting aspect of chiropractic adjusting is the firing of normal spinal reflexes from muscles, synovial capsules, disc, and ligaments to the spinal cord and dorsal root ganglion that occurs with the adjustment. These impulses are found to create normoexcitatory reflexes that inhibit hyperexcitatory impulses that generate pain. A discussion of the literature on this concept follows.

Mechanoreceptors of the Joint Capsule

All the synovial joints of the body (including the apophyseal joints of the vertebral column) have four types of receptor nerve endings.

Type I mechanoreceptors consist of clusters of thinly encapsulated globular corpuscles embedded in the outer layers of the fibrous joint capsule. They have a low threshold and respond to small increments of tension; some in each joint are of such low threshold that they fire continuously even when the joints are immobile. Type I receptors, therefore, function as static and dynamic articular mechanoreceptors.

Type II mechanoreceptors are embedded in the deep layers of the fibrous joint capsule, abutting the subsynovial tissue. They are activated by joint motion and behave exclusively as dynamic (or acceleration) mechanoreceptors.

Type III mechanoreceptors are larger, thinly encapsulated corpuscles on the surfaces of joint ligaments, but they are absent from the ligaments of the vertebral column. They respond only to high tension in joint ligaments, which is usually by powerful joint manipulation or the application of high traction forces.

Type IV mechanoreceptors lie in the fibrous capsule of joints, evoking pain when irritated. Mechanical or chemical irritants provoke this unmyelinated system. They are not present in synovial tissue, intra-articular menisci, and articular cartilage (91).

The facet capsule but not the ligamentum flavum is substantially innervated by sensory and autonomic nerve fibers, and it has a structural basis for pain perception (92).

Joint Nerve Supply and Its Irritative Factors

Joints are supplied by articular nerves containing myelinated and unmyelinated sensory afferent fibers and unmyelinated efferent sympathetic postganglionic fibers. Joint effusion and

edema stress afferent receptive fibers along with chemical inflammation, which releases a number of pain mediators (prostaglandins, thromboxanes, leukotrienes, kinins, and others). Articular pressure increases more in diseased joints than in normal joints with the same volume of fluid present in each.

Substance P affects articular vasculature and the cells involved in the inflammatory response. Injections of substance P into the synovial cavity of rat knee joints were shown to evoke plasma extravasation. Substance P can increase the production of prostaglandins (93).

Type II Mechanoreceptors Most Common in Facet Capsule

Chiropractic Adjustment Effects

McLain (94) found predominantly type II mechanoreceptors in the cervical facet capsules, proving that these tissues are monitored by the central nervous system and implying that neural input from the facets is important to proprioception and pain sensation in the cervical spine. Previous studies have suggested that protective muscular reflexes modulated by these types of mechanoreceptors are important in preventing joint instability and degeneration.

Avramov et al. (95) hypothesized that capsular stretching of the facet joint may activate mechanoreceptors and nociceptors to, in turn, activate low and high threshold sensory fibers. This may initiate the facet joint syndrome as we term the facet subluxation and its neurologic bed when irritated.

SPINAL ADJUSTMENTS NORMALIZE AFFERENT PATHWAYS AND STOP PAIN

Patterson and Steinmetz (96) found long-lasting changes occurred in spinal reflex pathway excitability with short periods (15 to 30 minutes) of intense afferent input to the spinal reflex (spinal fixation) and this fixation can cause several hours of neural excitability. Patterson states that manipulative therapy, which tends to restore free motion to the spinal joints, reduces muscle spasm and decreases abnormal and overactive afferent input, which should allow the affected reflex paths to regain more appropriate excitability levels. Therefore, spinal reflexes should be considered as actively participating in the signs and symptoms treated with manipulative therapy and their role should be recognized in the treatment. Adjustments seem to be an effective way to decrease the hyperexcitable central state that leads to further alterations in spinal function (97).

Arthritic joints contain six times more nociceptors for pain reception than normal joints, and they can be stimulated by stress, prior irritants, or exertion. If this stimulation continues for 20 minutes, *spinal fixation* or permanent painful reflex can result. This is a learned reflex of pain (97).

Pickar and McLain (98) found that manipulation of adult cat lumbar facet articulations stimulated afferent nerve pathways with receptive endings located in or near to the tissues of the facet and endings located in lumbar paraspinal muscles distant from the facet joint capsule. Distraction of the facet activates these sensory receptors more than compression. Stimulation or modulation of this system may explain the beneficial effects many patients receive through spinal manipulation and other therapies. Research is needed to determine the precise role played by activation of these receptors. As we discuss axial distraction adjustments, both manual and motion assisted, facet and disc receptor activation is a principle in the pain relief attained.

Zusman (99) states that passive joint movement relieves chronic articular soft tissue pain by breaking up reflex neurologic pain created by adhesion, loss of joint space, and muscle spasm. Passive joint movement creates normal neurologic reflexes that break up the abnormal hyperexcitatory ones. Zusman et al. (100) also find that passive joint movement relieves spinal pain by arousing to clinically effective levels a pain control system encoded by opioid peptides to statistically significant treatment levels.

Dorsal Root Ganglion Sensitivity

Figure 9.12 describes the anatomic location of the dorsal root ganglion schematically and on MRI (94, 101). Note the location of the DRG below the pedicle of the vertebra, lying within the osseoligamentous canal. The dimension of the DRG gradually increases from L1 to S1 with the S1 DRG the largest and located most intraspinally. It is felt that S1 radiculopathy may involve the nerve root and DRG as a result of disc herniation or degenerative changes of the L5–S1 facet. The increased incidence of disc degeneration and intervertebral narrowing in the lower lumbar region, coupled with the larger DRGs in the lower lumbar spine, may explain the susceptibility to compression (101).

The size and location of the lumbar and S1 DRGs are as follows:

Level	Size	Location
L1	3.7 mm × 4.3 mm	92% in the lumbar intervertebral foramen
L2	4.6 mm × 5.7 mm	98% in the lumbar intervertebral foramen
L3	5.7 mm × 7.1 mm	100% in the lumbar intervertebral foramen
L4	6.2 mm × 8.4 mm	100% in the lumbar intervertebral foramen
L5	5.9 mm × 9.4 mm	95% in the lumbar intervertebral foramen
S1	6.2 mm × 11.2 mm	79% in the intraspinal region

Nerve roots occupy 23 to 30% of the area of the intervertebral foramen. It is suggested that the DRG has less space as degeneration of the spine occurs in older people (101). It seems obvious that the lower lumbar spine is vulnerable to stenosis and compression of nerve root and DRG as degenerative changes occur in the spine.

The dorsal root ganglion is a source of afferent impulses that contribute a tonic, low level spontaneous background dis-

charge capable of firing with low level irritation. The straight leg raise pain may be the result of such irritation. Slight DRG pressure increases impulse firing. In anesthetized animals, the DRG is found to fire impulses with no central or peripheral axon stimulation, suggesting a pain-producing generator (102). At 100 mg of VonFrey hair force, a minor compression force, the DRG produces repetitive firing of impulses. Greater pressure produces more than 5 minutes of repetitive firing, but repeated compression damaged tissue resulting in inactivity after 5 or 10 such irritations. Tapping the DRG results in several minutes of firing of impulses (103).

Low Level Irritation of the DRG Activates A and C Fibers

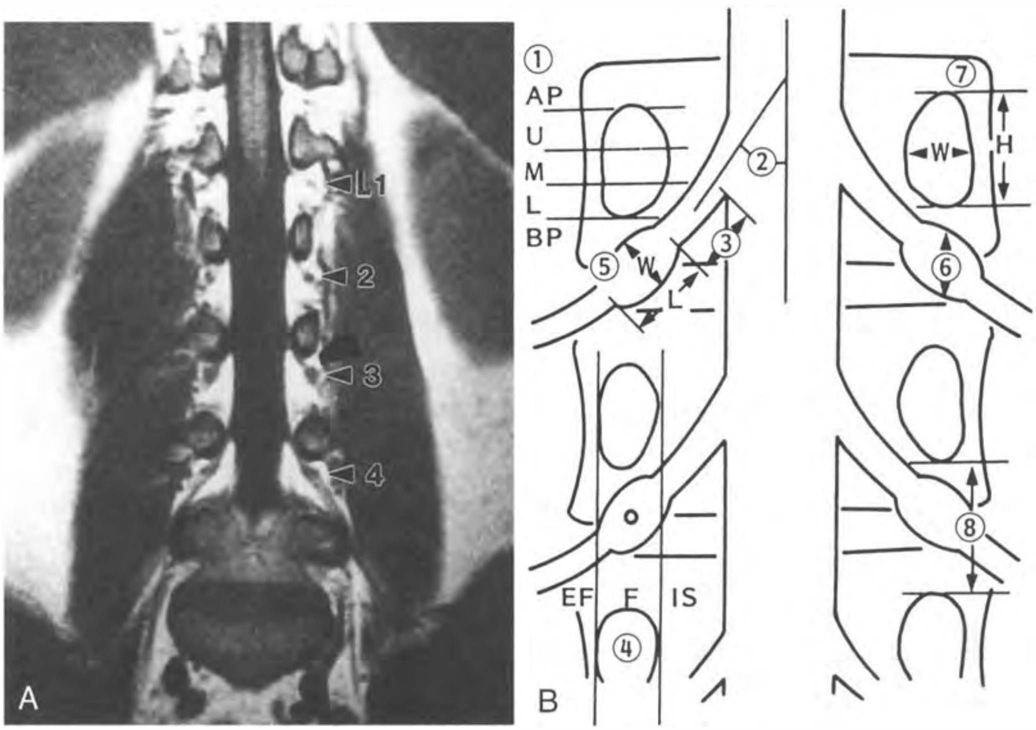
Activation of A and C fibers within the DRG occurs at low levels of irritation, and this response can last for a short duration, and it can become fixated if time frames of 20 to 30 minutes of irritation are applied. Hypertension and tachycardia have been seen in C fiber activation (104).

Radicular pain results from irritation of the DRG by an intervertebral disc herniation (105). The lumbar DRG can be trapped easily between a herniated disc and the facet. Small and repeated movements of the joint traumatize the DRG, resulting in impulse propagation of pain. Autopsy shows the DRG is compressed and distorted by disc herniation (105).

DRGs Compressed by Superior Facet

Kikuchi et al. (106) found the DRG indented by the superior facet at the intervertebral foramen in 71% of anatomic and radiographic studies.

Macnab (107) showed that repeated irritation of the nerve makes it sensitive to pain production. In comparing the nerve with skin, he found normal skin is not painful if touched, but sunburned skin is painful. A nerve is analogous to this. If a normal nerve is pressed on it is not painful, but if it has been irritated by either chemical or mechanical stimulus, it is produces pain with little irritation. Rydevik et al. (108) showed that compression of a normal nerve can be associated with numbness and motor weakness, but it does not usually cause pain. However, if the nerve tissue is chronically irritated, mechanical deformation may induce radiating pain. This intraneural inflammation seems to be a factor of importance in the pathogenesis of pain production in nerve root compression syndromes. It is debated whether such intraneural inflamma-



Figures 9.12. A. T1-weighted coronal magnetic resonance image of lumbar nerve roots and dorsal root ganglia (*arrowheads*). **B.** Lumbosacral nerve root and dorsal root ganglion (DRG) parameters. (*1*) Level of the nerve root origin. (*2*) Nerve root sleeve angulation (NRA). (*3*) Length of the nerve root (NRL). (*4*) Position of the DRG. (5) Dimensions of the DRG (DRGW, DRG midpoint width; DRGL, DRG midpoint length). (6) Height of the DRG. (7) Dimensions of the pedicle (W, pedicle midpoint width; H, pedicle midpoint height). (*8*) Height of the intervertebral foramen (FH). *IS*, intra-spinal; *F*, foraminal; *EF*, extraforaminal; *AP*, above the pedicle; *U*, upper third of the pedicle; *M*, middle third of the pedicle; *L*, lower third of the pedicle; *BP*, below the pedicle. (Reprinted with permission of Hasegawa T, Mikawa Y, Watanabe R, et al. Morphometric analysis of the lumbosacral nerve roots and dorsal toot ganglia by magnetic resonance imaging. Spine 1996;21(9):1005–1009. Copyright 1996, Lippincott-Raven.)

tion is the result of an inflammatogenic effect of nucleus pulposus on nerve tissue (chemical radiculitis) or is an effect of mechanical nerve root deformation by the herniated disc.

The dorsal root ganglion is a pain source, as cited above, and it can be stimulated to produce pain-relieving afferent impulses with passive range of motion adjustments. These movements are produced with automated axial distraction adjustments and the clinical advantages of pain relief result.

LIGAMENT LOADS IN AXIAL FLEXION-DISTRACTION ADJUSTMENTS

Gudavalli and Triano (109) quantified the ligament and disc loads on the lumbar spine during combined traction and flexion loading and reported that both increase the loads on the posterior ligaments. A computer modeled the posterior ligaments, and vertebral bodies, intervertebral disc with the anterior and posterior ligaments, and facet joints with equilibrium conditions were applied to the model. Flexion of the motion segment created a compressive load on the disc and the traction load created a tension load on the disc. The ligaments are loaded well below their failure loads at traction loads under 444 N and 6° of flexion load.

I am concerned that these limits not be exceeded with my technique and that tolerance testing is done on every patient prior to distraction adjustment. Chapter 8, Biomechanics Research on Flexion-Distraction Procedure by Ram Gudavalli, PhD, covers the research of the biomechanics of distraction adjusting.

PROTOCOL DESCRIPTION

Pre-Cox Distraction-Adjustment Procedure

Two classes of patients are candidates for distraction adjusting procedures:

1. The patient with low back pain and lower extremity pain extending to the knee but not beyond.
2. The patient with sciatic radiculopathy, which is diagnostically listed as an intervertebral disc herniation (contained or noncontained) or a spinal stenosis defect cited as causing anterior or posterior element degenerative changes.

INCLUSION AND EXCLUSION CRITERIA FOR DISTRACTION ADJUSTING

Algorithm 9.A outlines the exclusion and selection of patients for administration of distraction adjusting. Conditions not treatable with chiropractic distraction adjustments are excluded by following this screening procedural algorithm. The diagnosis of cauda equina syndrome, fracture, dislocation, neoplasm, infection, metastatic disease, diabetes, arthritides, vascular disease, systemic diseases, and hard or progressive neurologic signs indicative of significant nerve root irritation are diagnosed and appropriately managed, leaving those conditions with probable mechanical cause of pain for treatment with distraction adjustments.

Algorithm 9.B outlines management of patients with radicular low back pain using distraction adjusting, and includes time and visits considered customary to arrive at 50% relief and necessary decision-making if 50% relief is not attained at 2 to 5 weeks of therapy. Options for the patient who attains maximal relief are given.

Algorithm 9.C outlines management of nonradicular low back pain using distraction adjusting, including the time and visit parameters to arrive at 50% improvement and further treatment protocol when 50% relief is not attained within 4 weeks.

Determining Frequency of Distraction Adjustment Care

Treatment with Cox distraction adjustments is structured for frequency of care depending on the severity of the pain. Sciatica patients with great pain and neurologic signs are treated daily, whereas patients with less severe nonsciatic pain are treated from daily or two to three visits weekly, depending on pain and disability.

Fifty percent improvement is a determining factor for decreasing treatment frequency, shown in Algorithms 9.B and 9.C. Patients who fail to show 50% improvement, objectively and subjectively measured, within a month of care undergo further testing (e.g., imaging, electromyography, or other appropriate measures) to determine future treatment options.

Rehabilitation

Rehabilitation of the patient is started at the beginning of treatment depending on pain and weakness limitations.

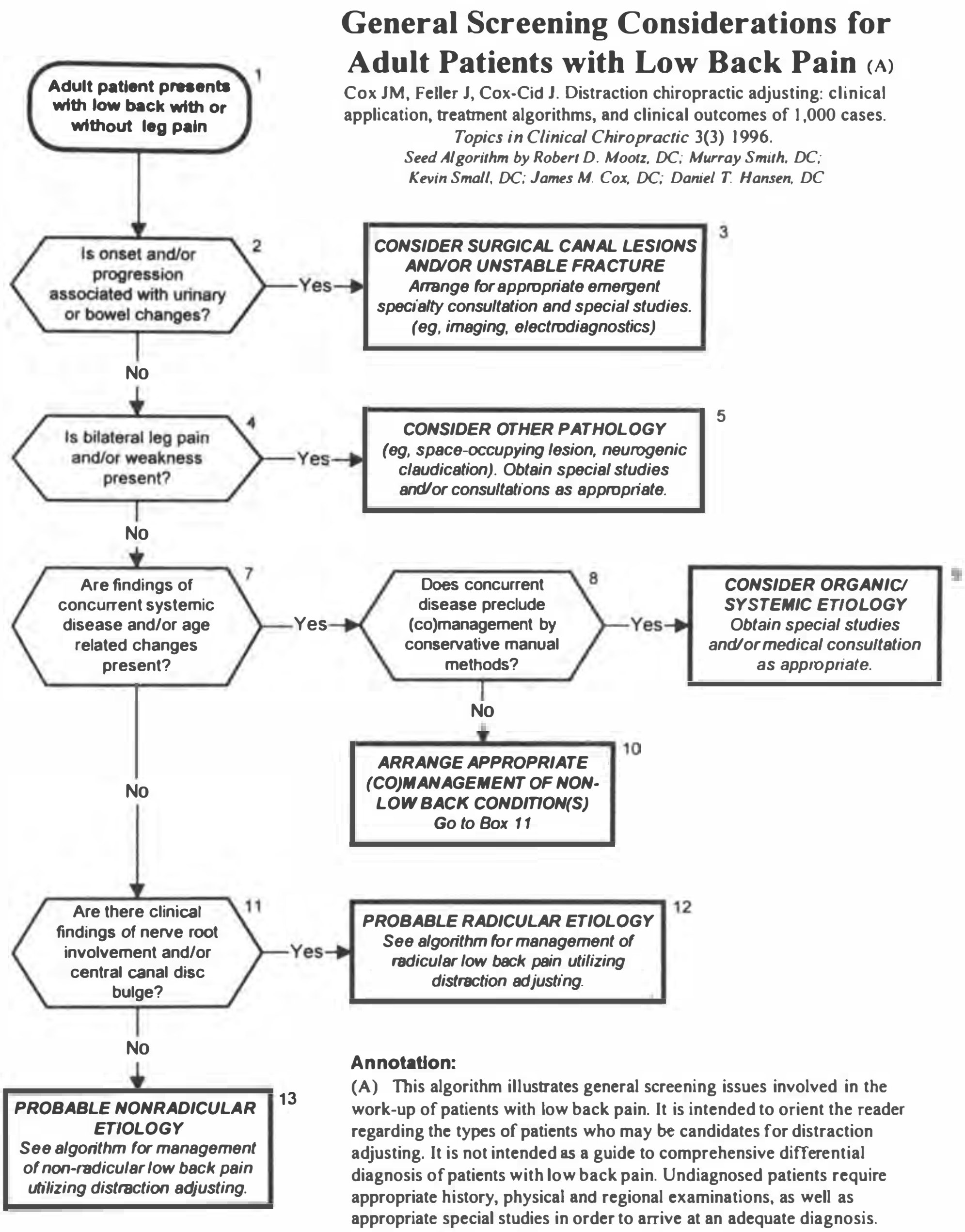
Patient Compliance

A patient who fails to attain 50% improvement within 1 month of care is evaluated for compliance. Patients not following treatment protocols are reminded of the need to do so. Other treatment options (e.g., epidural steroid injection, transcutaneous electrical stimulation, drugs, and so forth) for further conservative care are explained to the patient; this allows patients to be aware of their treatment options. If noncompliant, the patient is given the option of continuing with distraction adjustments for another month. Patients who are compliant, but who do not attain 50% relief in 1 month, are referred for appropriate consultation, imaging and testing, and (co)management.

Patient Treatment Options

Three options are available to patients attaining 50% relief of pain following 1 month of care who fail to attain further relief in the second month:

1. Continuing conservative and rehabilitative care.
2. Seeking other treatment options.
3. Being discharged.

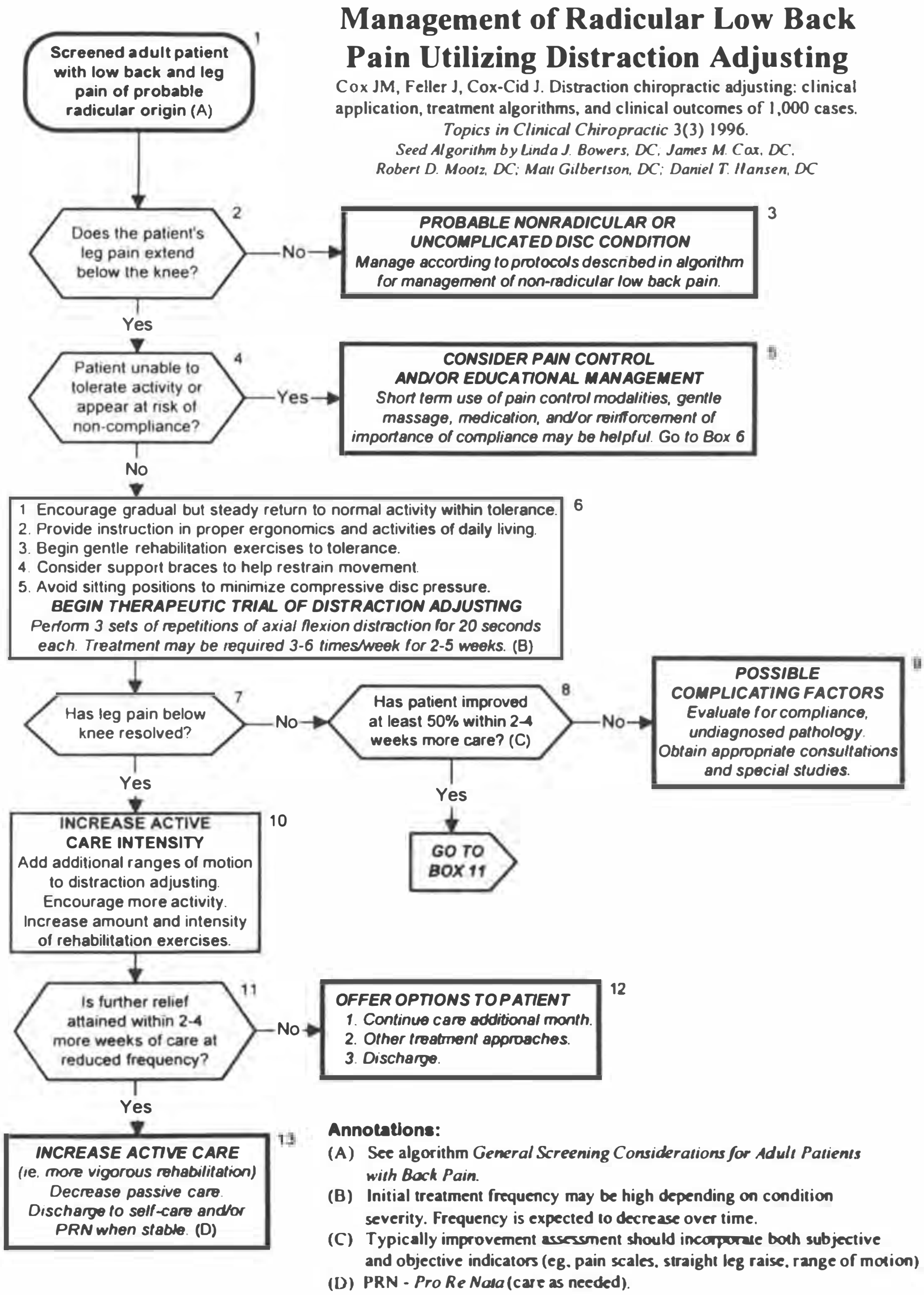


Algorithm 9.A.

The first option of continuing conservative and rehabilitative care is based on the research work of Shvartzman et al. (13) who stated that a patient not responding to the initial trial of conservative therapy should be given the option to undergo continued conservative treatment. He bases this opinion on the fact that the generally accepted protocol for management of an acutely herniated disc is several weeks to months of conservative therapy followed by discectomy in refractory cases. Saal (110) states that a herniated disc with sciatica can be successfully treated with aggressive nonoperative care. He cites 90% success with a 92% return to work rate among 64 patients with signs and symptoms of herniated disc confirmed by either CT or MRI of herniated lumbar disc.

Patients who, at 3 months of care, are still showing objective and subjective improvement of pain and disability are allowed further adjustments and increasingly vigorous rehabilitation therapy *as long as further relief is being attained.* When further relief is not being attained, the patient is presented with the three options listed above.

These patients are evaluated at 2-week intervals with objective and subjective evaluators as described in the annotations of the Algorithms 9.A and 9.B; this allows definitive demonstration of patient relief.



Algorithm 9.B.

Mandatory Imaging

Note in Algorithm 9.A that patients who are seen following trauma; those who have a history of systemic disease, or those with cauda equina or spinal cord compression signs receive clinical testing (e.g., imaging, electromyography, nerve conduction tests, and so on) when first seen. Patients with hard or soft neurologic signs are considered for these tests at the time of first examination, based on the clinician's judgment. However, note that a patient who does not attain 50% relief of pain and disability within 1 month of care is ordered to undergo diagnostic imaging. Testing results determine future treatment, whether care is continued with the present clinician, or a referral is made for proper (co)management. This protects both the doctor and patient from possibly missing an underlying organic disease that might be causing the pain. In today's managed care-dominated chiropractic world, imaging is often discouraged until conservative care fails to show positive clinical relief. A point is reached where good care demands imaging, and the steps required to reach that point are shown in Algorithm A.

DISTRACTION TREATMENT PROTOCOL

The actual application of Cox distraction adjusting will be described as it is taught in the certification course at the National College of Chiropractic. The actual technique is described with acceptable modifications of the doctor's spine contact and differing patient postures for application of Cox distraction adjusting.

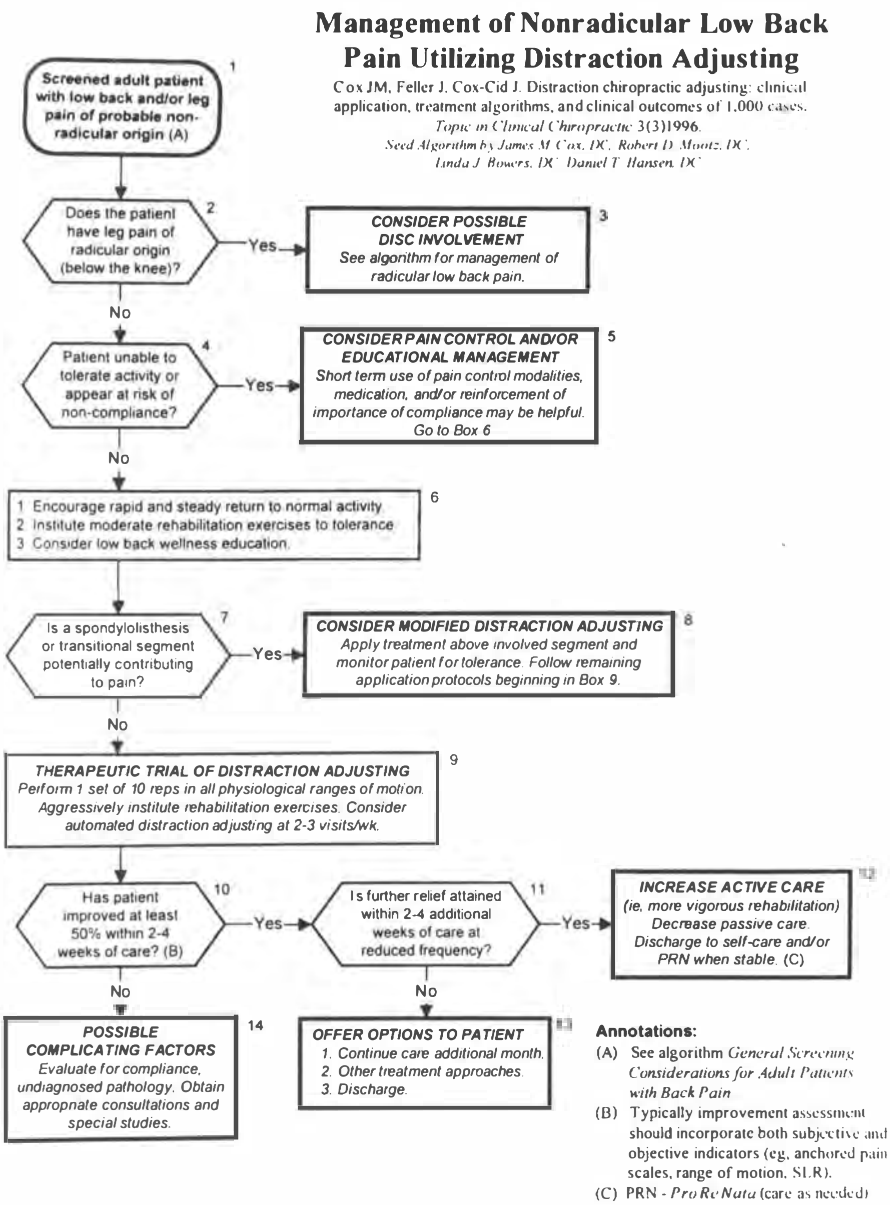
Patient Positioning Sequence

For the patient positioning sequence, follow the steps listed below.

1. Check that all locks on the table are secure and that the table height is satisfactory for easy patient access. If a flexion cushion is to be used in the patient care, as in spondylolisthesis, facet syndrome, or antalgic flexed posture, it is to be in place before the patient lies on the table (Fig. 9.13).
2. Figures 9.14 and 9.15 show how the patient gets on and off the table.

The doctor directs the patient to support his or her body weight with the arms while slowly lowering the torso and lower extremities onto the table. The patient forcefully tightens the abdominal and gluteal muscles to stabilize the lumbar spine during this maneuver.

A lumbar spine support is recommended to aid patients with a lot of pain or those with sciatica caused by herniated lumbar disc lesions in arising from the table. Regardless whether a support is used, the doctor assists the patient to rise while the patient stabilizes the lumbar spine by tightening the



Algorithm 9.C.

abdominal and gluteal muscles and pushing the body up with arm power. The patient slowly slides the lower extremities from the table and assumes the upright posture (Fig. 9.15).

3. Patient positioning is shown in Figure 9.16. A flexion cushion under the abdomen may be needed for patients with marked low back or leg pain to sustain the flexed posture that relieves their pain.

   The patient lies prone with the anterior superior iliac spine resting 2 inches anterior to the caudal edge of the thoracic section of the table. The ankles are positioned so the talotibial articulation rests comfortably on the ankle support. Arms rest on the arm rests (Fig. 9.16).

4. Some patients are in too much pain to lie prone and must be treated while side lying until such time as the prone position is tolerated. In such cases the patient lies on the side with the pelvis on the caudal section of the table, and flexion distraction is administered by using the lateral flexion capabilities of the instrument. This will be shown later. (See Fig. 9.17.)

5. The flexion-distraction lock is released and spring tension is set on the caudal section to provide proper resistance for the patient's body weight. This is done until the caudal section of the table slowly, and with minimal resistance, returns to horizontal from the flexed position.

Tolerance Testing of the Lumbar Motion Segment

Prior to adjusting with distraction procedures, the patient's tolerance to the procedure is checked and documented. Limitations of the type and amount of distraction adjustment is based on Cox (26) and Kramer's (86) testing procedures as follows:

1. If a decrease of pain can be demonstrated under distraction, traction adjusting treatment should be instituted. As a rule, the pain first positively changes its character by centralizing or diminishing. For instance, a lateral pain will be trans-

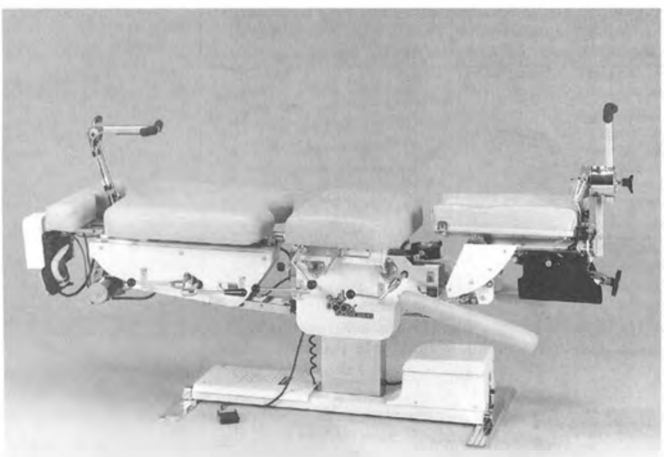


Figure 9.13. Table shown with all locks secure. A flexion roll use is optional.

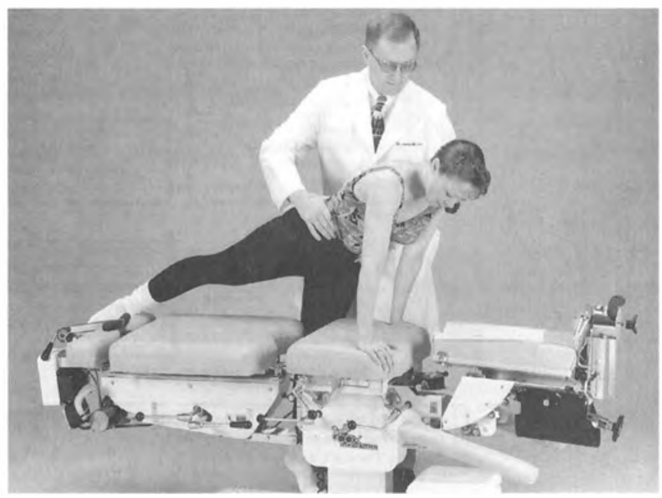


Figure 9.15. Patient assisted from table.

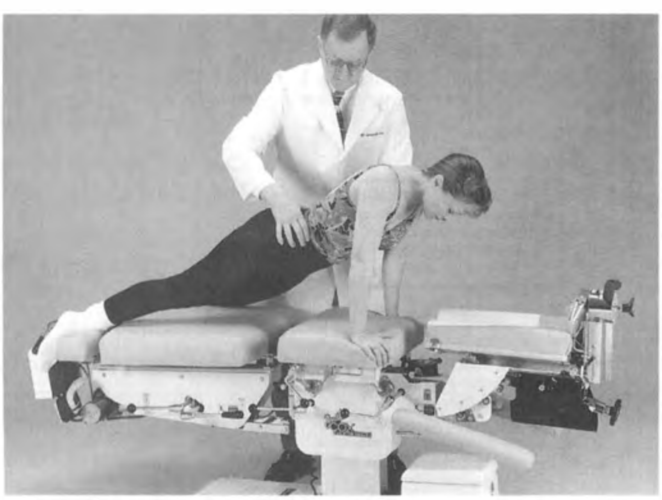


Figure 9.14. Patient assisted onto table.

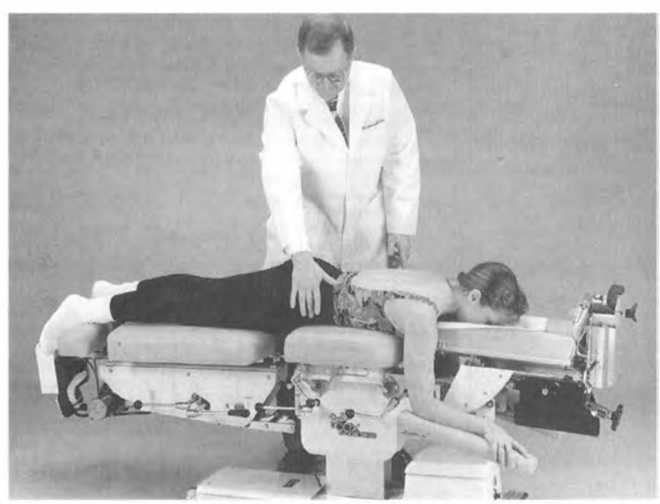


Figure 9.16. Patient positioning on table.

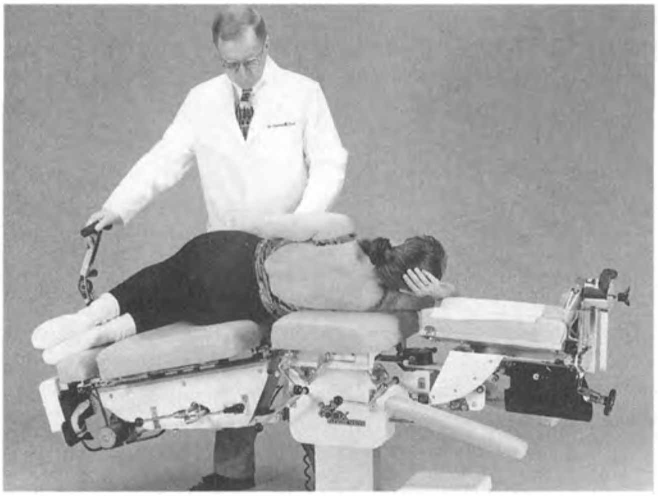


Figure 9.17. Patient lies on side due to pain when lying prone.

ferred centrally, and a sharp lancinating root pain can turn into dull low back pain.

2. Traction is contraindicated when it causes an increase in pain. Reasons for the pain include:

   - Shearing forces influence a displaced fragment and dislocate it completely (pain will always be increased when the prolapse is medial and near the nerve root)
   - A prolapse is still within the boundaries of the vertebral margins, but during traction becomes dislocated into the spinal cord
   - Adhesions are found around the nerve root
   - Adhesions in the spinal canal following surgery

Traction is also contraindicated in patients whose symptoms have increased during a long period of relaxation (e.g., sleeping, during which an "increase of disc volume" occurs). Moreover, traction should not be used in patients with hypermobile segments and muscle insufficiency.

Tolerance Testing of Patient's Ability to Withstand Distraction Movements

1. Release the flexion-extension lock.
2. Central distraction testing is shown in Figure 9.18. Apply specific palmar or thenar contact under the spinous process of the motion segment to be tolerance tested and stabilize the spine. No ankle cuffs are applied. While holding onto the assist (tiller) bar, flex the caudal section of the table to patient tolerance and/or a maximum of 2 inches or until the patient's occiput moves into extension nutation, which corresponds to the 2-inch downward table motion. The doctor can use either the 2 inches of downward movement or the occipital extension as the maximal caudal flexion point with the instrument. The patient's lower extremities act as the tractive force to test tolerance. Hold the contact for 4 seconds, asking the patient if he or she feels any pain in the lower extremity, pelvis, or low back. If no pain is produced, slowly return the caudal section of the table to the horizontal (neutral) position and slowly remove the specific spine contact.
3. Lateral distraction testing: Apply specific palmar or thenar contact under the spinous process of the level to be tested and grasp the lower extremity above the ankle of the uninvolved nonpainful side. Flex the caudal section of the instrument to patient tolerance, a 2-inch maximal downward movement of the caudal section or until the patient's occiput moves into extension nutation, and hold the position for 4 seconds. (See Fig. 9.19.)

   If no buttock, back, or leg pain is produced, slowly return the pelvic section to the neutral, slightly flexed position and slowly remove the specific spinal contact.
4. Grasp the ankle of the involved side and repeat step 3.

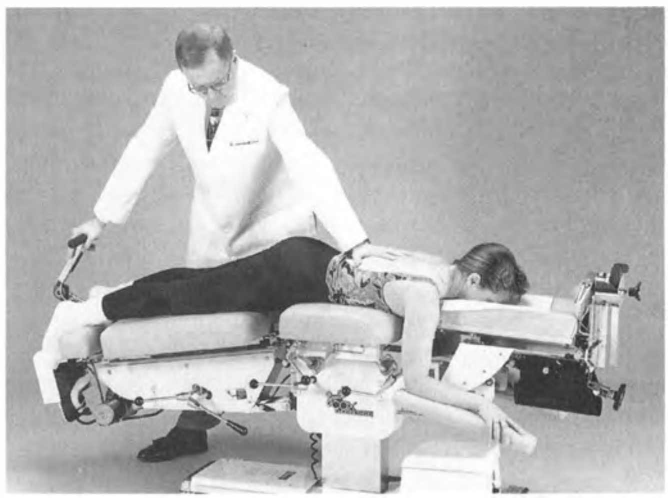


Figure 9.18. Central distraction testing.

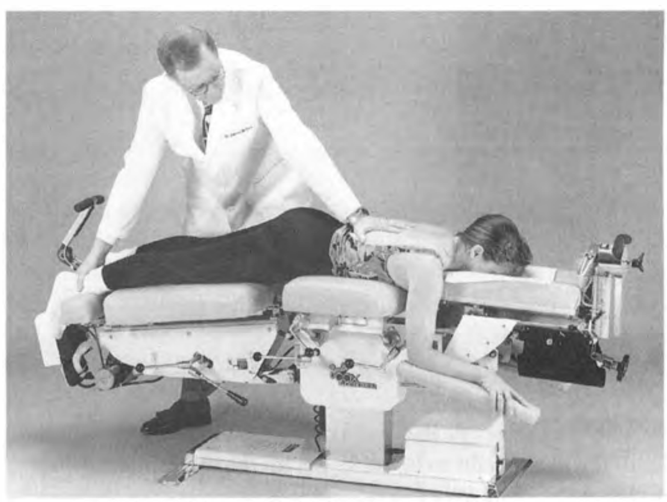


Figure 9.19. Lateral distraction testing.

Spinous process discomfort may be felt by the patient, but this is not a contraindication to application of distraction adjusting. This discomfort is caused by irritation of muscle or the dorsal ramus of the spinal nerve, and the doctor should attempt to apply bilateral paravertebral pressure with the thenar and hypothenar eminences of the hand to cradle the spinous process between these contact points and minimize pressure on it. Note: All segments to be treated are tolerance tested on the initial visit, prior to distraction adjustments being administered.

Procedure After Tolerance Testing

No Pain Felt on Tolerance Testing

Distraction manipulation can be instituted slowly while constantly monitoring patient reaction. The doctor can use either the ankle cuffs for increased traction or the patient's lower extremity weight or ankle as the traction force in applying distraction as shown in Figures 9.18 and 9.19.

Procedure When Pain Is Felt on Tolerance Testing

Always treat the patient with the distraction force below tolerance—that is, if the patient's pain is aggravated when the ankle cuffs are used, but not when the ankle is held as shown in Figure 9.19, then treat as shown in Figure 9.19 until such time as ankle cuff use does *not* cause any pain. If no pain is felt when using the patient's lower extremity weight as the traction force (see Fig. 9.18), but using ankle distraction force (see Fig. 9.19) causes pain, then start with using the patient's lower extremity weight as the tractive force until such time as the ankle distraction force is not painful. If any distraction force causes increased pain, do not use distraction adjustment. Instead, use another treatment approach such as physiologic therapeutics (e.g., positive galvanism, ice, interferential current) until such time as distraction with the patient's lower extremity weight alone does not cause pain. Pain occurs most frequently with distraction adjusting in the acute sciatica patient who is in marked antalgic posture with muscle guarding.

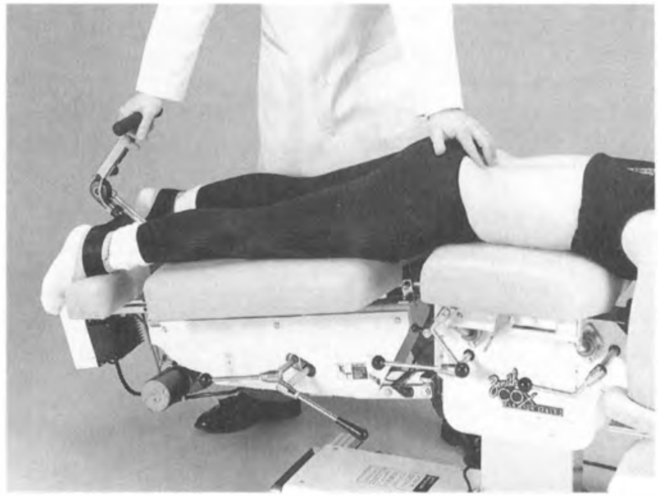


Figure 9.20. Palpatory contact point used to determine tissue tautness between the spinous processes.

Figure 9.21. Neutral starting point for distraction adjusting. The interspinous space is tautened and any further distraction will enter the elastic resistance of the motor segment unit. The neutral starting point, as shown, is attained with or without the cuffs in place.

Palpatory Contact to Increase Local Soft Tissue Tension

In administering distraction adjusting, the doctor must make a contact on the spinous process above the disc or facet articulation that is to be adjusted. Because flexion distraction is the first movement instituted in almost all low back conditions (excluding compression fracture and kyphotic sagittal curves, for example) the treatment position from which distraction flexion is applied is shown and described in Figure 9.20.

1. The doctor's cephalad hand palpates the interspinous space of the motor unit to be treated with the tip of middle finger.
2. Place the tips of the second and fourth digits over the middle region of the paravertebral muscles at the level being treated.

Treatment Position for Distraction Adjusting

1. Depending on patient response to tolerance testing, apply the cuff or use the patient's lower extremity weight as the traction force *or* grasp the patient's ankle.
2. Move the foot piece caudally, if the cuffs are used, until the lower extremities and spine become taut. If more distraction is needed, as with a tall patient, use the caudad crank and begin to slowly separate the caudal section from the thoracic section of the table until the lower extremity and spine become taut.
3. Release the flexion-extension lock.
4. Apply the palpatory contact to increase local soft tissue tension, as shown in Figure 9.20.

5. Use the caudal hand to grasp the assist bar, and slowly flex distract the caudal section of the table until the spinous processes separate and/or muscle tension increases. See Figure 9.21. To realize this taut point, I use the analogy of a rubber band being stretched to its taut point before expanding its length. Note: This last table treatment position of pelvic flexion distraction tautness will be the treatment position for all further table movements when distraction adjustments are administered.

At this point, all tissue relaxation of the lower extremities and spine has been removed so that further distraction application will act on the specific spinal segment below the doctor's spinous process contact. Also note that in treating the spinal segments under distraction, the downward caudal traction force of the table requires equal cephalward force by the doctor's spinous process contact when distraction is applied to a given vertebral segment.

COX DISTRACTION-ADJUSTMENT PROCEDURE PROTOCOL

Protocol I: Patients with True Sciatica

The only distraction adjustment received by a patient with sciatica is flexion distraction. Intermittent distraction is produced by distraction flexion application with the Cox instrument.

Orders

Three 20-second sets of five distractive repetitions per set performed at the level of the diagnosed disc herniation.

1. The patient is positioned as shown in Figures 9.14–9.17.
2. Tolerance testing is performed as shown in Figures 9.18 and 9.19.
3. Find the palpatory contact point and treatment position as shown in Figures 9.20 and 9.21.
4. Apply specific palmar or thenar contact on the spinous process above the disc herniation or stenotic level to be treated. Induce five 4-second distraction flexion movements of the caudal pelvic section, producing a total distraction time of 20 seconds. The limit of downward movement of the caudad pelvic section of the instrument will be 2 inches, patient discomfort, or the beginning of occipital extension nutation. The downward caudal traction force of the table will require equal cephalward force by the doctor's spinous process contact when distraction is applied to a given vertebral segment. See Figure 9.22*A*.

   The doctor's arm is parallel with the patient's spine, not at an angle to it. The thenar pad is in contact with the spinous process to minimize pressure force and thereby pain. The doctor should not hyperextend the wrist to avoid the danger of carpal tunnel stress; therefore maintain a low angle of cephalad lift under the spinous process. The doctor should dissipate pressure over the entire palm and finger surface of the contact hand with emphasis on the thenar contact of the spinous process. This type weight and force distribution minimizes patient awareness of pressure to the spine. Figure 9.22*B* shows the flexion roll in place for a patient whose pain is eased by flattening of lumbar lordosis.
5. After the first 20 seconds of five 4-second repetitions of the interspinous space, return the caudal section of the table to the neutral starting position.
6. Allow the patient at least a 10-second rest period during which time muscle and soft tissue trigger point, acupressure, or other therapy the doctor prefers is applied.
7. Steps 3 through 6 constitute one set. To fulfill the orders cited (three sets of five repetitions), two more sets of steps 3 through 6 are repeated.
8. Note: Securing the table locks between each set can be omitted if the doctor ensures no movement of the caudal section occurs during the rest period.
9. Following the third set, the caudal section is returned to a neutral slightly flexed position and locked in place.
10. Remove the ankle cuffs if they were used in treatment.
11. Assist the patient off the table as shown in Figure 9.15.

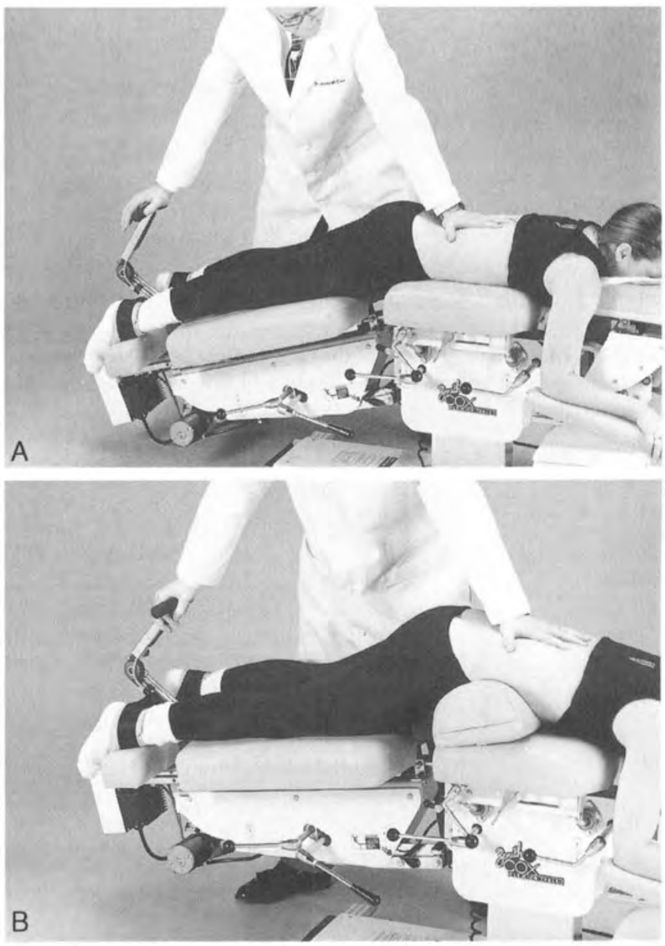


Figure 9.22. **A.** Application of the three 20-second pump distractions without the flexion roll. **B.** Application of the three 20-second pump distractions with the flexion roll in place.

Special Applications of Distraction Adjustment in Disc Herniation Patients

Antalgic Sciatic Lists of the Thoracolumbar Spine

If the patient has a right or left sciatic list, place the caudal section of the table to match the list to aid in relief of pain and treatment efficacy. Figure 9.23 shows the caudal section in right lateral flexion to accommodate a right list of the thoracolumbar spine that is often seen in right medial or left lateral disc herniations.

Patients Whose Pain Does Not Allow Them to Lie Prone

Figure 9.24 shows the side lying posture for the patient who cannot lie prone for distraction adjusting. The doctor stabilizes the spinous processes above and below the disc to be distracted, while using lateral flexion of the caudal section to place the patient's lumbar spine into flexion distraction. See the figure legend for details of this technique.

Pain that does not allow the patient to lie prone for distraction adjustments requires the patient to be placed on the side for adjustment correction. Flexion of the caudal section can also be used to produce a sciatic scoliosis list of the thoracolumbar spine, thus adding to patient comfort. In this case, the patient assumes a right lateral flexion of the thoracolumbar spine by flexing the caudal section of the instrument.

Lateral Flexion Used to Apply Flexion Distraction

With the patient lying on the side *with the pelvis resting on the caudal section of the table,* the doctor uses the lateral flexion movement of the caudal section to produce flexion distraction of the involved disc level in the painful patient who cannot lie prone on the table. The doctor's palpating fingers stabilize the spinous processes above and below the involved disc to accommodate opening of the interspinous processes as lateral flexion is applied. The doctor can hold the tiller bar with one hand and the spinous processes with the other, or my preference is to have both hands contacting the spinous processes, my thigh contacting the caudad table section, and using my caudad arm to move the patient into flexion and return to the neutral position. This allows both of the doctor's hands to control the interspinous movement. See Figure 9.25.

Frequency of Treatment of the Disc Herniation Patient

Daily treatments, sometimes more than once per day, are required for patients in severe pain and for those who are incapacitated. Patients with an acute disc lesion are best kept in the

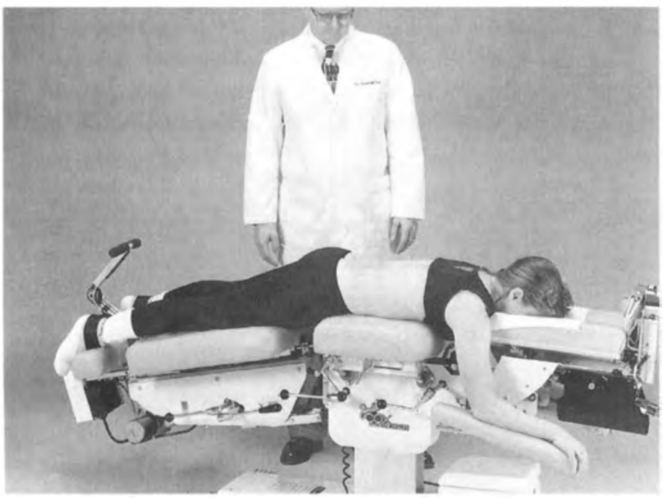


Figure 9.23. Caudal section of the table in right lateral flexion to accommodate and allow relief of pain of a patient who lists to the right side for pain reduction.

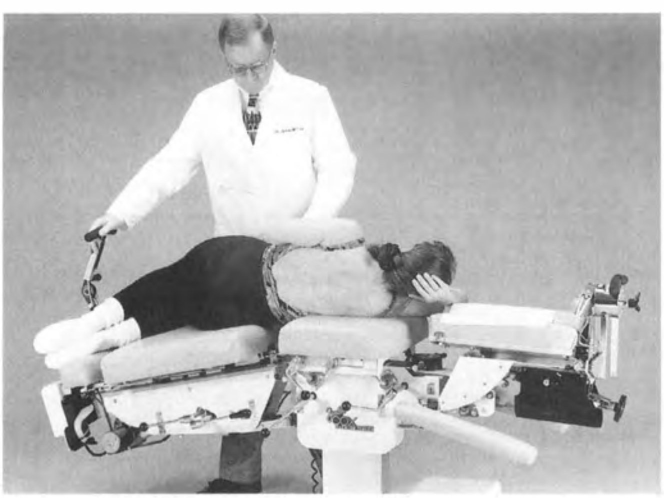


Figure 9.24. Patient lies on the side because pain prevents her from lying prone. Right lateral flexion is also attained by flexing the caudal section of the table.

Figure 9.25. Side lying flexion distraction to treat a painful disc case when pain prevents lying prone.

office throughout the day so that treatment can be given as needed. The patient needs to avoid sitting, which increases the intradiscal pressure and slows healing, even causing relapses. It is better for the patient stay recumbent in the office while undergoing distraction adjusting or therapies intended to reduce inflammation and help the patient avoid bending, lifting, and twisting, than to be driving a car, lifting, or doing other activities that slow or prevent healing.

As the patient attains 50% relief of symptoms, the visits are reduced by 50%. See Algorithms 9.B and 9.C for the reduction of treatment frequency as well as the referral mechanism in treating disc and nondiscal patients. As noted, a patient who does not show 50% relief of disc herniation symptoms, objectively and subjectively, within 3 to 4 weeks of distraction adjusting and care, undergoes imaging and if it is positive, a neurosurgical consultation made.

Three Months for Healing of a Herniated Disc

Herniation of a nucleus pulposus causing nerve compression can heal spontaneously provided low intradiscal pressure can be maintained for 3 months (111, 112).

It is commonly accepted that in the treatment of patients suffering from symptoms of herniated nucleus pulposus (lumbar disc lesion), conservative management should be tried before resorting to a surgical procedure. The danger of surgical complications, the certainty that laminectomy will cause damage to the stability of the spine, and the occasional failure of surgical procedures to relieve symptoms indicate the advisability of an initial trial of conservative treatment.

Under favorable circumstances the protruded portion of the nucleus pulposus shrinks by dehydration, and the symptoms of nerve root compression are relieved. Over a period of months the posterior wall of the anulus fibrosus heals by fibrosus, which can result in complete clinical recovery. However, if excessive pressure on the disc occurs before healing of the anulus fibrosus has progressed sufficiently, the tear will recur, additional disc material will be expelled, and symptoms will return or become aggravated. The purpose of a program of conservative management is to keep the intra-discal pressure sufficiently low for a period of time that permits adequate healing of the anulus fibrosus. In clinical experience, it takes approximately 3 months until a herniated disc patient can carry out the ordinary activities of daily living without the danger of recurrence (112).

Phases of Patient Response to Care

Three stages of patient response to treatment include:

Stage 1: Acute Inflammatory Response

In the inflammatory stage, the body responds to injury. This is a 2- to 7-day period devoted to controlling, but not eliminating, inflammation as the body starts the healing process. Vascular changes with the release of chemical reactants (e.g., bradykinins, prostaglandins, and histamines) occur in this stage with the release of proteolytic enzymes for the elimination of damaged tissue.

Stage 2: Repair Phase

Bleeding is minimized in injured soft tissue to reduce healing time. Collagen deposition starts in this 3- to 14-week postinjury period, and the tensile and elastic properties of the newly formed collagen depend on the mechanical stresses to which it is subjected. It is important to encourage the collagen fibrils to form in an alignment pattern that will allow elasticity and tensile motion in the normal motion range of the involved soft tissue. If the collagen forms in a dysfunctional scar, motion is lost and pain may occur.

Stage 3: Remodeling Phase

This phase lasts from 3 weeks to 2 years, with great overlap between stages 2 and 3. It is important that collagen is formed in the orientation of normal soft tissue alignment. This phase usually starts within 1 month of injury; therefore, rehabilitation needs to incorporate motion of the healing soft tissue into the stress motions that are normal for the tissue. This ensures ultimate motion and return of the patient's occupational and recreational activities (113).

Distraction adjustment is the principal method I use to ensure that collagen tissue is aligned in the orientation of physiological motion of the triple joint complex (intervertebral disc and two facet articulations), muscles, ligaments, and tendons. The physiologic ranges of motion of the vertebral motion segments are insured by placing each vertebra and its adjacent articulations through the motions of flexion, extension, lateral flexion, circumduction, and rotation. Coupled motions and even triple motions are used also.

Exercise Rehabilitation Protocol

No matter how much a patient is suffering from sciatica and back pain, exercises are started on the first day. These may be only tolerable knee-chest or pelvic stabilization and tilt, but patients are conditioned to know the importance of early self-care of their condition. Low back wellness school, which is taught every 2 weeks in our clinic, stresses proper lifting postures, conditioning exercises, ergonomic training, understanding of conditions for which no cure is available such as transitional segment, spondylolisthesis, disc degeneration, and so forth. Patients are told that they absolutely need to establish parameters around their daily activities to protect themselves from any pain flare ups. Nautilus rehabilitation is started when 50% relief of pain is attained. Family members are taught how to assist in the Cox exercise program and to perform massage and trigger point therapy to the patient at home. We instill in the patient that, although often no cure exists for their back pain, they can control it. It is up to us to teach them how to do so.

Protocol II: Patient Without True Sciatica

The patient without true sciatica—no pain extends below the knee—receives distraction adjustments to restore full range facet joint motion and pain relief. Full range of motion is given to the facet joints of the lumbar spine.

Orders: Distraction of the facet joints is first administered followed by passive facet joint adjustment in the physiologic ranges of motion (flexion, right-left lateral flexion, right-left circumduction, right-left rotation, and extension). One set of 10 repetitions is performed under distraction in each range of motion.

I refer to treatment of the posterior elements of the functional motor unit of the spine as treatment of back pain originating from facet syndrome. Facet syndrome is a subluxation complex of the articular processes in which increased weight-bearing occurs due to intervertebral disc narrowing and degeneration or hyperextension subluxation of the superior vertebra on the inferior segment. Facet capsule irritation, intervertebral foramen stenosis, and arthrosis may be the result. See the chapter on facet syndrome in this book for the biomechanics and mechanism of facet syndrome.

Distraction-Adjustment Procedure for Facet Syndrome

Distraction adjustment for facet syndrome is performed as follows.

1. Patient positioning. See Figures 9.13–9.17.
2. Tolerance testing sequence. See Figures 9.18 and 9.19.
3. Apply ankle cuffs securely and move footpiece caudally until taut.
4. Contact the spinous process and administer the distraction adjustment.

Two methods of contacting the spinous process are:

Figure 9.26. Contact of spinous process with index finger and thumb.

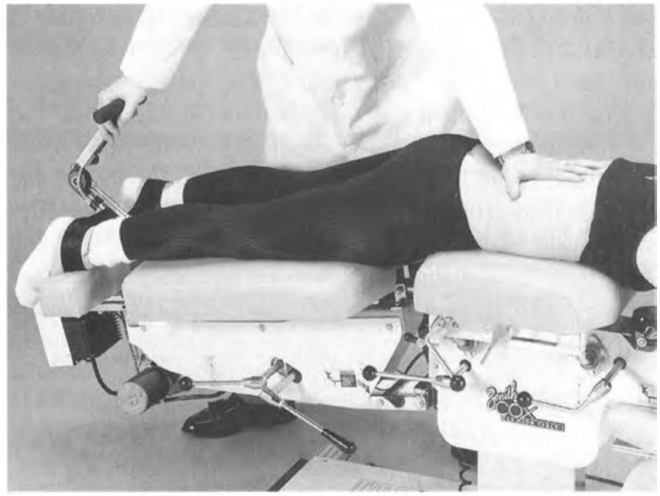


Figure 9.27. Contact of spinous process with thenar part of doctor's hand. Flexion distraction is applied for flexion of the facet joints.

1. Figure 9.26 shows the spinous process held between the thumb and index finger. This allows more precise control of stabilization than the thenar contact shown in Figure 9.27, and it is suggested for the beginning doctor to use in distraction adjustments. With this contact, the spinous process is resisted on the side of lateral flexion; if laterally flexing the spine to the right side, for example, the spinous process would be resisted on the right side by either the thumb or index finger.
2. Figure 9.27 shows the thenar contact by the doctor on the spinous process. This contact is comfortable to the patient as it allows less pressure because of the padding of the doctor's contact; however, it does not allow the delicate control against motion of the spinous process as the contact shown in Figure 9.26.

Range of Motion Adjusting Under Distraction

Flexion range of motion adjustment is shown in Figure 9.27. Apply the specific spinous process contact as shown in Figures 9.26 and 9.27. Stabilize the spinous process at the neutral starting point (Figure 9.21) and induce flexion movement of the caudal section of the table in a rhythmic and oscillatory type of motion for a total of 10 repetitions. Velocity is approximately 1 repetition per second. See Figure 9.27.

A flexion cushion can be placed under the abdomen of thin patients to flatten the sagittal lordotic curve of the lumbar spine and accommodate easier distraction of the posterior facet complex (Figure 9.22*B*).

A push-pull pumping effect is created at the interspinous space as the facets are distracted. The ease of opening the interspinous space often improves with subsequent pumping openings of the vertebral space. During flexion distraction, testing is done of the normal mobility, hypermobility, or hypomobility of the vertebral motion. When hypermobility or

normal range of motion is found, the doctor moves to the next level to be treated. If hypomobility is found, the doctor applies distraction adjustment until a normal or increased range of motion is produced. Of course, this may require sequential structured visits to regain full range of motion.

The limit of distraction application to a given vertebral level is 2 inches of downward caudal table section movement, occipital extension nutation, or patient tolerance.

Return the pelvic section to the neutral starting position, and remove the palmar stabilization force on the spinous process. The table can be locked by engaging the flexion-extension lever or left unlocked for the next step, depending on doctor preference and ability. Note: The distraction adjustments of lateral flexion and rotation are performed under flexion distraction, which are shown next while the patient is under the flexion distraction mode. The purpose of performing lateral flexion and rotation ranges of motion under distraction is the mechanical principle that the intervertebral foramen and disc space are maximally opened and patent, and the zygapophysial joints are positioned in an open, non-hyperextended position. With this anatomic posture, lateral flexion, circumduction, and rotation can be performed with lower risk of introducing stenotic entrapment of the dorsal root ganglion or nerve root by the superior facet of the inferior vertebra telescoping into the lateral recess and osseoligamentous canal. Refer to Figures 9.1–9.4 for further clarification. We reduce stenosis and its adverse effects when we apply distraction to the vertebral segments, and this reduces the possibility of iatrogenesis by further nerve root compression during the adjustment.

Flexion and extension are strong movements in the lower lumbar spine. The zygapophysial joints are capable of five normal ranges of motion, with combinations of these motions also possible. The five physiologic motions are flexion, extension, lateral flexion, circumduction, and rotation. I feel that a facet articulation that can elicit these ranges of motion will be free from subluxation. Certainly, patients are seen with conditions such as osteoporosis, osteoarthritis, degenerative disc disease, stenosis, rheumatoid arthritis, other seronegative arthritides, and collagen vascularizing diseases, as well as other pathologies, who cannot tolerate high velocity vectored thrusts to their facet joints, and distraction type adjustment is premier treatment.

Lateral Flexion Range of Motion Adjustment (Figs. 9.28 and 9.29)

1. The patient is in flexion distraction position as shown in Figure 9.27.
2. The lateral flexion lock is released.
3. Grasp the spinous process between the thumb and index finger or use the thenar contact (Figs. 9.26 and 9.27) superior to the facets to be adjusted and resist the spinous process with the contact as described for these figures. Right lateral flexion adjustment is shown in Figure 9.28 using the thumb and index finger contact and Figure 9.29 shows the thenar contact. Velocity of motion is approximately one repetition per second.
4. Step 3 is repeated to the left side. Resist the spinous process with the thumb during left lateral flexion.

   Note: The doctor can stand on either side of the table, in which case the contact of the thumb and index finger on the spinous process would be reversed. Also, instead of doing each movement 10 times right and then left, the 10 repetitions can be done in combined right and left lateral flexion at each repetition, which allows smoother comparison of right to left motion.

   Also, if hypermobility or normal motion is palpated at a given level, the doctor can stop lateral flexion. If hypomobility is felt, the doctor will perform all 10 repetitions, and then again on subsequent visits, to restore normal range of lateral flexion.
5. Return the caudal section of the table to the neutral (midline) position and secure the lateral flexion lock.

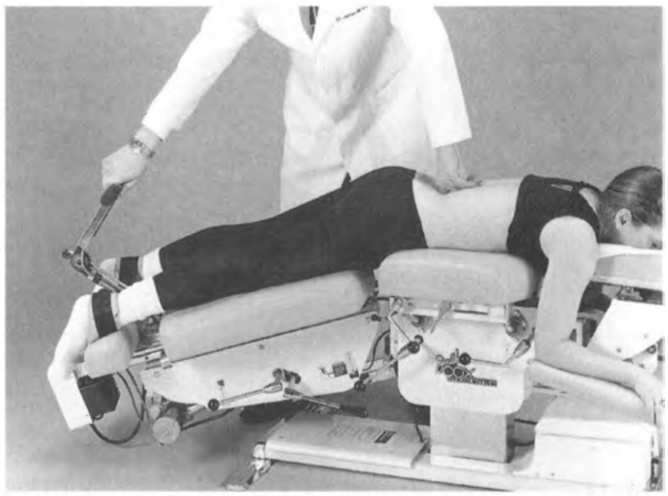


Figure 9.28. Right lateral flexion adjustment under distraction using thumb-index spinous process contact.

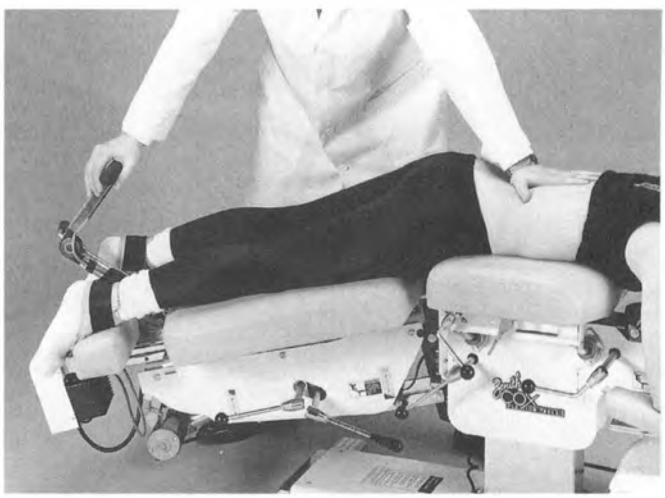


Figure 9.29. Left lateral flexion adjustment under distraction using thenar spinous process contact.

Rotation Range of Motion Adjustment (Figs. 9.30 and 9.31)

1. The patient is in flexion distraction as shown in Figure 9.27.
2. Release the rotation and flexion lock.
3. Use the thumb and index finger grasp contact to stabilize the spinous process in the midline while inducing flexion distraction and right rotation movement with the pelvic section of the table. A rhythmic and oscillatory type of motion is applied for a total of 10 repetitions. Velocity is approximately one repetition per second (Fig. 9.30).
4. Repeat step 3 on the left side (Fig. 9.31).

   Note: Steps 3 and 4 can be done concurrently to the right and left on each repetition instead of 10 to the right and then 10 to the left. This allows smoother comparison of right to left motion.

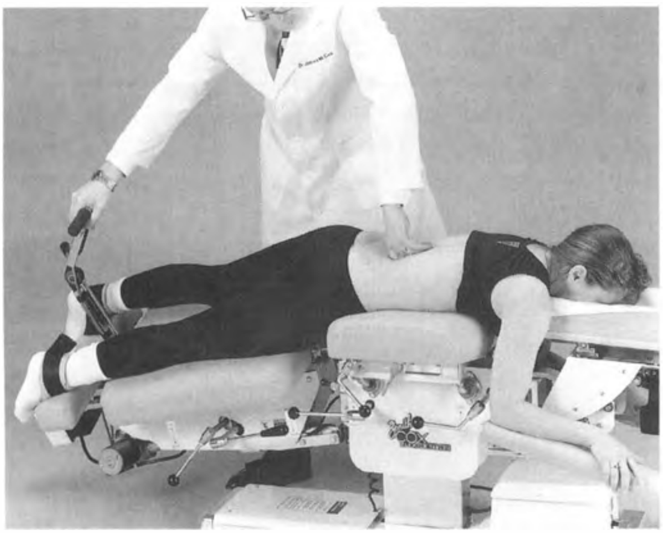


Figure 9.30. Right rotation adjustment under distraction.

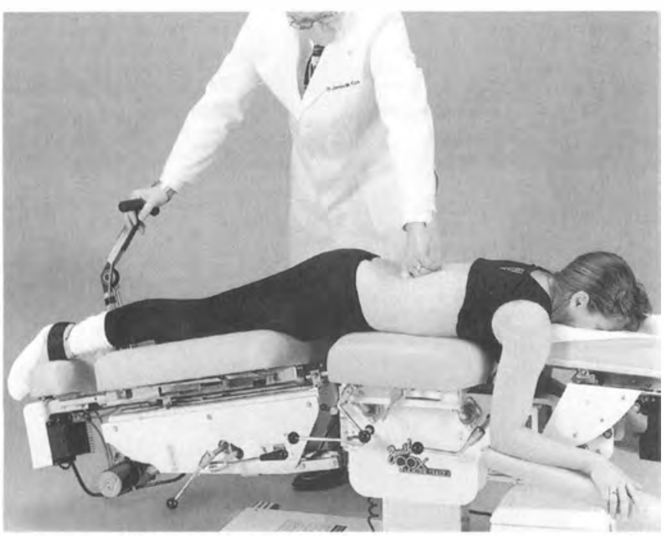


Figure 9.31. Left rotation adjustment under distraction.

Also, if hypermobility or normal motion is palpated at a given level, the doctor can stop rotation. If hypomobility is felt, the doctor applies the full 10 repetitions, and then again on subsequent visits, to restore normal range of rotation.

Rotation is applied from the L3–L4 facet levels cephalward only. Rotation is limited at the L4–L5 and L5–S1 levels and, therefore, is not applied.

The facet joints being rotated are those below the spinous process contact (i.e., if the third vertebral spinous process is contacted and stabilized the L3–L4 facets are being rotation adjusted). The spinous process pressure is applied on the side to which the segment is rotated (i.e., if right rotation is applied with the table) the pressure is applied to the right spinous process.

5. Thoracolumbar rotation is applied with the thoracic section of the table.

   Figure 9.32 shows the thoracic section of the table being used to apply right rotation to the thoracic and lumbar areas of the spine. Figure 9.33 is left rotation applied. Note that the patient is held in distraction as rotation is administered. The patient's ankles are in the ankle cuffs and distraction is applied to the spine.
6. Return the table to the neutral midline position and secure all locks.

Circumduction Range of Motion Adjustment (Fig. 9.34)

1. Release the flexion and lateral flexion locks. Circumduction is a coupled movement of flexion and lateral flexion and it starts from the neutral position of the patient at rest. The caudal section is brought into flexion distraction and then lateral flexion is applied in a smooth rhythmic coupled motion.
2. Stabilize the spinous process at the level to be adjusted with either the thumb and index finger or thenar contact as shown in Figures 9.26 and 9.27.

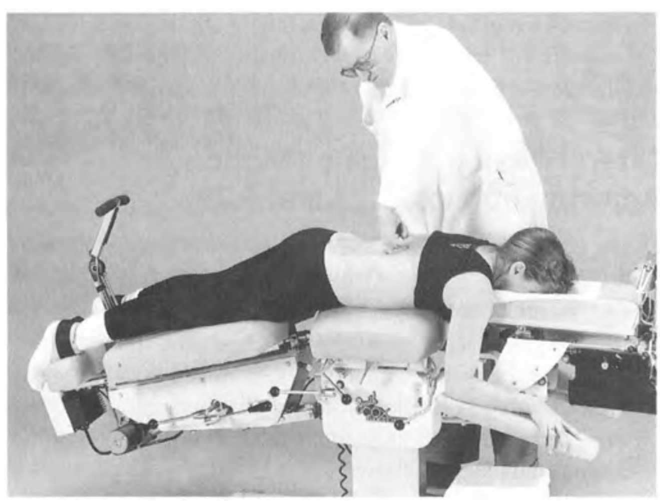


Figure 9.32. Right thoracolumbar spine rotation applied under distraction using the thoracic table section.

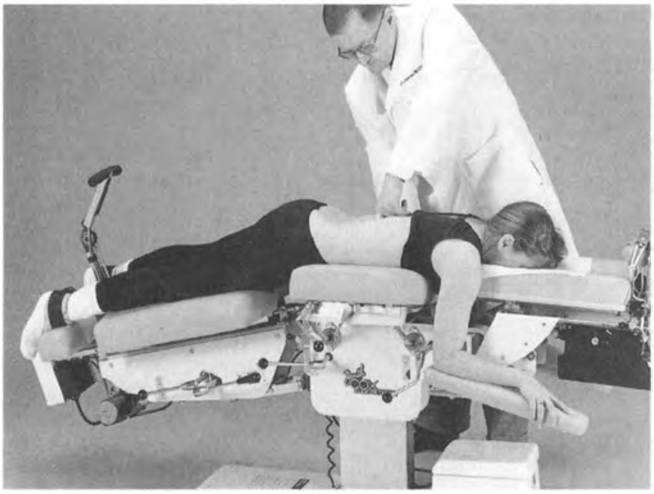


Figure 9.33. Left thoracolumbar spine rotation applied under distraction using the thoracic table section.

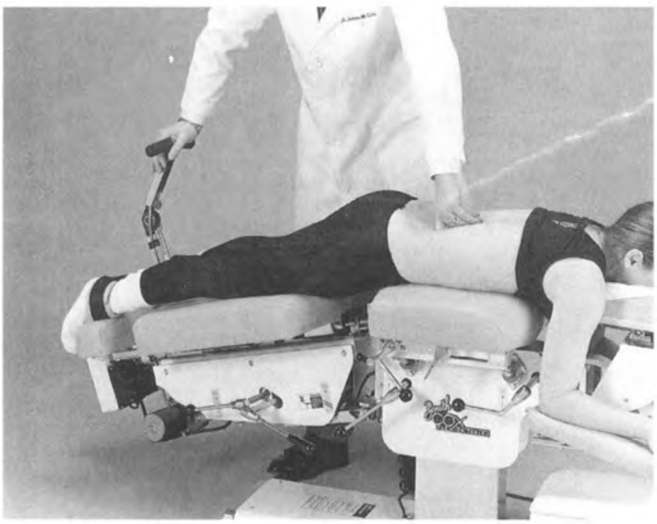


Figure 9.34. Circumduction distraction adjustment.

3. Figure 9.34 shows the procedure of circumduction with the thumb and index finger contact.
4. Circumduction can be applied to the right and left sides separately or concurrently to the right and left. Concurrent movement allows better comparison of right versus left mobility of the facet joints. Ten repetitions are performed right and left, clockwise or counterclockwise or both. If normal or hypermobility is motion palpated, stop circumduction. If hypomobility is motion palpated, apply the 10 repetitions, and again on subsequent visits, until normal or maximal range of motion is attained.

   Circumduction is the strongest motion that can be applied by the instrument to a facet joint. It elicits normal motion return of the facet joint more effectively than any other motion of the distraction adjustment subset.
5. Return the table to the neutral midline position and secure all locks.

Extension Range of Motion Adjustment (Fig. 9.35)

1. Release the flexion-extension lock.
2. Contact the spinous process of the segment to be extension adjusted and gently apply a posterior-anterior force as the caudal section of the table is brought slowly into an increased extension position. This motion is applied 10 times or until return of normal, rhythmic extension is felt.
3. Return the table to the horizontal position and secure all locks.
4. Remove the ankle cuffs.

Note: This extension adjustment can be used for compression defects of the thoracic or lumbar vertebrae to establish extension of a flexion deformity caused by compression defects of the vertebral bodies. The spinous process is held in place by the doctor's thumb and index finger or thenar contact as the caudal section of the table is slowly brought into extension, carefully monitoring patient comfort, and stopping if any discomfort is elicited.

SPECIALIZED COX DISTRACTION TECHNIQUES

Side Lying Distraction Technique for Facet Adjusting

Side lying techniques are used for pregnant females and for patients in too much pain to lie prone. Distraction flexion adjustment using this technique is shown in Figure 9.36.

Side Lying Flexion Adjustment

1. Patient lies on side with the pelvis on the caudal section of the table. Release the lateral flexion lever.

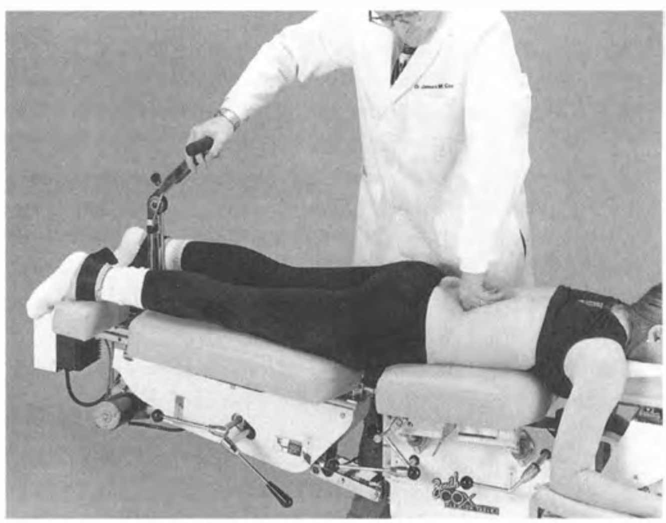


Figure 9.35. Extension adjustment.

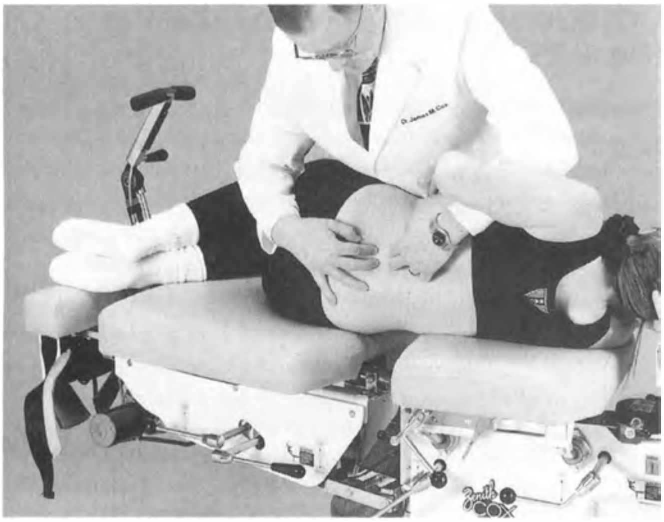


Figure 9.36. Side lying distraction flexion adjustment.

2. The doctor contacts the spinous process with the thumb and index or middle finger, index finger contact above and below the segment to be adjusted. This contact directs the application of distraction and flexion adjustment.
3. Lateral flexion motion of the caudal section is directed toward the doctor to induce flexion at the desired vertebral level while the doctor's fingers sense and direct motion. The doctor's right arm applies pelvic force on the patient to elicit flexion of the caudal section. The table is brought back to neutral by the doctor's thigh pressure.
4. Ten repetitions are used or until a smooth, rhythmic motion is attained.

Side Lying Extension Adjustment (Fig. 9.37*A*)

1. The patient lies on the side with the pelvis on the caudal section of the table. Release the lateral flexion lever.
2. The doctor contacts the spinous process as in Figure 9.36.
3. The lateral flexion motion of the caudal section is directed away from the doctor to induce extension at the desired vertebral level while the doctor's fingers sense and direct the extension motion. The doctor's finger contact on the spinous process induces an anterior force as the table moves the patient's spine into extension. The doctor applies pressure with the thigh to elicit extension with the caudal section and pulls against the patient's pelvis to return the caudal section to the neutral position (Fig. 9.37*A*).
4. Ten repetitions are used or until smooth, rhythmic motion is attained.

This is an excellent technique to place compression fractures into extension motion to reduce the hyperkyphosis formed by the vertebral body compression defect. It mobilizes and creates physiologic motion to a compromised motion segment.

Another good technique for treating compression defects of the thoracolumbar spine is shown in Figure 9.37*B*. Here the patient lies supine with the compression defect on the lower thoracic section. Slow, controlled flexion of the caudal piece allows extension of the flexion deformed area of the spine. Perform the same 10 repetitions with careful tolerance testing of the patient.

Side Lying Lateral Flexion Adjustment (Fig. 9.38)

1. The patient lies on the side with the pelvis on the caudal section of the table. Release the flexion lever.
2. The tension on the caudal section is set to the weight of the patient so that the table slowly returns to neutral from the flexed position. This affords easier adjustment control for the doctor during the procedure.
3. The doctor contacts the spinous process with the thumb and

Figure 9.37. **A.** Side lying distraction extension adjustment. **B.** Supine extension adjustment.

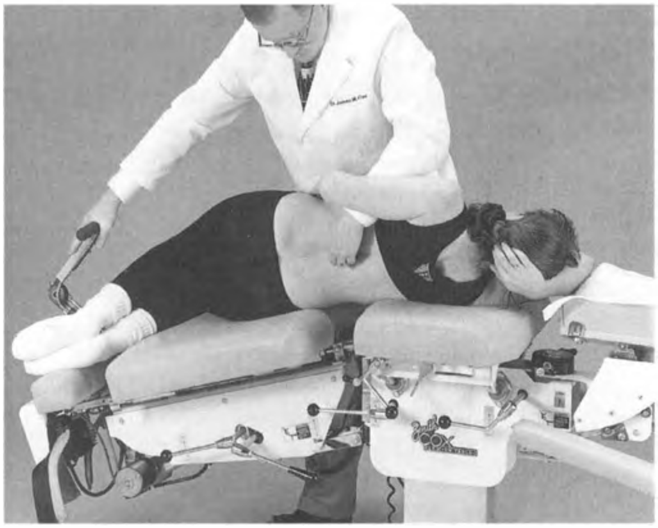


Figure 9.38. Side lying distraction lateral flexion adjustment.

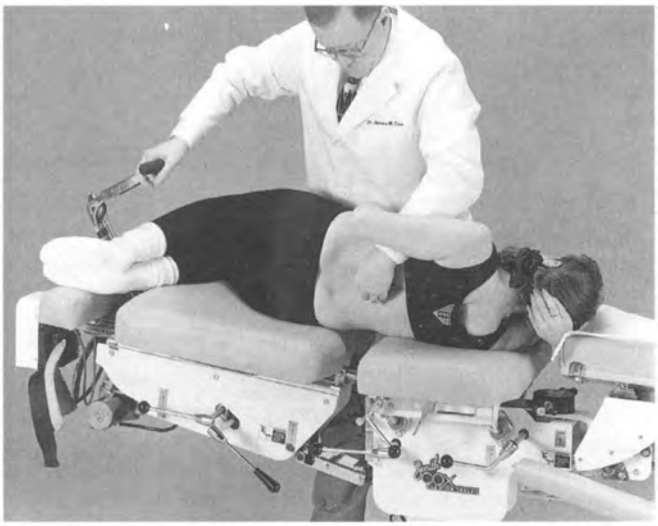


Figure 9.39. Side lying distraction circumduction adjustment.

index finger above the facet joints to be adjusted. The spinous process contact stabilizes the motion segment as the doctor moves the table into flexion and extension to elicit lateral flexion of the vertebral segment below it.

4. The doctor moves the caudal section into flexion and extension to patient tolerance to elicit lateral flexion movement to the facet joints.
5. Ten repetitions are used or until smooth, rhythmic motion is attained.

Side Lying Circumduction Adjustment (Fig. 9.39)

1. The patient lies on the side with the pelvis on the caudal section of the table. Release the lateral flexion and flexion levers.
2. The tension on the caudal section is set to the weight of the patient so that the table slowly returns to neutral from the flexed position. This eases adjustment stress for the doctor during the procedure.
3. The doctor contacts the spinous with the thumb and index finger above the facet joints to be adjusted. The spinous process stabilizes the motion segment as the doctor moves the table and patients motion segment into flexion and lateral flexion coupled motion (circumduction).
4. The doctor moves the caudal section into flexion and lateral flexion to produce circumduction motion. Patient tolerance is monitored.
5. Ten repetitions are used or until smooth, rhythmic motion is attained.

Scoliosis Distraction Adjustment (Figs. 9.40–9.42)

1. Figure 9.40 shows the table set for treating an S-shaped thoracolumbar scoliosis, thoracic curve right and lumbar curve left. The thoracic spine dextroscoliotic curve is reduced by using the thoracic section to remove the right posterior convexity. The levorotatory curve of the lumbar spine is reduced by using the caudal section and placing it into left derotation. The contact hand of the doctor applies cephalward pressure over the rib hump convexity of the scoliotic curve in the thoracic spine while distraction is applied with the caudal section.
2. Figure 9.41 shows the levorotatory lumbar scoliotic curve adjusted by contacting the left lumbar convexity with the doctor's left hand while applying flexion distraction and left lateral flexion with the caudal section.
3. Figure 9.42 is a side lying distraction adjustment into the convexity of a dextrorotatory thoracolumbar scoliotic

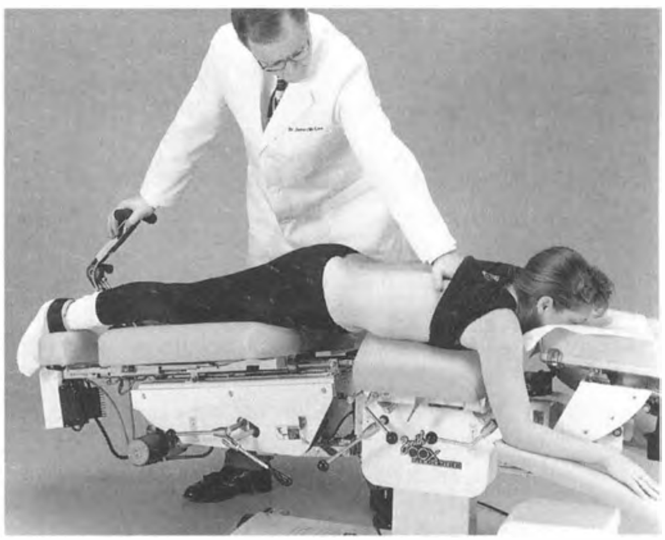


Figure 9.40. Thoracolumbar "S" scoliosis distraction adjustment of the dextrorotatory thoracic component of the scoliosis.

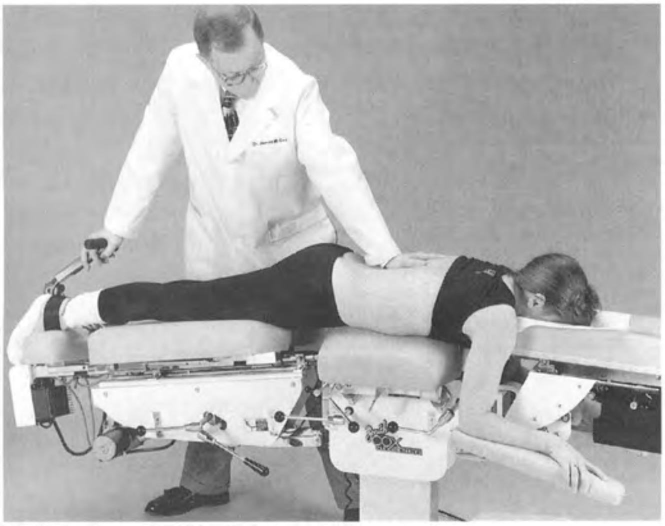


Figure 9.41. Thoracolumbar "S" scoliosis distraction adjustment of the levorotatory lumbar component.

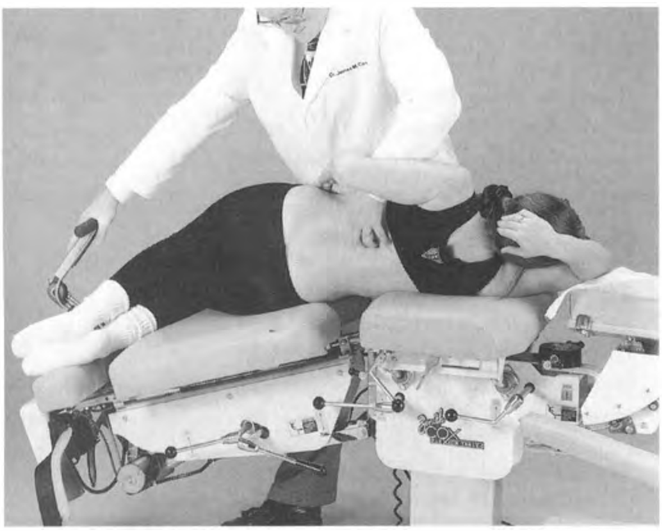


Figure 9.42. Side lying distraction adjustment for dextroscoliosis of the thoracolumbar spine.

curve. The doctor contacts the paravertebral space between the spinous and transverse processes at the apex of the scoliotic curve. An upward lift of the convexity of the curve is given by the doctor's contact as the caudal section is taken into flexion. This is a leverage technique into the convexity of the scoliotic curve to regain motion and relieve pain. It is applied to patient tolerance. Flexion and extension of the spine can be applied with this maneuver to further the physiologic motion of the involved motion segments of the curve. Scoliotic curves often do not respond well to vector high velocity–low amplitude adjustments, and this technique offers relief when other techniques are too painful or not possible to perform.

Sacroiliac Joint Distraction Adjustment (Figs. 9.43–9.45)

1. Figure 9.43 shows positioning of the patient for correction of a right posterior sacroiliac joint subluxation or a left anterior sacroiliac joint subluxation. Note that the right posterior sacroiliac joint subluxation is reduced by bringing the thoracic section of the table anterior and the caudal section posterior. This allows the ilium to pivot anteriorly and the ischium to pivot posteriorly. The left anterior sacroiliac joint subluxation is reduced by bringing the ilium posteriorly and the ischium portion of the pelvis anteriorly. As the doctor applies flexion to the lumbar spine, as shown in Figure 9.43, the sacroiliac joints are leveraged into proper alignment.

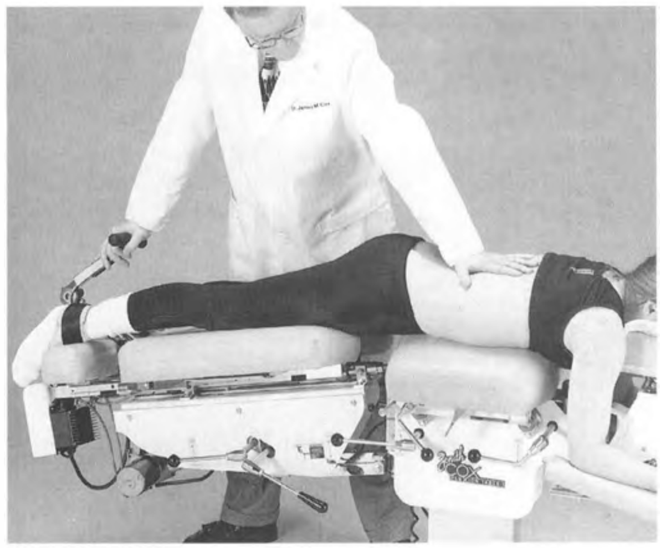


Figure 9.43. Thoracic and caudal section setting for reducing a right posterior and left anterior sacroiliac subluxation.

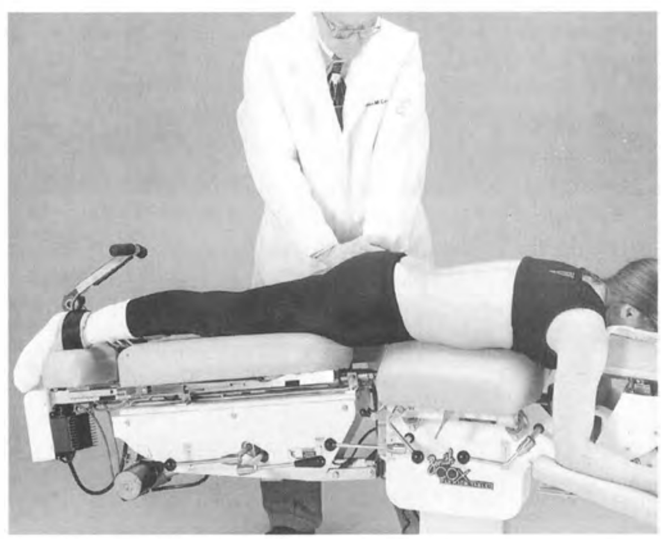


Figure 9.44. Vector thrust adjustment of the anterior sacroiliac joint subluxation with the table set for its reduction.

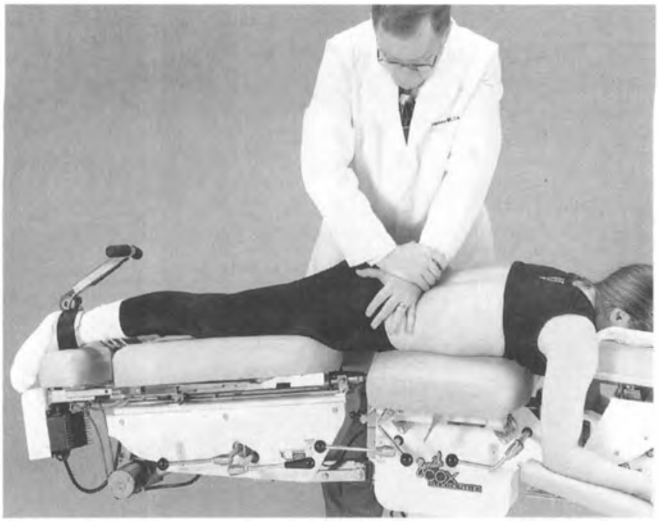


Figure 9.45. Vector thrust adjustment of the posterior sacroiliac joint subluxation with the table set for its reduction.

2. Figure 9.44 shows the assisted vector thrust or posteroanterior pressure applied to the ischium to reduce the anterior left sacroiliac joint subluxation. The table is in the position described in Figure 9.43 for the reduction of left anterior sacroiliac joint subluxation.
3. Figure 9.45 shows the assisted vector thrust or posteroanterior pressure applied to the posterior superior iliac spine and ilium on the side of posterior sacroiliac joint subluxation. The table is in the position described in Figure 9.43 for the reduction of right posterior sacroiliac joint subluxation.

Spondylolisthesis Distraction Adjustment (Fig. 9.46)

1. The patient lies prone with a flexion roll under the spondylolisthesis segment.
2. The doctor contacts the spinous process above the level of spondylolisthesis (e.g., if it were an L5 spondylolisthesis, the doctor would contact the L4 spinous process).
3. If the patient with spondylolisthesis has sciatica with a herniated disc, the treatment would be with the protocol for the disc herniation patient described in Figures 9.22–9.25.
4. If the spondylolisthesis patient does not have true sciatica and a herniated disc, full range of motion via the protocol for patients without sciatica is followed as shown in Figures 9.26 to 9.34, 9.36, 9.38, and 9.39.
5. Note that with the flexion roll under the abdomen, the distractive force necessary to elicit disc or facet motion is reduced substantially. Gentle distraction with spondylolisthesis patients is the ideal therapy.

AUTOMATED DISTRACTION ADJUSTING

In 1976, I developed the first automated flexion distraction instrument for applying distraction adjustments (Fig. 9.47). Use and distribution of the instrument was stopped in 1977 because of lack of investigation into safety and use of motorized distraction.

With Williams Healthcare Systems, Inc., I have studied the necessary safety and increased clinical benefits of automated distraction and have developed a procedure of automatic distraction adjusting that is applied with strict parameters for patient comfort and safety.

Benefits of Automatic Distraction Adjusting

1. Doctor ease. In treating patients, doctor fatigue is lessened by mechanical assistance, which equates to improved doctor concentration. Stamina is used to treat and care for patients.
2. Improved patient care. The new automated distraction assures consistent, rhythmic, smooth, oscillatory distraction to the spine and allows the doctor to concentrate entirely on the vertebral segment motion when applying distraction.
3. The necessary factor is safety in using automated distraction and why it is superior on the Zenith-Cox instrument is discussed below.

Treatment Parameters with Automated Distraction Adjusting

1. Automated distraction adjusting is never used in treating patients with sciatic radiculopathy caused by intervertebral disc herniation. Only manual adjustment under distraction is used for this patient.
2. When treating the patient without sciatic radiculopathy, careful tolerance testing is done in the same manner as with manual distraction. Tolerance testing is performed prior to placing facet joints and disc into flexion and distraction movement. Any increased pain indicates that automated distraction is not to be used.
3. Tolerance testing is performed using the protocol in the

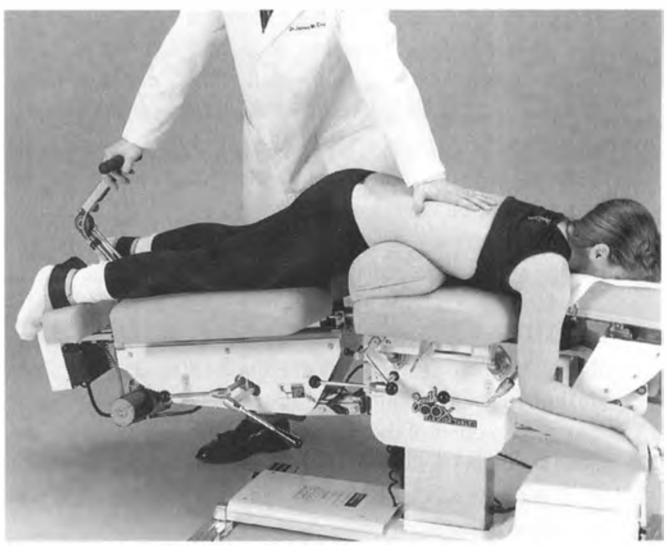


Figure 9.46. Spondylolisthesis distraction adjustment.

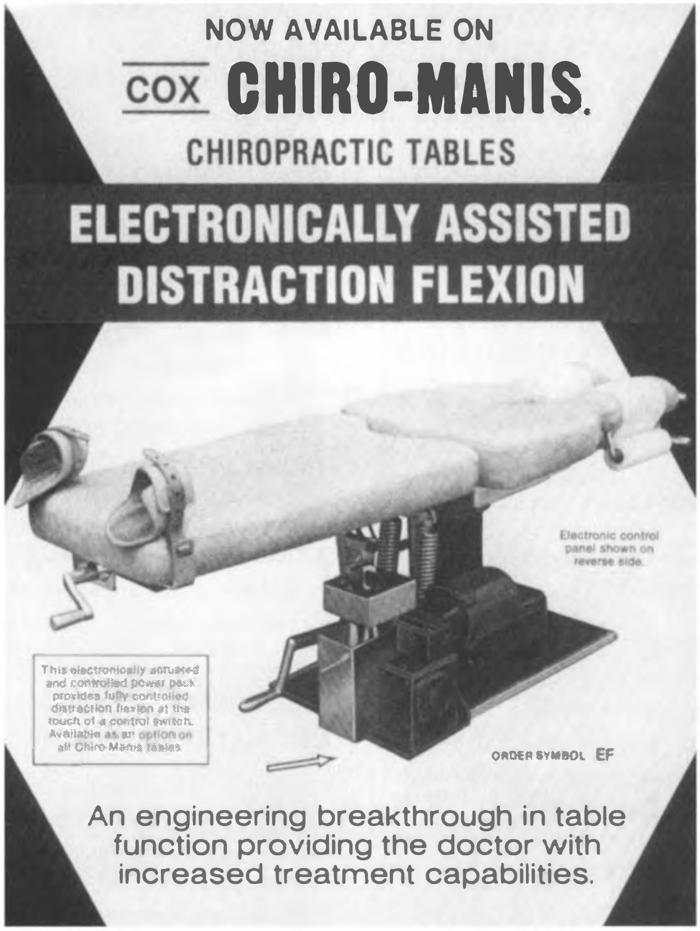


Figure 9.47. The first Cox automated flexion distraction adjustment instrument, which was made in 1976.

Cox distraction certification course taught by the National College of Chiropractic as outlined earlier in this chapter. Again, remember that this automated distraction technique is only used for nonsciatica patients.

- The patient is placed into the flexion, neutral, or extension angle that feels comfortable and/or relieves pain.
- Tolerance testing is then performed. This is the same tolerance testing done for manual use of the table. The doctor contacts the spinous process above the vertebral segment to be distracted and slowly applies automated axial distraction, first only using the patient's lower extremity weight as the tractive force. If no pain is elicited, grasping the uninvolved lower extremity at the ankle and applying axial automated distraction is performed. Then the involved lower extremity is tested by lateral tolerance. Note that in axial automated distraction testing, the hold time on distraction is not for 4 seconds, but only for the time of axial automated distraction to be delivered by the caudal section of the table. This varies, depending on the speed set by the doctor. Use a slow speed to elicit a slow distraction of the motion segments to be tested. Test the levels that are to be adjusted under distraction.

4. Automated distraction adjustment is never applied without the doctor being present in the treatment room to supervise and apply the technique. This is not just traction. It is controlled distraction adjusting performed by the chiropractic doctor.

Automated Distraction Adjusting Set for Patient Comfort

The clinical advantage of Cox automated distraction adjustment is distraction applied while the clinically necessary lumbar sagittal (flexion) angle of the instrument is set to allow patient comfort.

See Figures 9.48 to 9.59 on how to use automated distraction in adjusting the lumbar spine.

Automated Axial Distraction at a Preset Flexion Angle (Fig. 9.48)

1. The patient lies prone, undergoes tolerance testing, and the doctor assumes the palpatory contact and treatment position for distraction for the manual technique described above (Figs. 9.20–9.22).
2. The flexion angle of the table is either preset at a fixed point

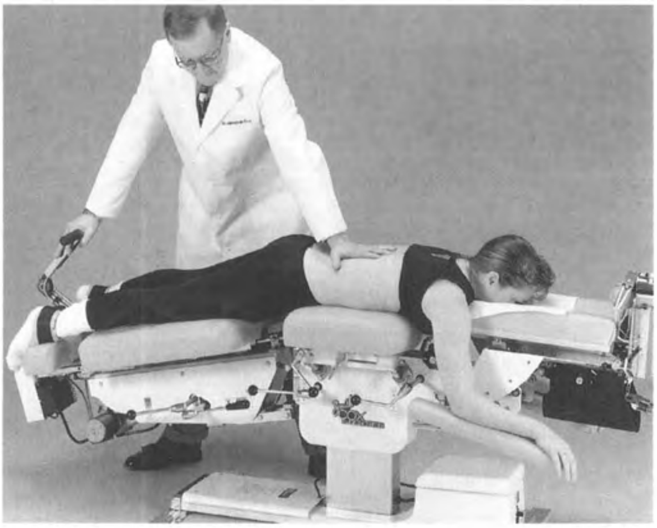


Figure 9.48. Automated axial distraction applied at L5-S1.

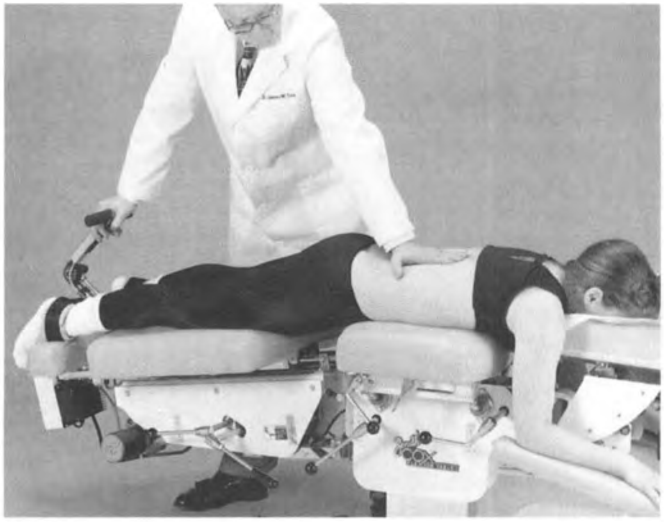


Figure 9.49. Left lateral flexion with automated axial distraction as a coupled motion.

for patient comfort, or it can be constantly altered by the doctor to elicit patient comfort and response.

3. In Figure 9.48 is shown the doctor's thenar contact on the spinous process of L5 as automated axial distraction is applied.
4. The speed of axial distraction is selected and altered by the doctor to match patient comfort and response.
5. The limit of distraction is patient occipital extension nutation, the feeling of interspinous space separation at the level of applied distraction adjustment, or patient comfort.
6. Ten rhythmic, smooth, steady distractions are applied until normal range of motion is appreciated. Less than ten repetitions may be used if normal range of motion is elicited earlier.

Automated Axial Distraction Combined with Left and Right Lateral Flexion (Figs. 9.49 and 9.50)

1. Using the protocol in Figure 9.48, lateral flexion is applied to both the right and left.
2. The doctor can choose to couple the motions of distraction and lateral flexion, or place the patient in distraction, stop the distraction at the desired point, and apply lateral flexion.
3. Lateral flexion is applied only to the right or left under distraction, or continuous right and left lateral flexion applied. The latter allows continuous monitoring of the equality of lateral flexion in both directions. This is an excellent motion palpation procedure.
4. If the doctor chooses to preset the distraction force prior to applying lateral flexion, a switch on the handle of the table is pressed to stop the axial distraction at the desired limit.
5. Ten rhythmic, smooth, steady distractions are applied until normal range of motion is appreciated. Less than the ten can be used if normal range of motion is elicited earlier.

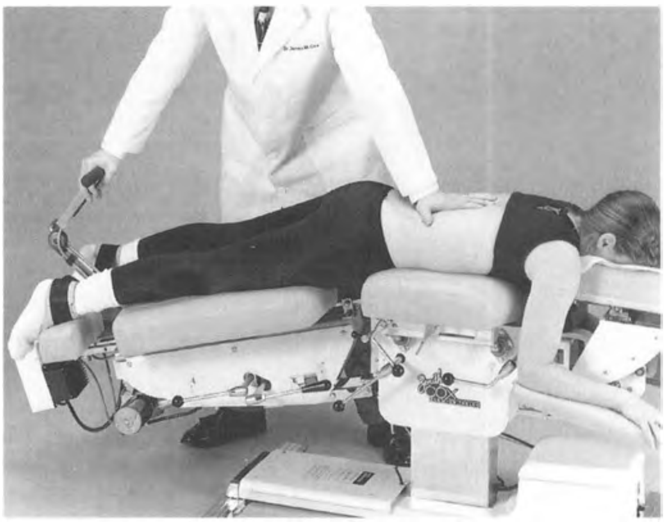


Figure 9.50. Right lateral flexion with automated axial distraction as a coupled motion.

Eight-Finger Glide Palpation (Fig. 9.51)

The doctor places the finger tips between the spinous processes to appreciate the tautness and tractioning effect on the interspinous spaces, which enables the doctor to set the limit of distraction as this tautness and stretch is palpated.

Automated Axial Distraction Combined with Left and Right Rotation (Figs. 9.52 and 9.53)

1. Using the protocol in Figure 9.48, rotation is applied to both the right and left.
2. The doctor can choose to couple the motions of distraction and rotation, or place the patient in distraction, stop the distraction at the desired point, and apply rotation.

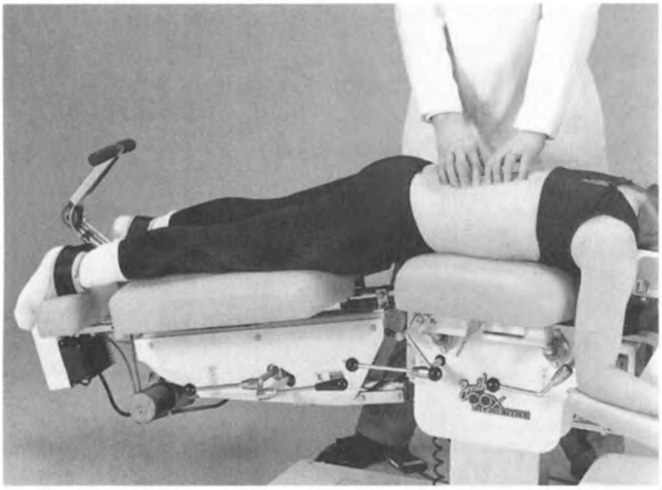


Figure 9.51. Eight-finger glide palpation.

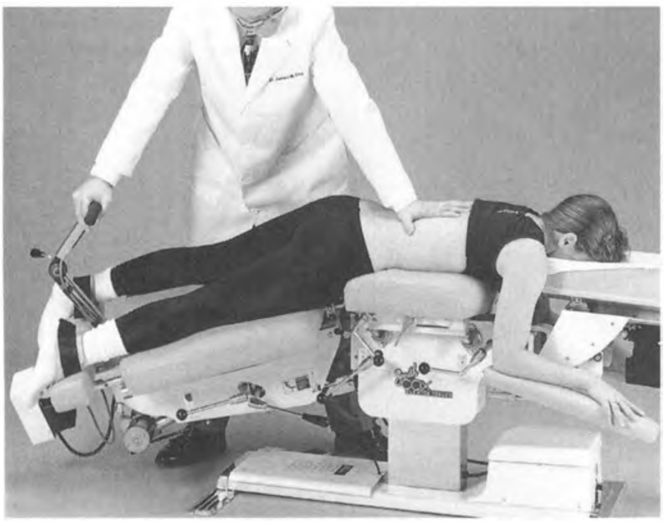


Figure 9.52. Right rotation with automated axial distraction applied in a coupled motion.

3. Rotation can be applied only to the right or left under distraction, or continuous right and left rotation applied. The latter allows continuous monitoring of the equality of rotation in both directions. This is an excellent motion palpation procedure.
4. If the doctor chooses to preset the distraction force prior to applying rotation, a switch on the handle of the table is pressed to stop the axial distraction at the desired limit.
5. Ten rhythmic, smooth, steady distractions are applied until normal range of motion is appreciated. Less than the ten can be used if normal range of motion is elicited earlier.

Automated Axial Distraction in the Thoracic Spine (Fig. 9.54)

1. The protocol in Figure 9.48 can be applied to the thoracic spine.

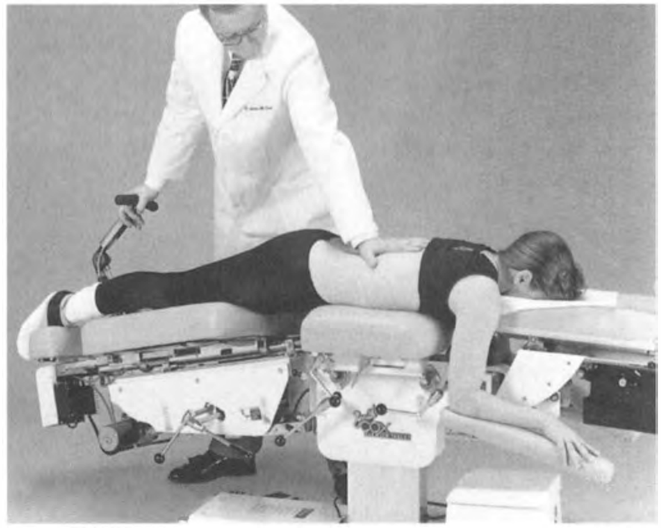


Figure 9.53. Left rotation with automated axial distraction applied in a coupled motion.

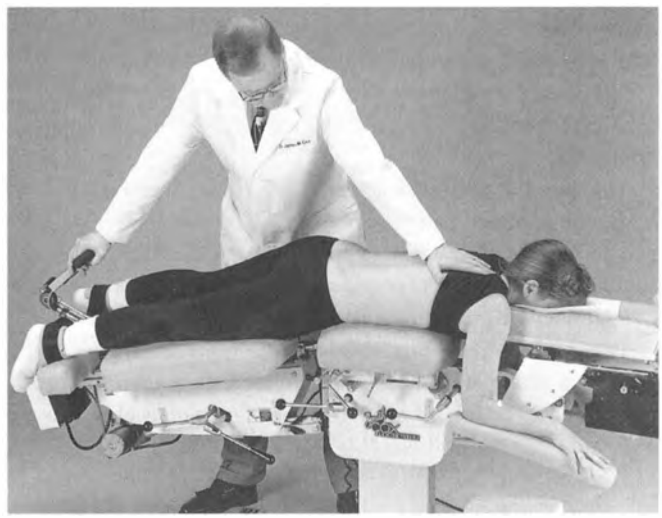


Figure 9.54. Automated axial distraction of the thoracic spine segments and thoracolumbar spine below the spinal contact.

2. Segments of the thoracic spine can be automatic axially distracted as the doctor applies spinous process contact to the segments of the thoracic and lumbar spine.

Foramen Magnum Pump (Fig. 9.55)

1. The protocol in Figure 9.48 can be applied to the full spine.
2. The full spine is distracted by cradling the occiput in the doctor's hands while automated axial distraction is slowly applied to patient monitoring and tolerance. This is a sedating and relaxing sensation for the patient, but it must be carefully administered according to the protocol in Figure 48.
3. Note that the doctor has both treating hands on the patient's cervical spine. This can be done because the foot switch on the floor allows automated axial distraction administration without using one hand to press on the handle switch.

Automated Axial Distraction for Scoliosis (Figs. 9.56 and 9.57)

1. Following the protocol in Figure 9.48, automated axial distraction can be used in patients with scoliosis.
2. Figure 9.56 shows thoracic dextroscoliosis being automated axially distracted. Note the table is set to the reduction of the curve as described under manual distraction in Figure 9.40.
3. Figure 9.57 shows application of the foramen magnum pump (Fig. 9.55) for the treatment of scoliosis. Note that the thoracic and lumbar components of the scoliotic curve are reduced by the rotation sections on the thoracic and lumbar parts of the table as shown in Figure 9.40.
4. This allows full spine distraction of the scoliotic spine as the convexity of the curves are derotated. It proves sedating to scoliotic patients when applied to patient tolerance.

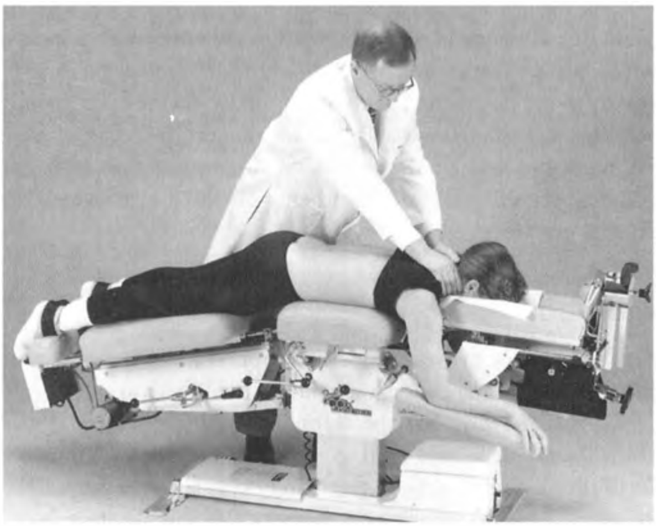


Figure 9.55. Foramen magnum pump administered with automated axial distraction.

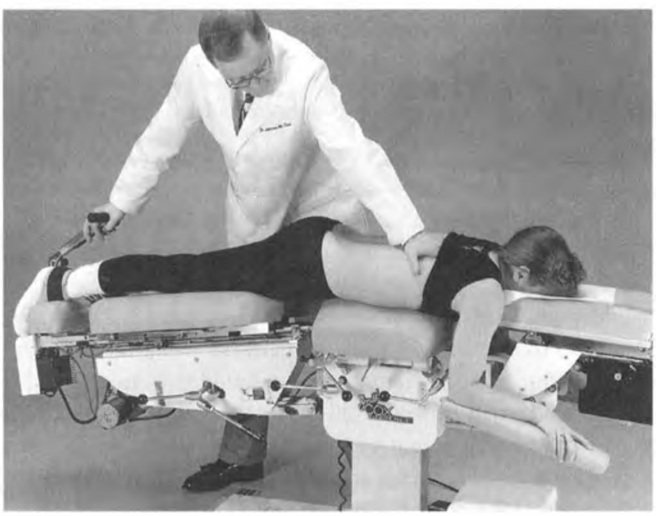


Figure 9.56. Automated axial distraction to the thoracic spine for scoliosis adjustment.

Automated Axial Distraction with Bilateral Hand Contact of the Spine (Fig. 9.58)

1. The foot switch is used instead of the pressure button on the handle to allow both of the doctor's hands to apply automated axial distraction.
2. Following the protocol of Figure 9.48, both hands are used to apply the automated axial distraction.
3. This technique allows the doctor more control of the vertebral motion segment by bilaterally contacting the arch of the vertebra. Vector direction of the doctor's cephalward force, either laterally or inferiorly, is possible with this two-handed distraction adjustment.

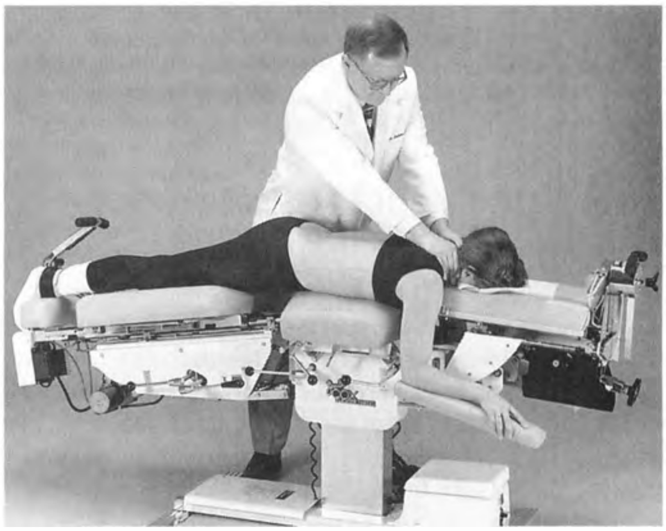


Figure 9.57. Foramen magnum pump applied with automated axial distraction for the treatment of scoliosis. Note the reduced spinal convexities with the table sections prior to distraction.

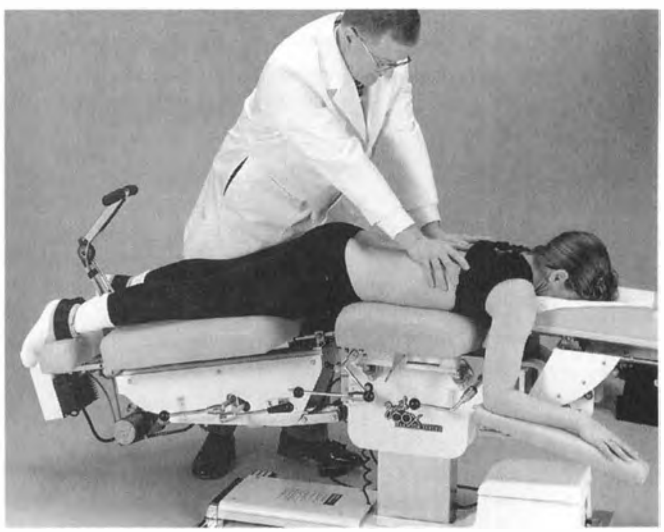


Figure 9.58. Automated axial distraction with both of the doctor's hands available to deliver the adjustment.

Automated Axial Distraction Vector Thrust Adjustment (Fig. 9.59)

1. As in Figure 9.58, the doctor can apply posteroanterior vector low amplitude, high velocity adjustments to the vertebral segments while automated axial distraction is applied to the segment. Hold the torque contact while automated axial distraction is delivered to the spine at the doctor's hand contact. Often this gentle distraction produces cavitation of the joints. It is a gentle way to adjust individual motion segments, especially those at the site of degenerative disc or facet disease.
2. Automated axial distraction can make hypomobile or fixated segments easier to cavitate. It allows physiologic motion to be applied to the spine in preparation for the vector type of adjustment. For many patients, this makes the adjustment less discomforting.
3. Adjustment of the vertebral motion segment under distraction is an exciting part of progressive chiropractic procedures.

OUTCOME MEASURES OF 1000 CASES STUDIED USING CHIROPRACTIC DISTRACTION ADJUSTMENT

It is incumbent on chiropractors to rely on meaningful patient outcomes to determine the patient's health disposition clinically or generally. Procedures in chiropractic must document worthwhile change in functional health status such as quality of life, activities of daily living, return to work, or economic efficiency (116). As the technique of chiropractic distraction adjusting has evolved to the extent described above, so too has the interest in research results stemming from its utilization.

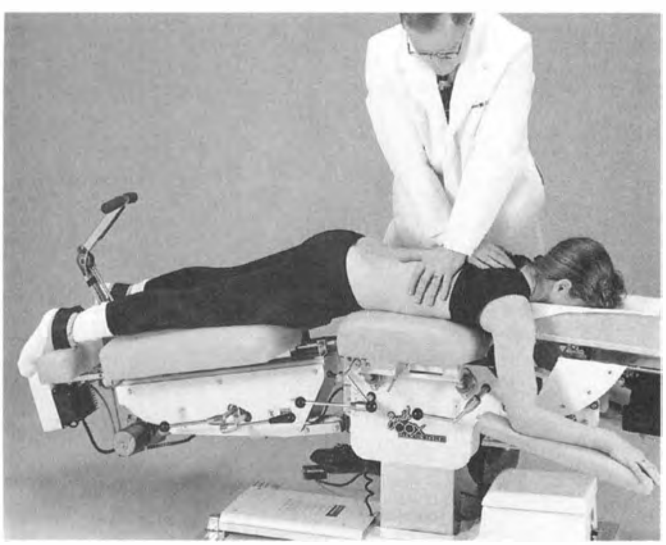


Figure 9.59. Vector thrust adjustment administered under automated axial distraction.

Objective

Study of clinical efficacy of Cox Distraction Adjusting, as with any technique, is confronted with the ethical considerations of comparing care with no care. Paterson addressed the problem of conducting such clinical controlled trials:

> *"In a survey of personal practice, where measurement of criteria is largely unattainable, and where subjective impressions form the very basis of assessment of pain and altered sensation, it is inappropriate to attempt anything in the nature of a controlled trial. In particular is this so when attempting to demonstrate the efficacy of a mode of therapy compared with regimes and treatments totally dissimilar: the essence of the controlled trial lies in the comparison of basically similar variables. Clearly it would be ethically quite unacceptable to offer manipulative therapy to one patient with pain of mechanical origin, in the reasonable expectation of rapid relief, while sending another to bed to languish there quite unnecessarily, the choice being made on numerical labeling rather than on clinical grounds. For this reason this paper is presented as a simple series, relying upon numbers to merit significance" (114).*

With this dilemma in mind, I set out to collect data on as many consecutive cases as possible without predetermination of who may respond positively to this manipulative therapy. I certainly acknowledge that this study lacks a randomized clinical trial regimen, but was an attempt to document with available resources, the clinical outcomes being obtained in chiropractic clinical practices.

Method

The 1000 cases presented were compiled from two separate but identical data collection studies published in 1984 and 1994, respectively. For the compiled 1000 cases studied, 30 chiropractic physicians used an identical six-page examination form (available from the author) and collected data on at least 20 consecutive low back pain patients who sought their care. The chiropractors involved in the study used distraction adjustments as the primary technique in 92% of the cases treated. Additionally, adjunctive modalities such as electrical stimulation, massage, hot/cold therapy, trigger point therapy, and bracing were administered.

Results

Prior to viewing the data, it is imperative to understand the stages of low back pain—acute, subacute, and chronic—and that less than 20% of back pain sufferers usually progress to the third, chronic, stage. Pain duration of less than 6 weeks is classified as acute; that lasting 12 weeks is subacute; and thereafter it is defined as chronic if symptoms persist (115). Within 6 weeks 80 to 90% of low back pain attacks will resolve (115). In this 1000-case compilation, it was found that following the algorithms shown earlier in this chapter used in making decisions to the management of the low back pain patients using Cox Distraction Protocols accordingly, only 8.7% of patients progressed to the chronic stage of pain (Table 9.1).

Maximal improvement with Cox Distraction Adjusting is defined as 3 months of conservative care, re-establishment of

Table 9.1

Days to Maximal Improvement Under Chiropractic Care

Condition	No. of Days to Maximal Improvement: 10 or fewer	11–20	21–30	31–45	46–60	61–90	91+
Discogenic spondyloarthrosis							
Level L4	27	27	39	24	11	25	11
(164 cases)	16.5%	16.4%	23.8%	14.6%	6.7%	15.3%	6.7%
Level L5	58	39	54	37	20	31	18
(257 cases)	22.6%	15.1%	21.1%	14.4%	7.7%	12.1%	7.0%
Disc protrusion							
Level L4	7	16	11	16	9	15	15
(89 cases)	7.9%	17.9%	12.4%	18.0%	10.1%	16.8%	16.9%
Level L5	11	17	25	19	9	17	12
(110 cases)	10.0%	15.5%	22.7%	17.3%	8.1%	15.5%	10.9%
L-5 transitional segment							
Level L5	7	3	6	8	2	5	3
(34 cases)	20.6%	8.8%	17.7%	23.5%	5.9%	14.7%	8.8%
Lumbar spine sprain/strain							
Level L4	12	11	11	5	4	6	2
(51 cases)	23.5%	21.6%	21.6%	9.8%	7.8%	11.8%	3.9%
Level L5	27	20	21	13	7	14	3
(105 cases)	25.7%	19.1%	20.0%	12.3%	6.7%	13.3%	2.9%
Facet syndrome							
Level L4	26	19	24	24	10	22	14
(139 cases)	18.7%	13.7%	17.2%	17.3%	7.2%	15.8%	10.1%
Level L5	66	65	66	50	25	42	34
(348 cases)	19.0%	18.6%	19.0%	14.4%	7.2%	12.0%	9.8%
Spondylolisthesis							
Level L4	4	2	4	8	2	7	1
(28 cases)	14.3%	7.1%	14.3%	28.6%	7.1%	25.0%	3.6%
Level L5	8	5	5	2	3	4	2
(29 cases)	27.6%	17.2%	17.3%	6.9%	10.3%	13.8%	6.9%
Overall average for all conditions							
All conditions	153	138	153	110	65	105	69
(793 cases)	19.3%	17.4%	19.3%	13.9%	8.2%	13.2%	8.7%

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Average no. of days to maximal improvement = 29.

the preinjury state, or 100% relief of pain. The mean number of days to maximal improvement is 29 (Table 9.1), and number of treatments to maximal improvement is 12 (Table 9.2). Overall patient response to care was 70.7% good to excellent (Table 9.3).

Table 9.4 shows patient response to care by diagnosis. Lumbar sprain or strain at L4 and L5 was highest in the good to excellent responses—83.1 and 83.5%, respectively. Disc herniation at L4 and L5 responses produced 60.7 and 65.8% good to excellent responses, respectively. Only spondylolisthesis at L4 had lower good to excellent responses, 58.8%.

Many factions—patients, insurance companies, doctors, employers—are concerned with the number of days and visits it will take to help the patient. Preventing a patient from moving into the chronic phase, more than 3 months or 90 days of care, is critical. By condition, disc herniation has the greatest chance of becoming chronic (14%), and facet syndrome is second, 10% (Table 9.5). Overall, 9% of patients required care for more than 90 days (Table 9.5).

As to the number of visits required to reach maximal improvement, L4 disc herniation leads the list: 56% of L4 herniations required more than 20 visits and 55% of patients with L4 spondylolisthesis required more than 20 visits (Table 9.6). Overall, 29% of patients required more than 20 visits, and 17%

Table 9.2

Treatments to Maximal Improvement Under Chiropractic Care

Condition	No. of Treatments to Maximal Improvement								
	10 or fewer	11–20	21–30	31–40	41–50	51–60	61–80	81–100	101+
Discogenic spondyloarthrosis									
Level L4	61	57	19	10	10	3	3		3
(166 cases)	36.7%	34.4%	11.4%	6.1%	6.0%	1.8%	1.8%		1.8%
Level L5	112	76	27	17	11	5	8		4
(260 cases)	43.1%	29.2%	10.4%	6.5%	4.3%	1.9%	3.1%		1.5%
Disc protrusion									
Level L4	17	22	23	4	6	6	6	1	4
(89 cases)	19.1%	24.7%	25.9%	4.5%	6.7%	6.7%	6.8%	1.1%	4.5%
Level L5	35	36	7	9	9	3	6	3	3
(111 cases)	31.5%	32.5%	6.3%	8.1%	8.1%	2.7%	5.4%	2.7%	2.7%
L5 transitional segment									
Level L5	13	10	8		1	1			
(33 cases)	39.4%	30.3%	24.2%		3.1%	3.0%			
Lumbar spine sprain/strain									
Level L4	27	16	4	2	1	1			
(51 cases)	52.9%	31.4%	7.9%	3.9%	1.9%	2.0%			
Level L5	57	34	6	2	3	1	2		
(105 cases)	54.3%	32.4%	5.7%	1.9%	2.8%	1.0%	1.9%		
Facet syndrome									
Level L4	63	34	17	9	7	2	3	2	3
(140 cases)	45.0%	24.3%	12.1%	6.5%	5.0%	1.4%	2.1%	1.5%	2.1%
Level L5	152	93	36	24	15	8	10	3	6
(347 cases)	43.8%	26.8%	10.4%	6.9%	4.3%	2.3%	2.9%	0.9%	1.7%
Spondylolisthesis									
Level L4	7	6	7	4	2			1	2
(29 cases)	24.1%	20.7%	24.2%	13.8%	6.9%			3.4%	6.9%
Level L5	14	8	5	2					1
(30 cases)	46.7%	26.6%	16.7%	6.7%					3.3%
Overall average for all conditions									
All conditions	338	224	99	45	34	14	19	7	13
(793 cases)	42.6%	28.3%	12.5%	5.6%	4.3%	1.8%	2.4%	0.9%	1.6%

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Average no. of treatments to maximal improvement = 12.

Table 9.3

Overall Patient Response Regardless of Diagnosis (n = 977)

Response	No. of Patients	Percent of Total	Cumulative Total Score (%)
Excellent	460	47.1	47.1
Very good	134	13.7	60.8
Good	97	9.9	70.7
Fair	72	7.4	78.1
Poor	40	4.1	82.2
Surgery	34	3.5	85.7
Stop, no start	104	10.6	96.3
Exam, not treated	36	3.7	100.0

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Table 9.4

Patient Responses to Care (n = 977)

	L4 Discogenic Spondylo-arthrosis (%)	L5 Discogenic Spondylo-arthrosis (%)	L4 Disc Herniation (%)	L5 Disc Herniation (%)	L5 Transitional Segment (%)	L4 Sprain/Strain (%)	L5 Sprain/Strain (%)	L4 Facet Syndrome (%)	L5 Facet Syndrome (%)	L4 Spondylo-listhesis (%)	L5 Spondylo-listhesis (%)	Overall Patient Response (%)
Excellent	33.8	43.0	31.6	41.6	48.8	64.4	65.3	41.1	49.0	23.5	29.7	47.1
Very good	18.4	14.6	15.4	14.1	14.6	8.5	9.1	14.9	13.2	26.5	21.6	13.7
Good	9.2	7.6	13.7	10.1	12.2	10.2	9.1	10.1	10.3	8.8	16.2	9.9
Fair	12.6	13.6	10.3	6.0	7.3	6.8	4.1	10.7	7.2	14.7	10.8	7.4
Poor	4.3	5.0	5.1	2.0		5.1	4.1	4.8	4.1	5.9		4.1
Surgery	2.4	1.3	6.8	10.7	4.9		0.8	2.4	1.7	5.9	2.7	3.5
Stopped care	13.5	11.3	10.3	12.8	7.3	5.1	7.4	11.9	11.7	14.7	8.1	10.6
Examination, not treated	5.8	3.6	6.8	2.7	4.9			4.2	2.9		10.8	3.7

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Table 9.5

Days to Maximal Improvement Under Chiropractic Care (n = 1000)

Condition	Fewer Than 90 Days (%)	More Than 90 Days (%)
Discogenic spondylosis	93	7
Disc herniation	86	14
Sprain/strain	91	9
Transitional segment	93	7
Facet syndrome	90	10
Spondylolisthesis	95	5
All conditions	91	9

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Table 9.6

Treatments for Maximal Improvement Under Chiropractic Care (n = 1000)

Condition	More Than 20 Visits (%)	More Than 30 Visits (%)
Discogenic spondylosis	28	17
Disc herniation	L-4–56	L-4–30
	L-5–36	L-5–30
Sprain/strain	29	8
Transitional segment	30	6
Facet syndrome	L-4–31	L-4–19
	L-5–30	L-5–19
Spondylolisthesis	L-4–55	L-4–31
	L-5–27	L-5–10
All conditions	29	17

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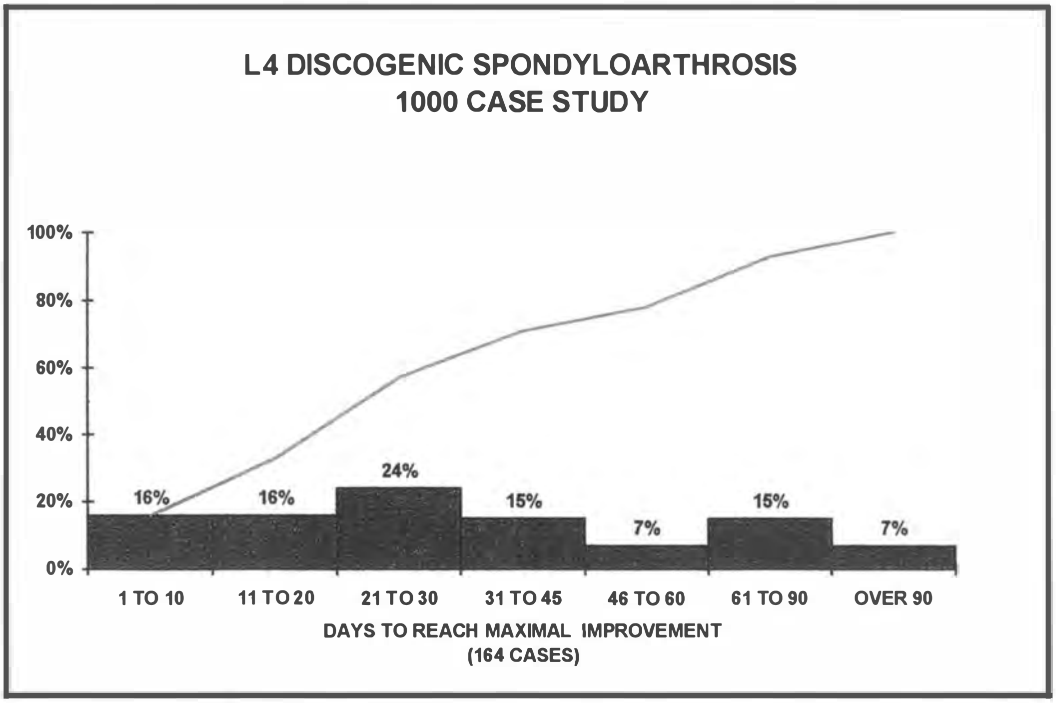
Table 9.7

Days and Treatments to Maximal Improvement of L4 Discogenic Spondyloarthrosis Diagnosis



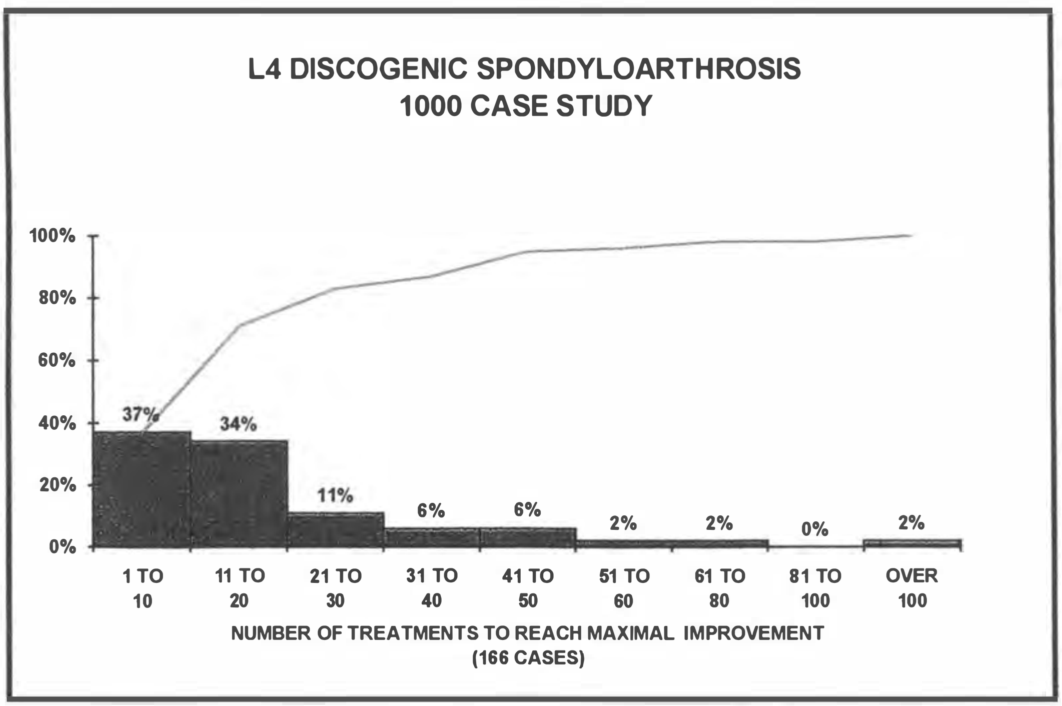


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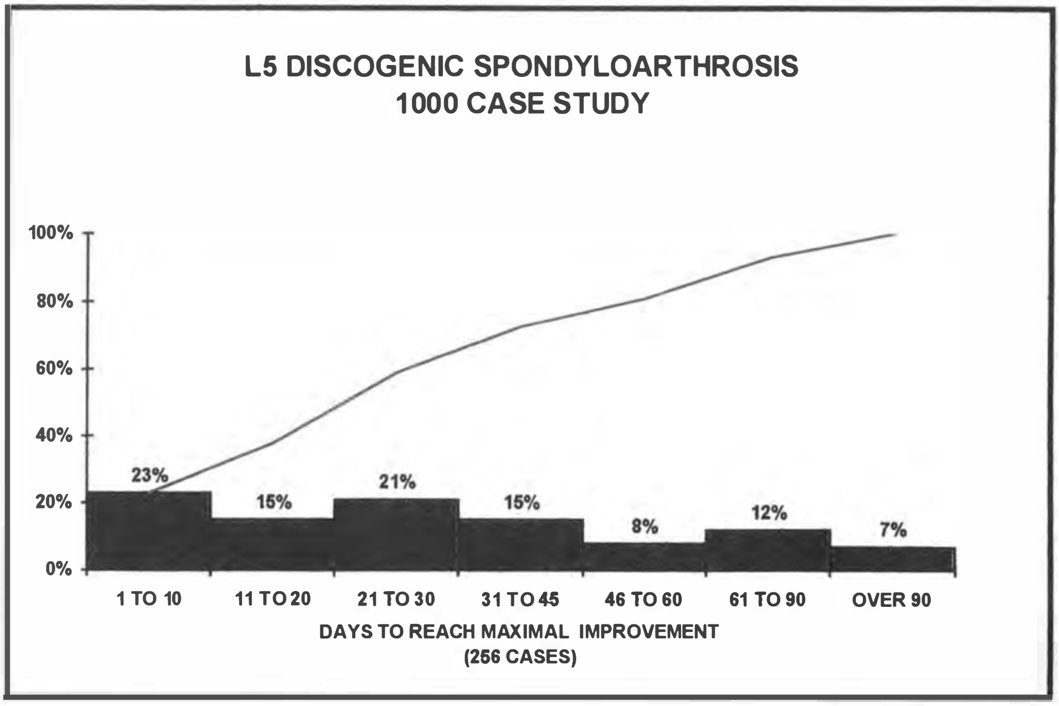
Table 9.8

Days and Treatments to Maximal Improvement of L5 Discogenic Spondyloarthrosis Diagnosis



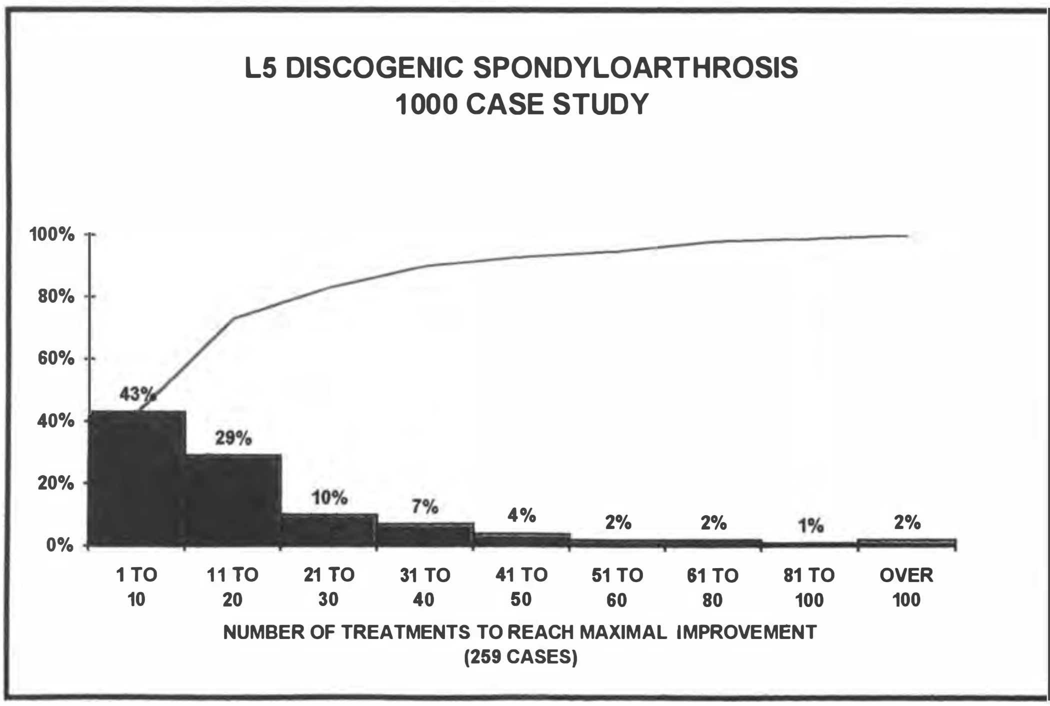


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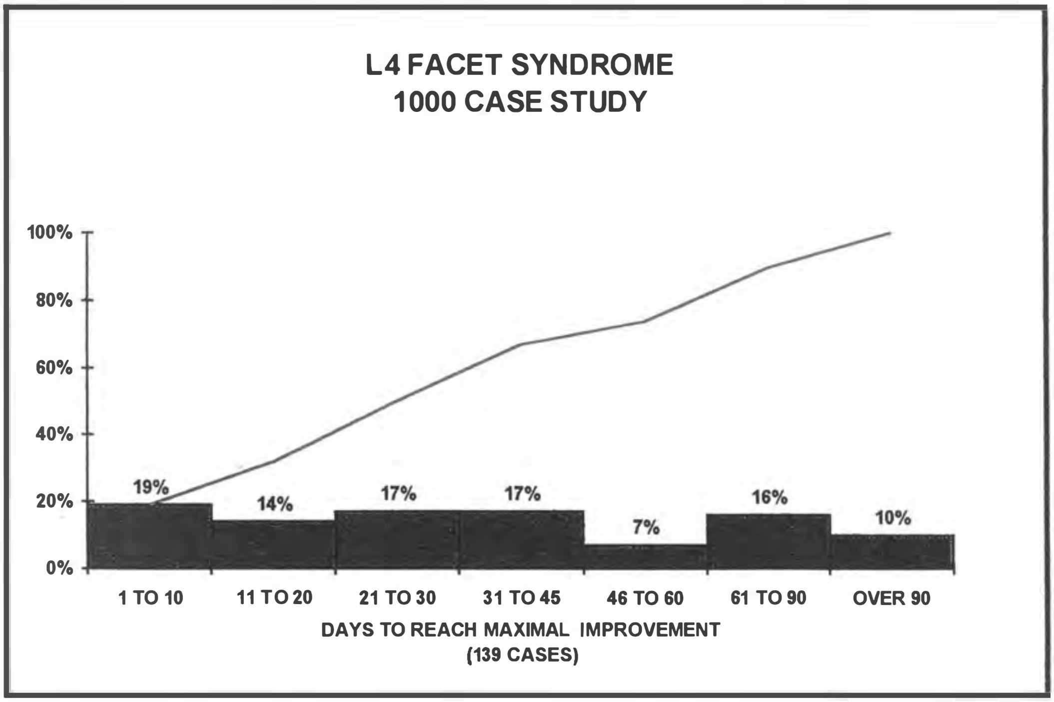
Table 9.9

Days and Treatments to Maximal Improvement of L4 Facet Syndrome Diagnosis



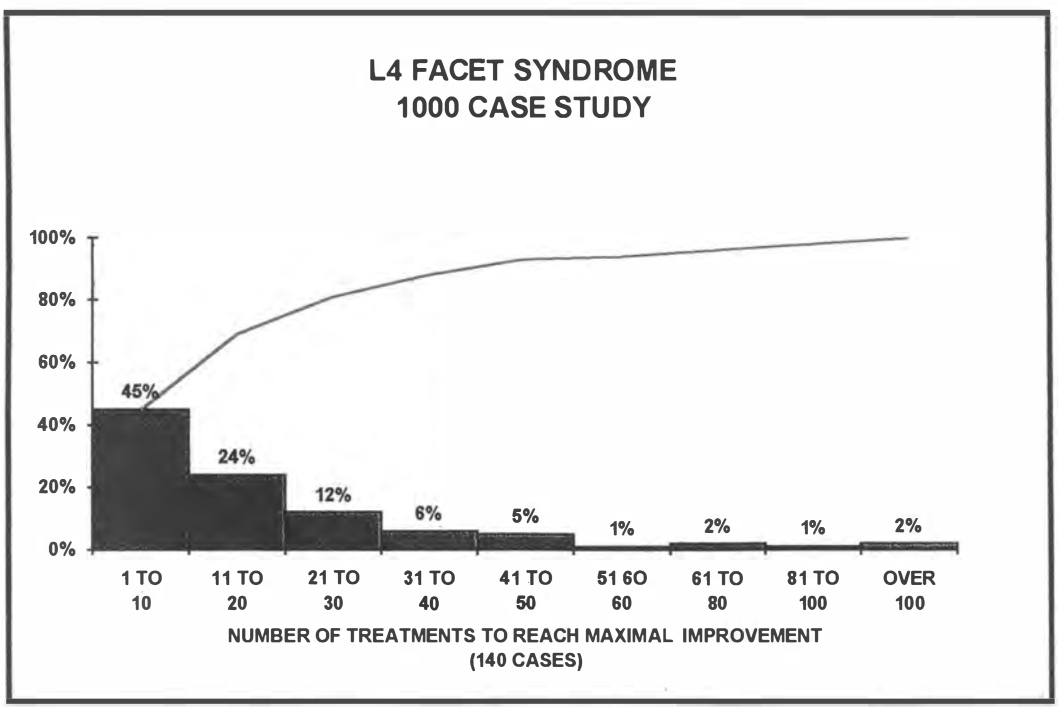


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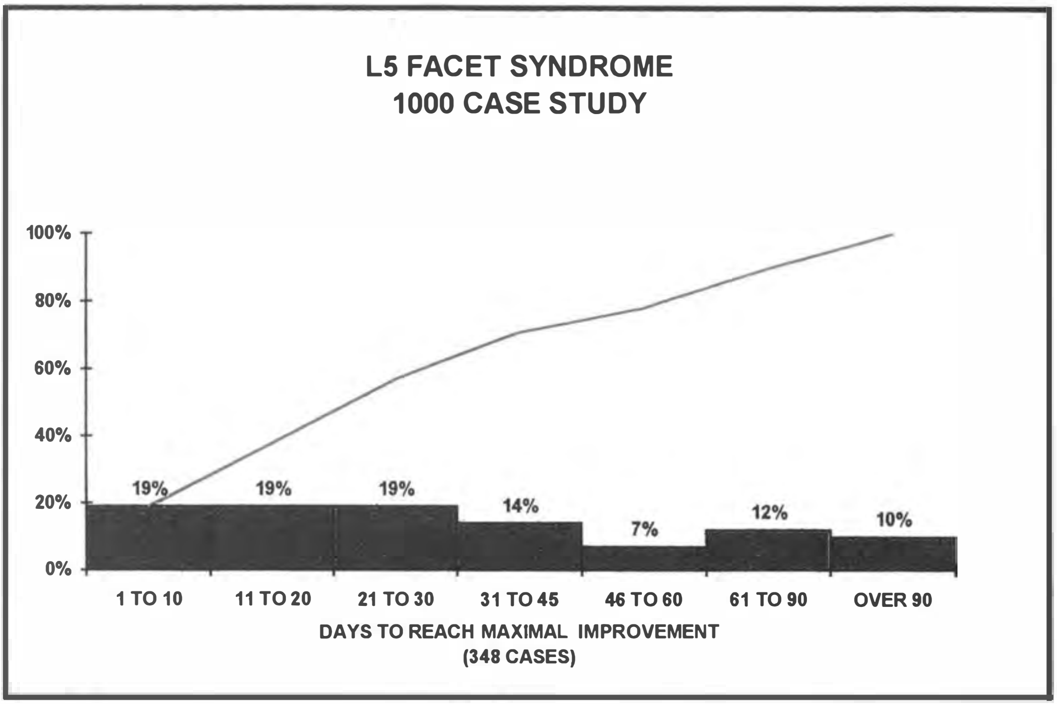
Table 9.10

Days and Treatments to Maximal Improvement of L5 Facet Syndrome Diagnosis



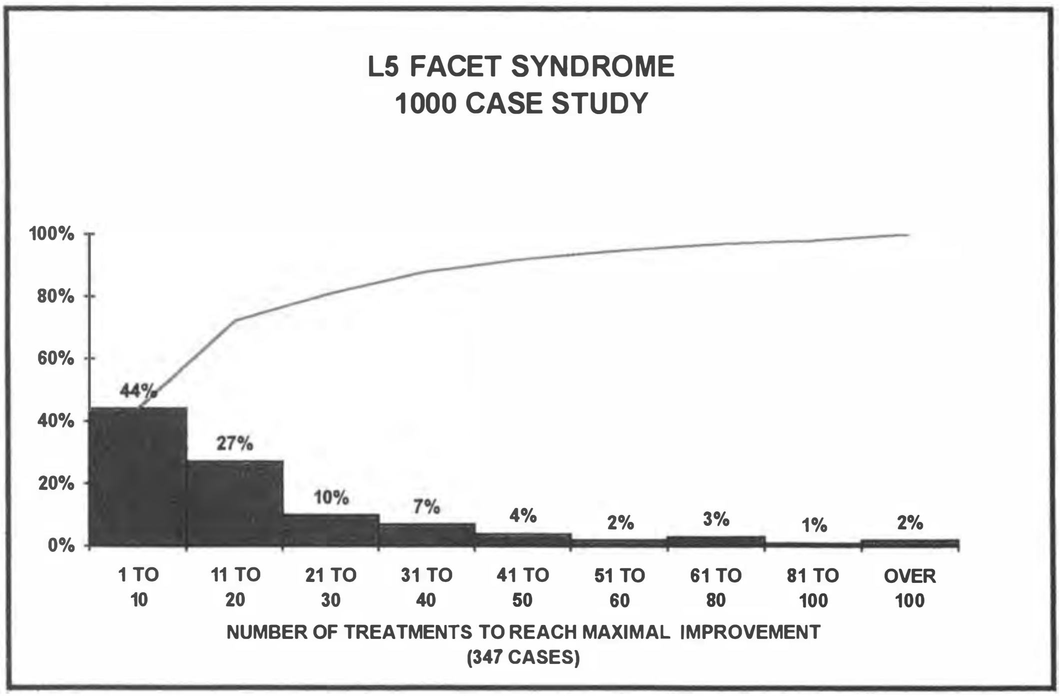


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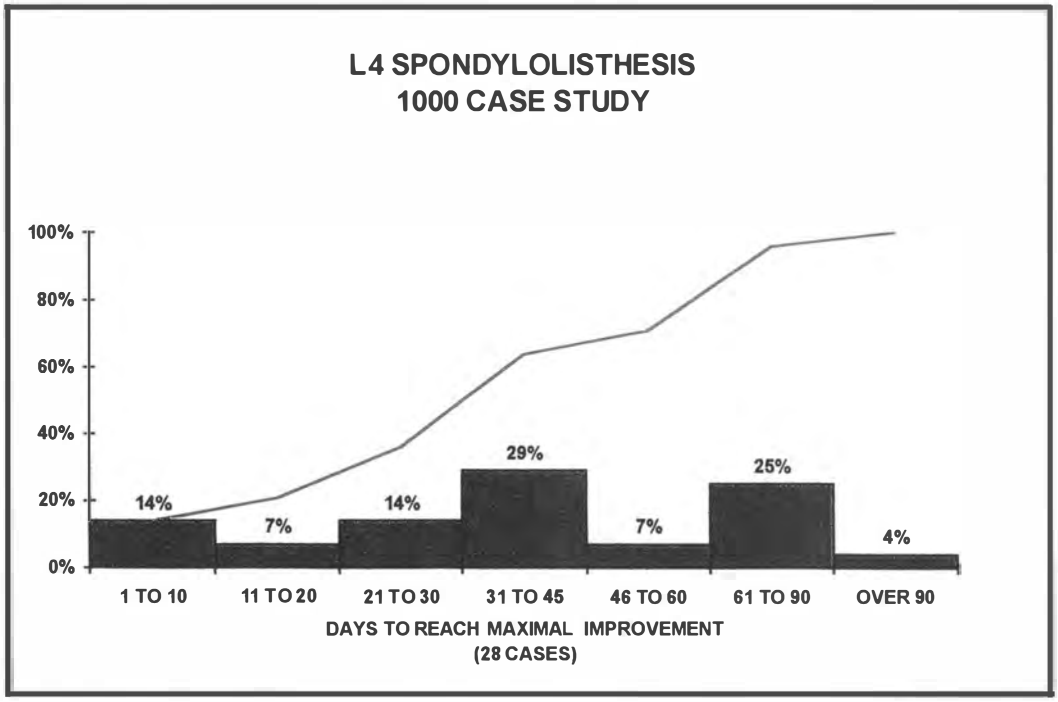
Table 9.11

Days and Treatments to Maximal Improvement of L4 Spondylolisthesis Diagnosis



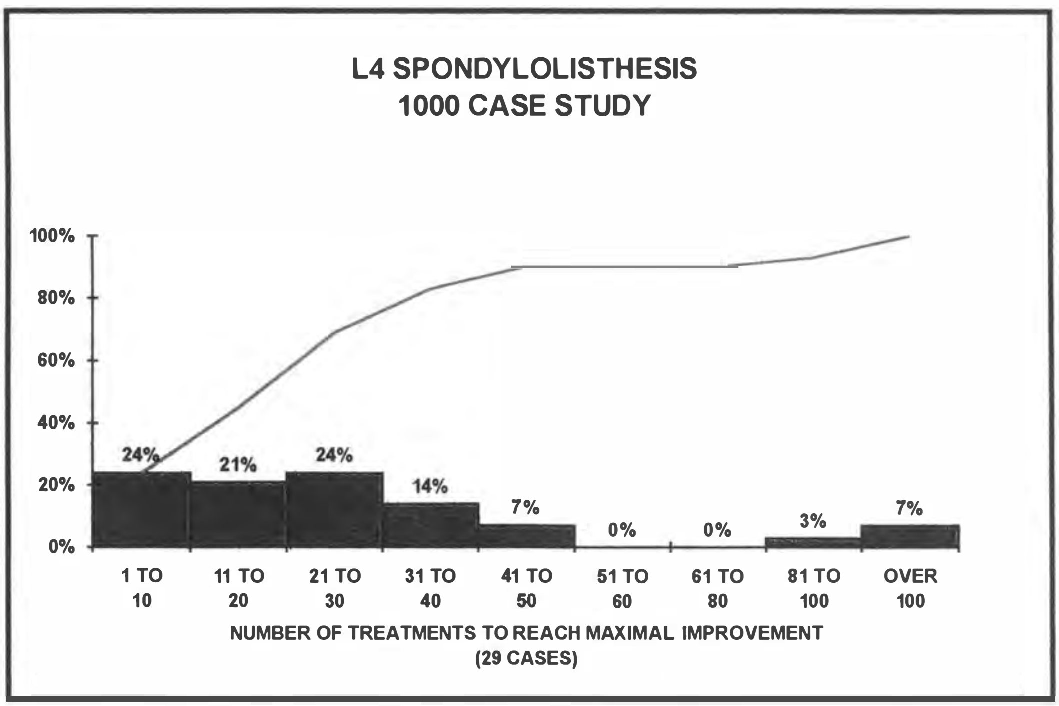


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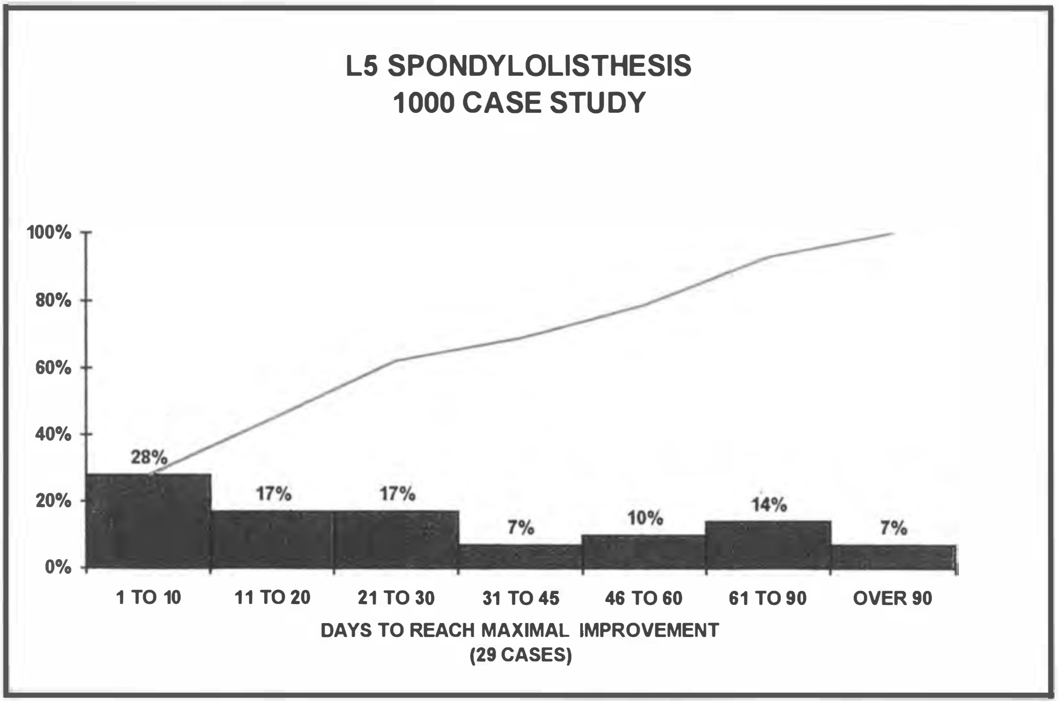
Table 9.12

Days and Treatments to Maximal Improvement of L5 Spondylolisthesis Diagnosis



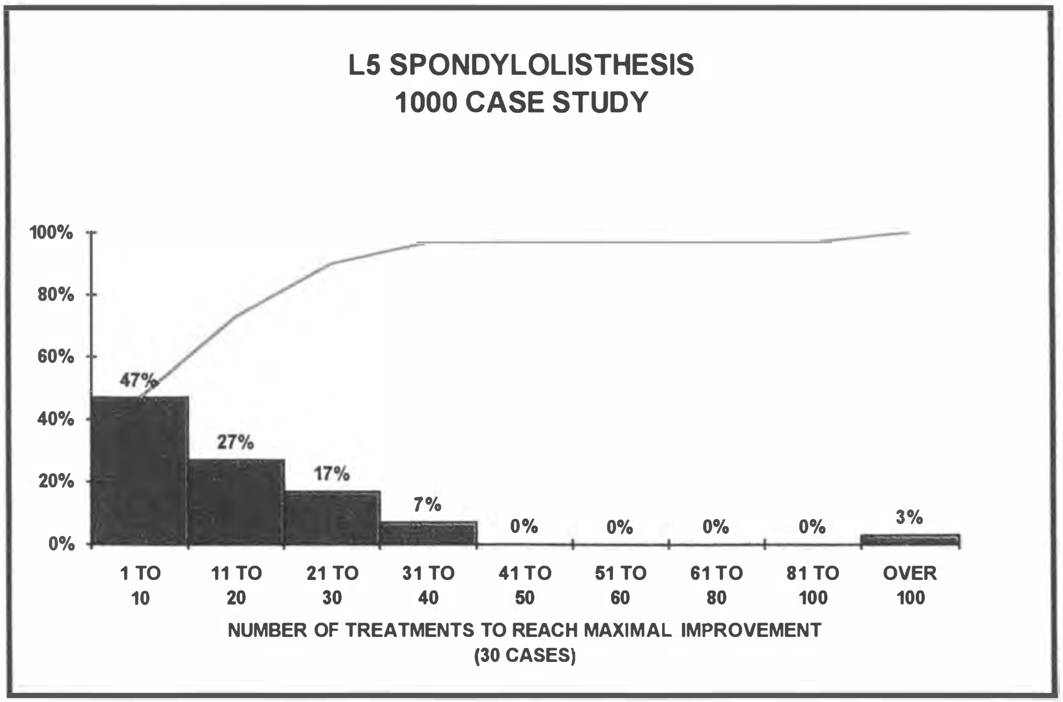


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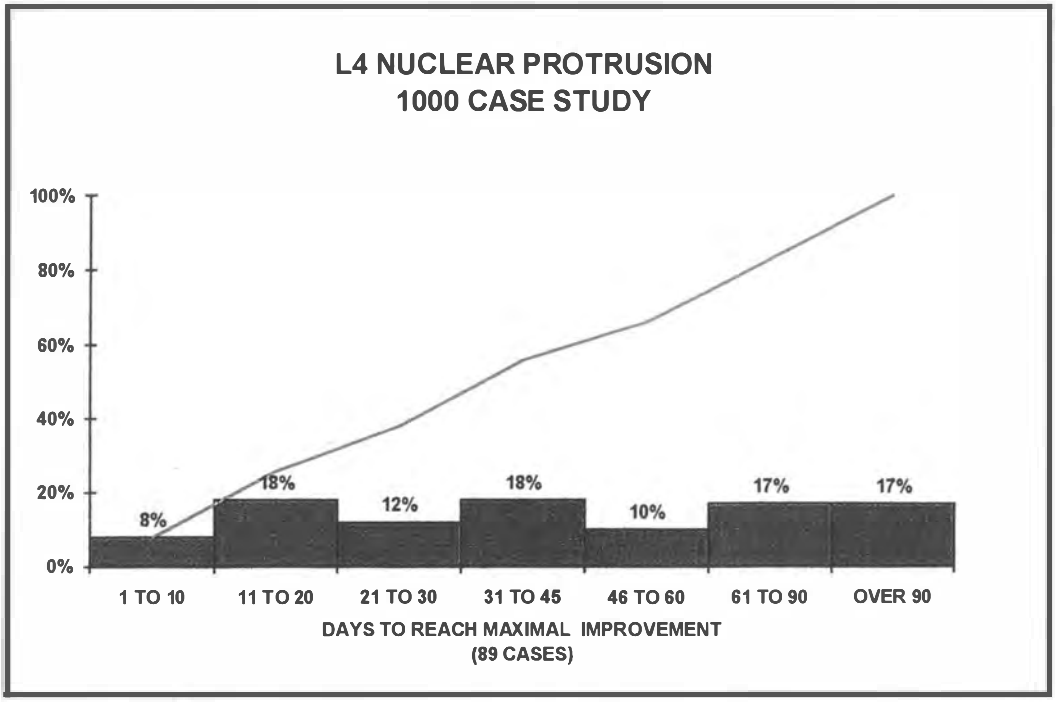
Table 9.13

Days and Treatments to Maximal Improvement of L4 Nuclear Protrusion Diagnosis



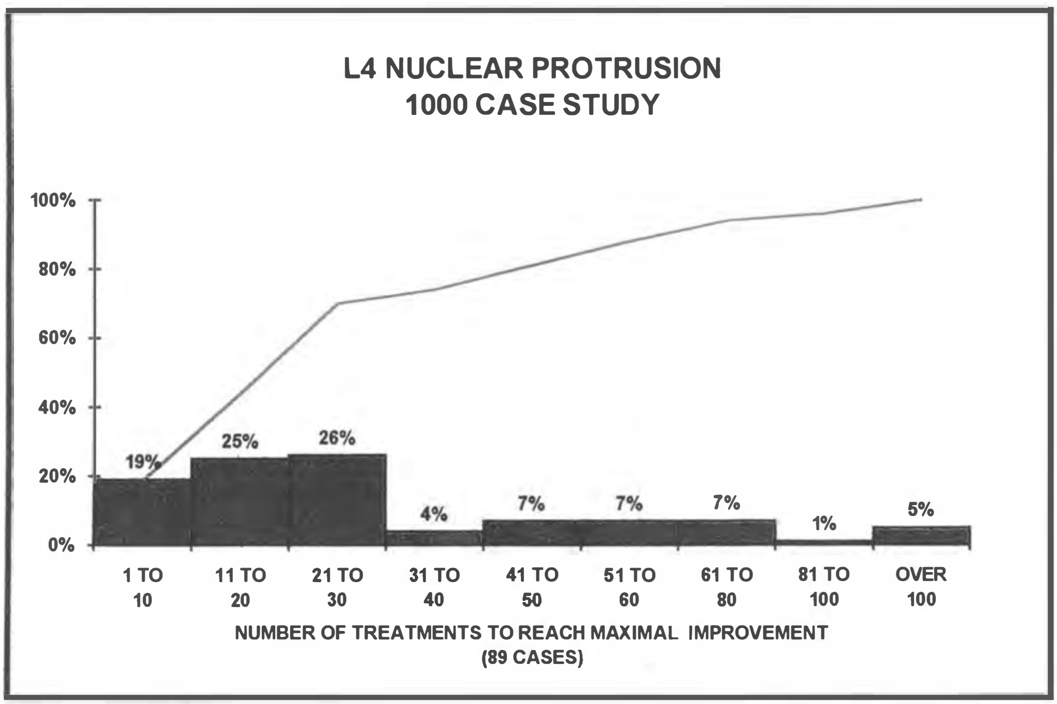


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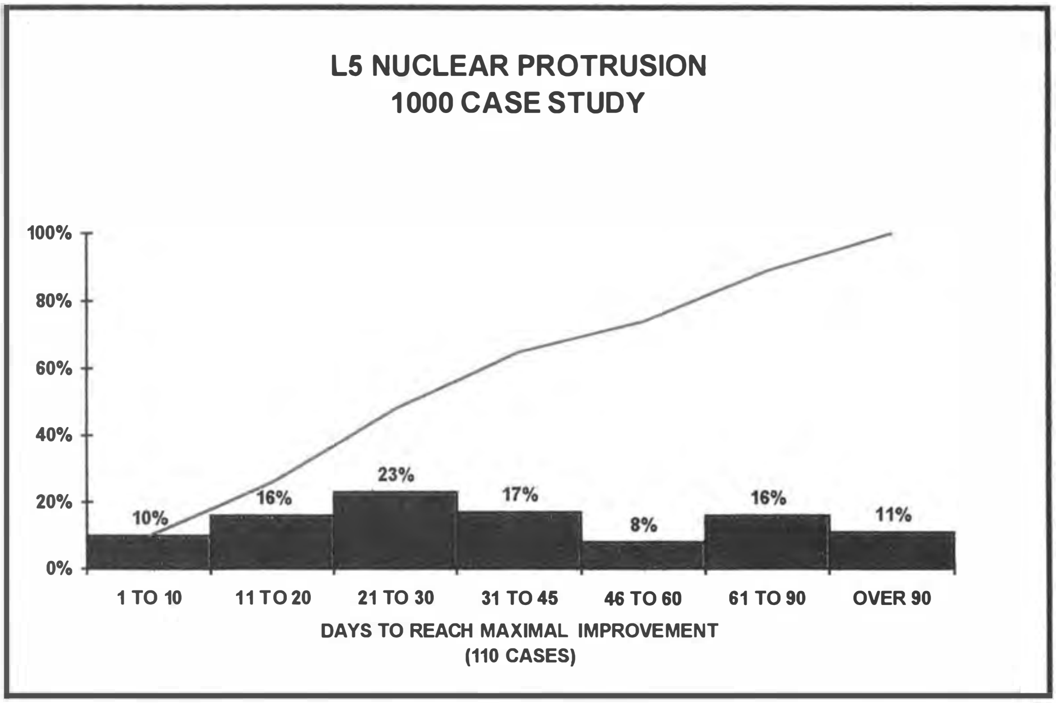
Table 9.14

Days and Treatments to Maximal Improvement of L5 Nuclear Protrusion Diagnosis



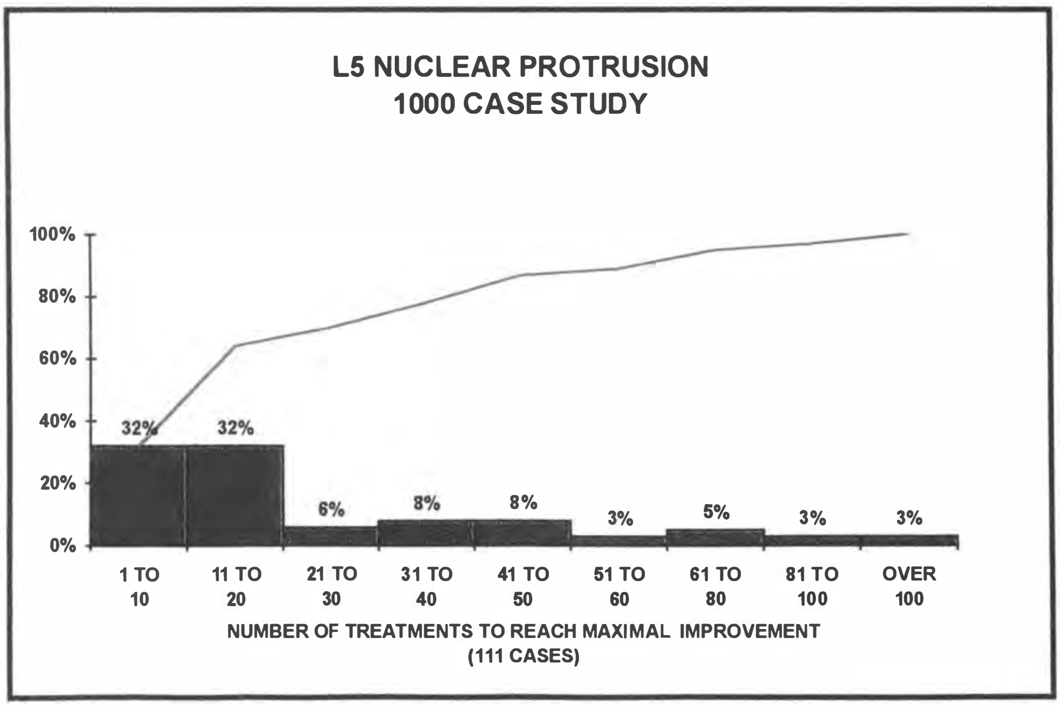
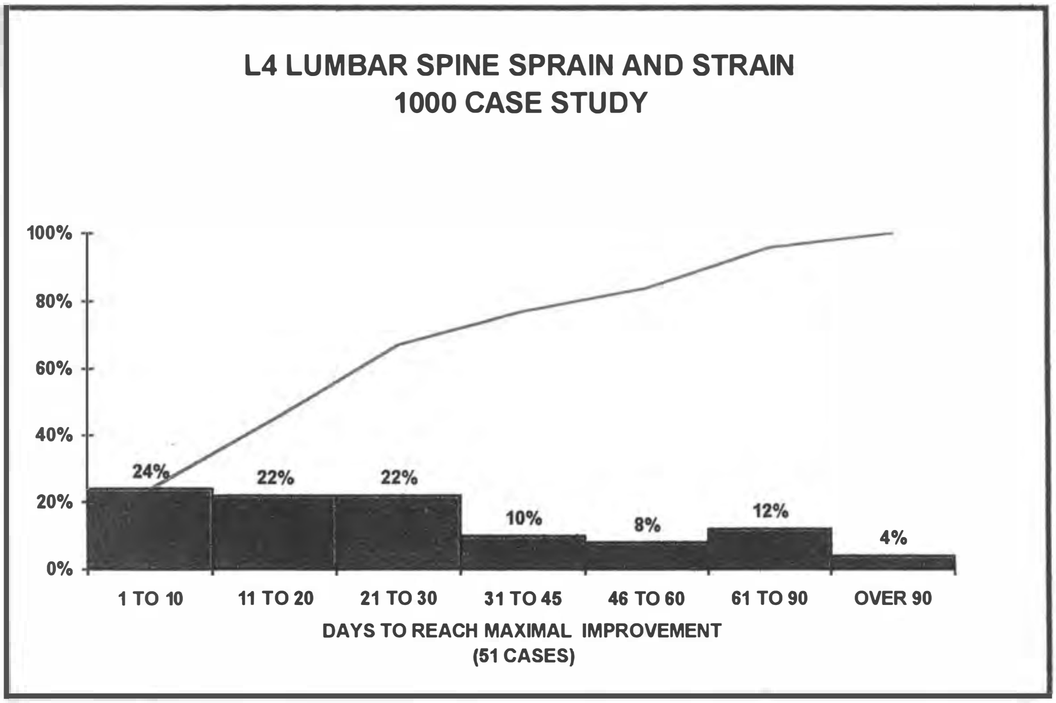


Table 9.15

Days and Treatments to Maximal Improvement of L4 Sprain/Strain Diagnosis



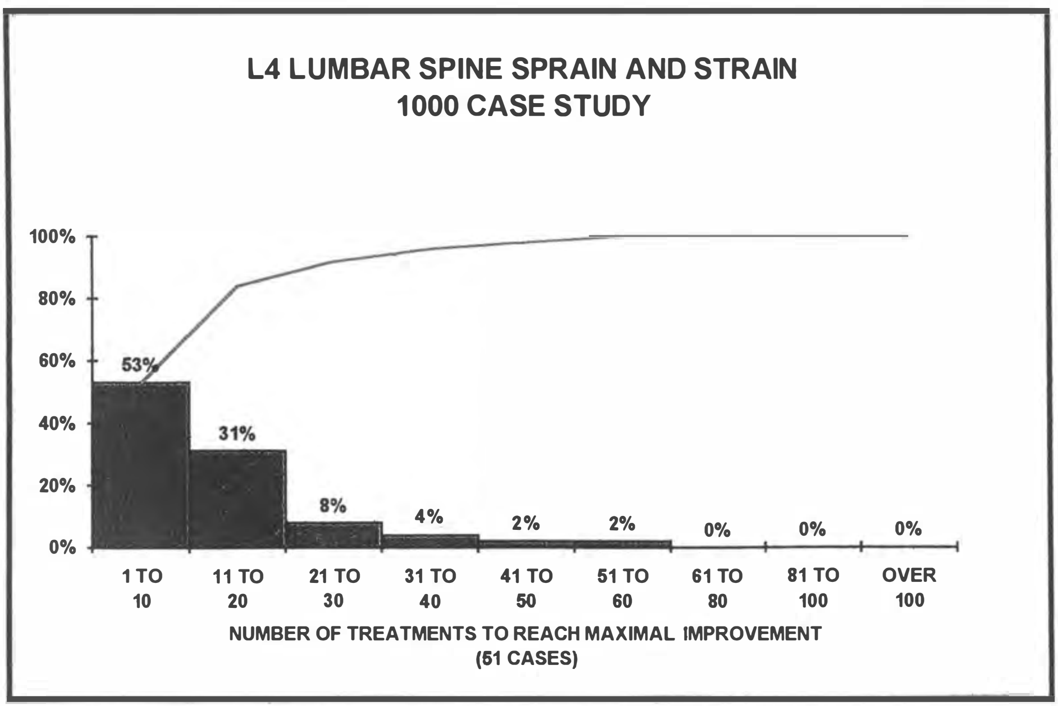


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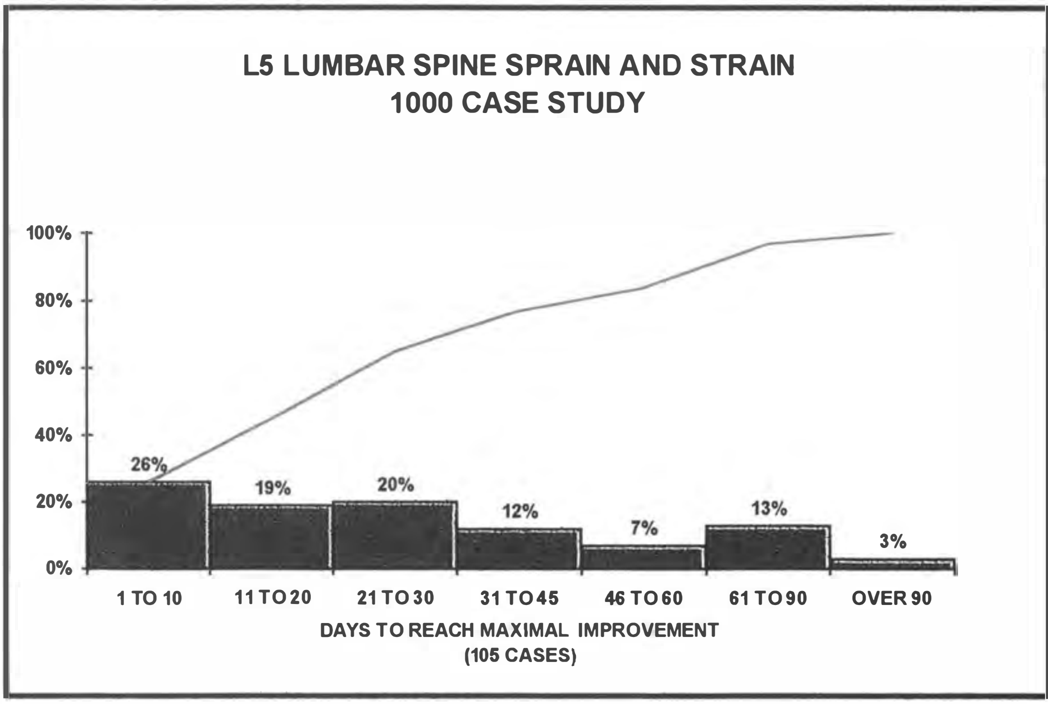
Table 9.16

Days and Treatments to Maximal Improvement of L5 Sprain/Strain Diagnosis



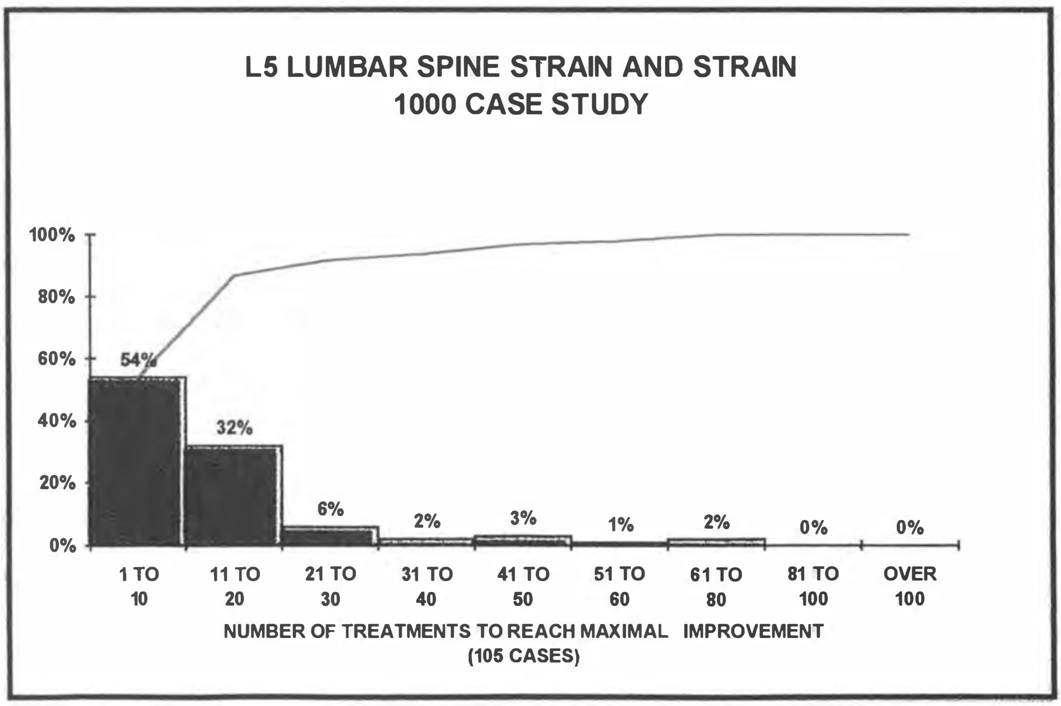


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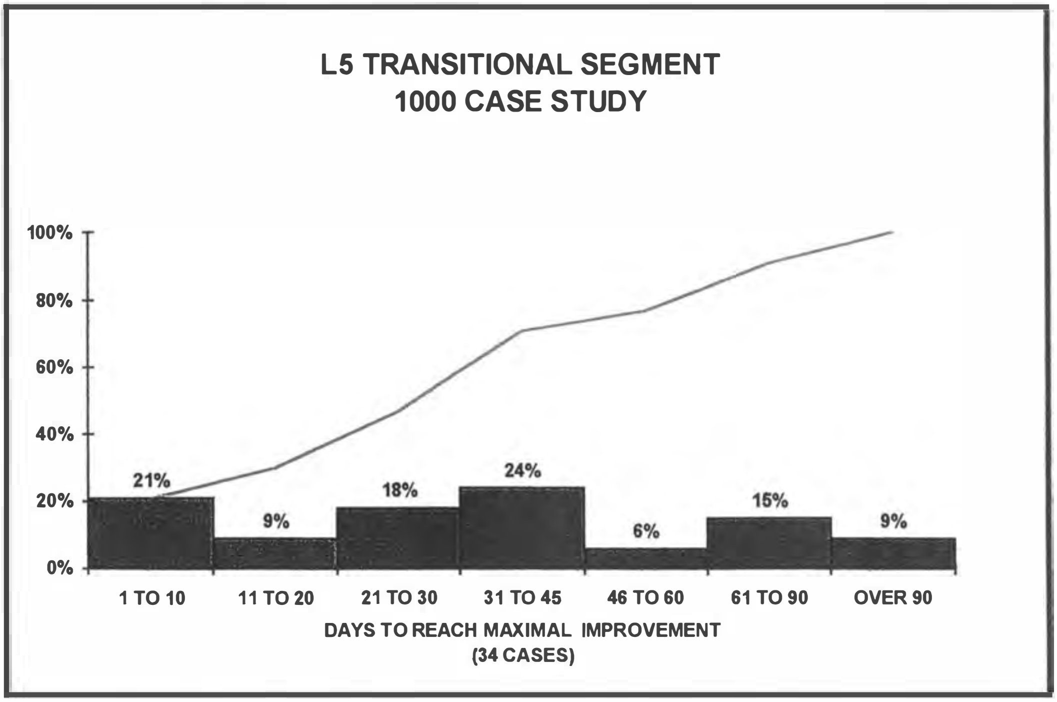
Table 9.17

Days and Treatments to Maximal Improvement of L5 Transitional Segment Diagnosis



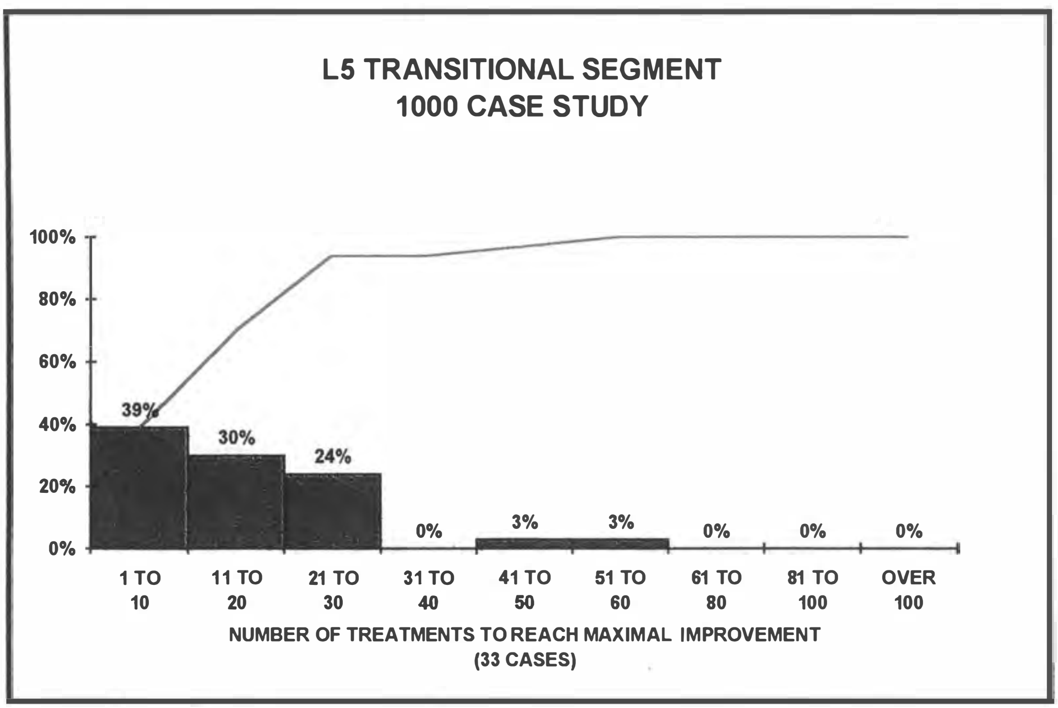


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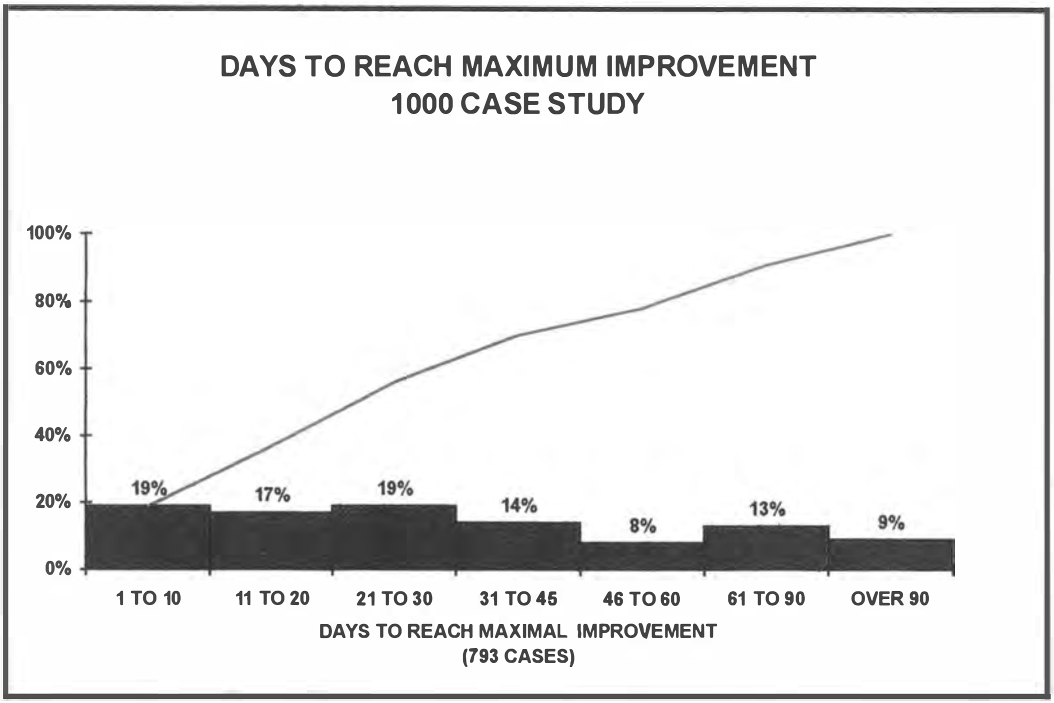
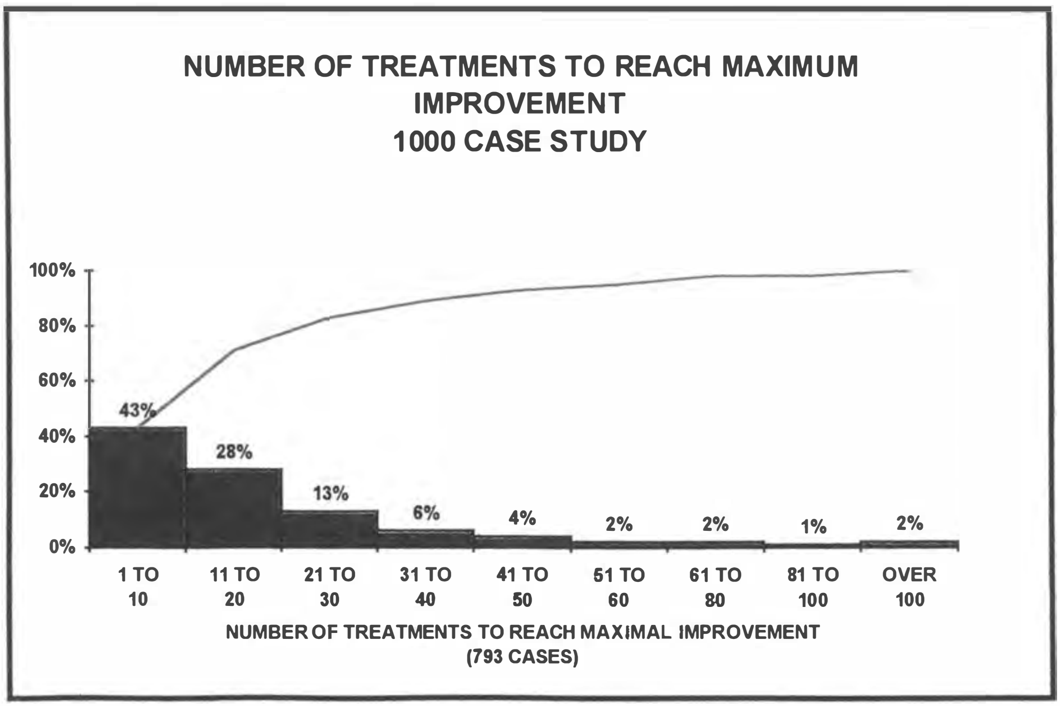
Table 9.18

Days and Treatments to Maximal Improvement Regardless of the Diagnosis





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required more than 30 visits in this study (Table 9.6). A small percentage of patients do respond positively to chiropractic care, but they may require more than 40, 50, 60, or even 100 visits (Table 9.2). All involved—patient, family, employer, insurance company, doctor—must understand that every patient's condition is unique, and the treatment regimen must be just as individualized. Not every condition will respond in the mean 12 visits in 29 days.

Tables 9.7 to 9.18 are graphs of specific diagnoses of low back pain patients showing the number of days and treatments for each condition to attain maximal improvement.

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PRE- AND POSTDISTRACTION ADJUSTMENT CARE

Acupressure and Trigger Point Therapy (Fig. 9.60)

A deep goading pressure is applied as shown in Figures 9.61 and 9.62 in preparation for distraction. The goading pressure is applied over the paravertebral areas of the upper lumbar spine through the coccyx. These areas coincide with bladder meridian points B24 through B35 at the coccyx.

Goading is then applied into the belly of the gluteus maximus muscle (Fig. 9.63). Further information on the treatment of this muscle is given in Figure 9.110. The gluteus maximus is supplied by the inferior gluteal nerve, which has a common spinal origin with the sciatic nerve. The pain and spasm of the gluteus maximus muscle will recede as the disc lesion heals and the sciatic nerve is relieved. Therefore, a deep goading pressure is placed into the belly of this muscle for 15 to 20 seconds both before and after treatment. The relaxation and loss of pain in this muscle is an indicator of patient response.

Next, the gluteus medius and minimus muscles are goaded (Fig. 9.64) at their origin and insertion prior to distraction. These abductor muscles of the lower extremity are painful when palpated and they are usually weak in muscle testing.

Bladder meridian point B54 in the popliteal space is goaded vigorously for 15 to 20 seconds (Fig. 9.65). This point is used in acupuncture to relieve sciatic pain.

Goading of the adductores and gracilis muscles at their origins and insertions is shown in Figures 9.66 and 9.67. These muscles are supplied by the obturator nerve from the second, third, and fourth lumbar nerve roots. They are extremely tight and painful in the patient with a disc lesion. These muscles are also discussed in a later chapter on muscle treatment.

Acupuncture Meridian Tracing

Identification of acupuncture points and meridians was reported by observing the migration of a radioactive tracer, ^{99m}Tc injected subcutaneously in a volume of 0.5 mL using a hypodermic needle of 0.5 mm, at an average depth of 3 to 5 mm. When the injection is made at an acupuncture point, the migration shows the following characteristics: It begins after a mean delay of 2 minutes after injection; it is carried out from the very first injection site along an axial course immediately visible on the control screen; and, using a probe with a radioactive tip, the observed course is followed through different reference points with a line traced on the skin that corresponds to the acupuncture meridian at a point where the technetium was injected. The 12 classically described meridians have been noted by Darras et al. (1).

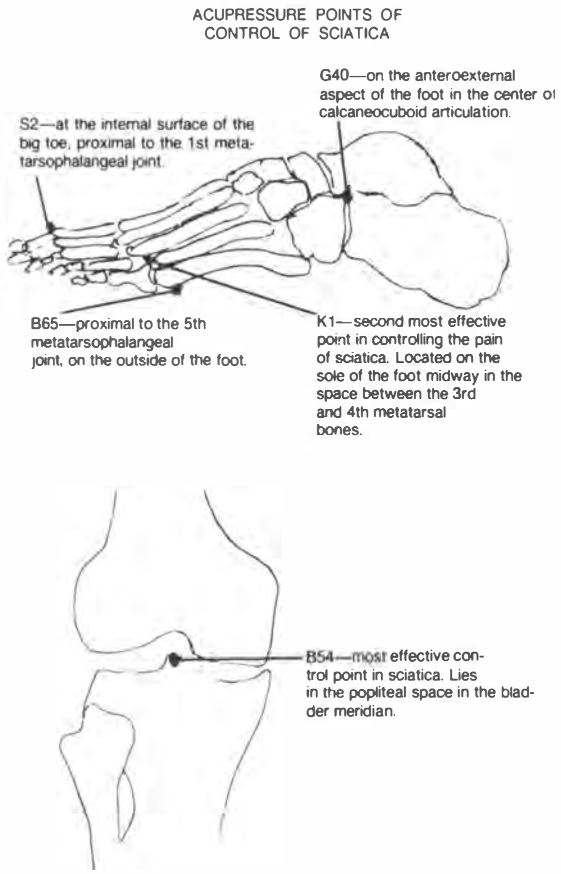

Figure 9.60. Acupressure point therapy for sciatica.

Figure 9.61. Paravertebral bladder meridian acupressure points being goaded.

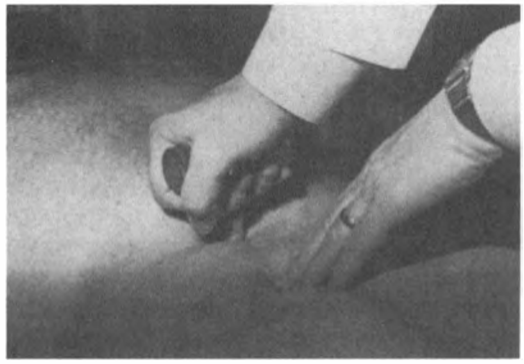

Figure 9.62. Deep pressure is applied with an instrument for ease of application.

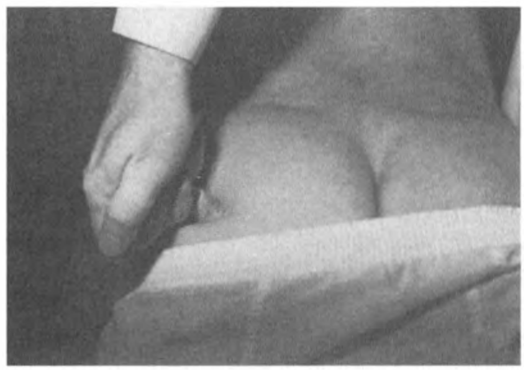

Figure 9.63. Gluteus maximum B49 acupressure point being goaded.

Figure 9.64. Abductor muscle origin and insertion pressure applied.

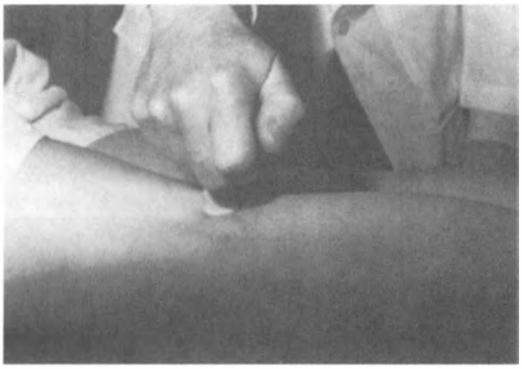

Figure 9.65. Bladder meridian point B54 being goaded.

Figure 9.66. Pressure goading of the adductores muscle origins.

Figure 9.67. Pressure goading of the adductor and gracilis insertions.

ELECTRICAL STIMULATION

Positive Galvanic and Tetanizing Currents

Following distraction manipulation, the muscles and acupressure points shown in the section on acupressure and trigger point therapy (Fig. 9.60) are treated again.

Physical therapy in the form of positive galvanic current and hot and cold therapy is applied to the involved disc and acupressure points, as shown in Figures 9.68 through 9.71. One positive pad is placed directly over the disc protrusion with the negative pad next to it, and the other positive pad can be placed on the gluteal region to sedate the sciatic nerve there, or it can be placed over B54 in the popliteal space with the negative pad opposite to it (Figure 9.72). The benefits of galvanic current are given as follows.

Galvanism is a continuous, waveless, unidirectional current of low voltage commercially called "direct current." Galvanic current is decidedly chemical in action and, as it passes through the body, it breaks up some of the molecules it encounters into their component atoms or *ions* as they are more properly called. All ions have either a *positive* or *negative* electric charge and attract or repel each other, with *like* charges repelling and *unlike* charges attracting. When two dissimilar ions unite, a neutral molecule is formed, but when the galvanic current

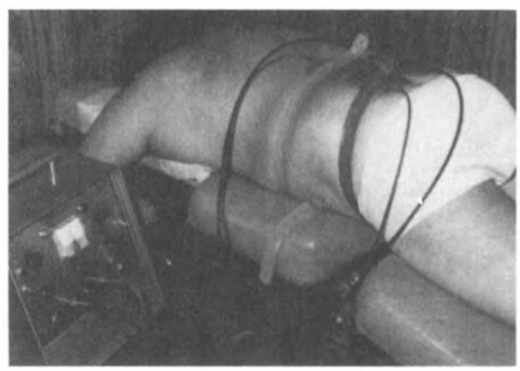

Figure 9.68. Positive galvanism is applied to the disc as the patient lies prone.

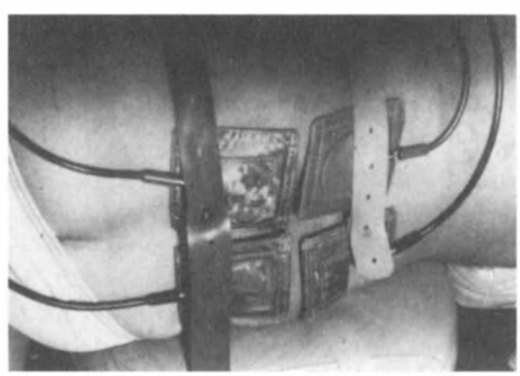

Figure 9.69. Positive galvanism is applied to the disc as the patient lies on the side.

Figure 9.70. Moist heat is applied to the low back and extremity.

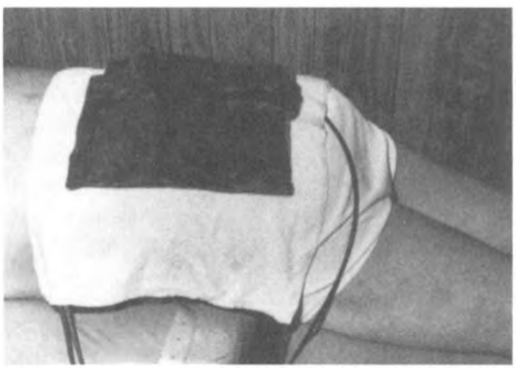

Figure 9.71. Cryotherapy is applied to the low back as galvanic or tetanizing currents are applied.

breaks this union, the original positive and negative ions are liberated. Table 9.19 outlines the action produced at the respective poles.

The active pole, either positive or negative, is the one that produces the effects desired. The other is the inactive or indifferent pole. The active pole should be the smaller in order to concentrate the current locally and thus intensify the action.

The number of milliamperes to be used depends on the smoothness of the current and the susceptibility of the patient, with from 5 to 20 mA being the average. Treatment length is determined by the milliamperes used, with from 5 to 15 minutes usually being sufficient time for application of the current.

Application Rules

1. Caution should be used to prevent galvanic burns.
2. Never dispute the patient. If he or she complains, investigate.
3. Be careful with paralyzed patients.
4. Avoid shocks.
5. See that the pads are properly placed (i.e., active and indifferent).
6. See that the intensity control is completely turned off before placing the pads.
7. Do not place or remove the pads while the instrument is running.
8. Be sure to have pads thoroughly moist but not dripping wet.
9. Turn current on and off slowly.
10. Have the patient remove sufficient clothing for exposure and protect the remainder from getting damp.
11. Never change poles while the current is flowing, except when testing.
12. Protect scars or wounds.

Remember: *Positive* ions are driven in under the positive pole. *Negative* ions are driven in under the negative pole.

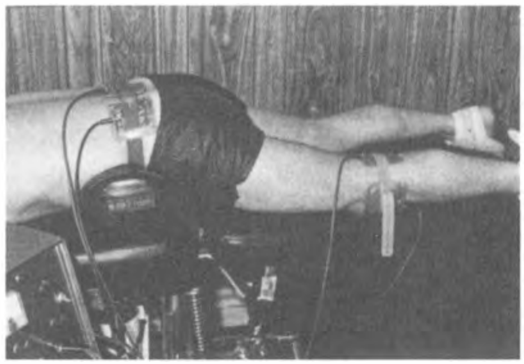

Figure 9.72. Positive galvanism being applied.

Table 9.19

Actions Produced by Galvanic Current

Positive	Negative
Attracts acids	Attracts alkali
Repels alkali	Repels acid
Hardens tissue	Softens tissue
Contracts tissue	Dilates tissue
Stops hemorrhage	Increases hemorrhage
Diminishes congestion	Increases congestion
Sedative	Stimulating
Relieves pain in acute conditions due to reduction of congestion. If scar is formed, it is hard and firm.	Reduces pain in chronic conditions due to softening of tissues and increase of circulation. If scar is formed, it is soft and pliable.

Polarity

The most important feature of the galvanic current is its *polarity*, with each pole having distinctive attributes and, consequently, being productive of certain specifically definite therapeutic effects. The action of one pole is opposed to that of the other. Polarity must be well understood. The direct current (DC) decomposes liquid as it passes through it. This decomposing of a liquid by an electric current is termed "electrophoresis." The liquid decomposed is the "electrolyte," and the parts of the separated electrolyte are the "ions." The current enters the electrode by the *anode (positive pole)* and leaves by the *cathode (negative pole)*.

There are positive ions and negative ions. Those ions possessing an excess negative charge are termed "electronegative," and those possessing an excess positive charge are termed "electropositive." It is a universal law of electrical physics that like poles repel and unlike poles attract; therefore, negative ions travel toward the positive pole and positive ions travel toward the negative pole. Oxygen, being electronegative, is repelled from the negative pole and forms at the positive pole; hydrogen, being electropositive, is repelled from the positive pole and collects at the negative pole. Consequently, when we treat a pain, we use the *positive* pole over the seat of pain because the positive pole is a sedative and is acidic in reaction. We desire this reaction because where pain exists, an alkaline reaction occurs, and by using the positive pole the alkalinity is driven toward the negative pole.

The slogan for pain is *positive pole;* however, there are exceptions. For instance, if inflammation has been sufficiently prolonged to cause distinct organic tissue changes (fibrosus, adhesions) that, in turn, causes pain on motion of the parts involved, the *negative* pole is used because of its liquefying and vasodilative properties.

Electrode Application

The active electrode is always the smaller of the two electrodes; the opposite electrode is known as the indifferent electrode, and it should be placed as nearly opposite to the active electrode as possible. The indifferent electrode is usually a well-moistened pad.

Electrodes must be secured or held in contact with the patient before the instrument is turned on and current is allowed to flow. Also, contact between the patient and the electrode must not be broken while the current is flowing. Lastly, the current must not be turned off until it has been reduced to zero; otherwise the patient will receive a shock.

Low Volt Galvanic Principles in Treating Low Back Pain

Low voltage galvanism is a highly effective and dependable modality that has brought about favorable clinical results after other therapies have failed. Failure of its widespread use is based on three things:

1. The existing literature on it is not extensive.
2. Confusion over the many possible variations of the low volt currents.
3. It demands more user attention to detail than other devices (2).

Application of Tetanizing Current

Figure 9.73 shows the application of tetanizing current. For muscle spasm, we use tetanizing current to the paravertebral muscles to create relaxation and positive galvanism to relieve pain and release myofascial inflammation.

Some patients are in too much pain to lie on their abdomen when treatment or therapy is being applied. We have these patients lie on their sides and apply the therapy as shown in Figure 9.74. The tetanizing current is applied and the hydrocolator or cryotherapy applied over it, with straps holding the packs in place. In clinical practice, we use alternating hot and cold therapies consisting of 10 to 15 minutes of heat followed by 5 to 10 minutes of ice, using three or four heat sessions with two or three ice sessions in between. Always begin and end with the heat, as this tends to leave the patient more relaxed.

Analgesic liniment is massaged into the lumbar spine paravertebral muscles, with emphasis on the acupressure bladder meridian points from the second lumbar paravertebral area, between the transverse and spinous processes, to the coccyx, which is bladder point B35 (Fig. 9.75).

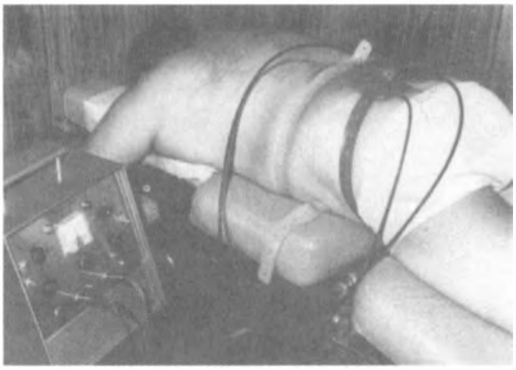

Figure 9.73. Application of electrical stimulation following manipulation.

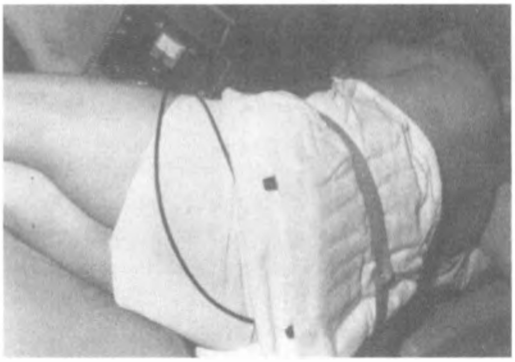

Figure 9.74. When in too much pain to lie on the abdomen, the patient may lie on the side and have therapy applied.

Figure 9.75. Massage of acupressure points.

Caring for the Patient If Testing Elicits Pain

The most common condition in which pain is elicited on testing for tolerance to flexion distraction is the acute disc lesion in which the patient has sciatica. Second most common, in my experience, is pain in acute lumbago conditions in which severe muscle spasm and forward flexion of the lumbar spine are seen. When this negative reaction to the use of flexion distraction is found, the following treatment program is recommended: Apply positive galvanic current through the involved disc and paravertebral muscles for 15 minutes, as shown in Figure 9.68. If the patient is in too much pain to lie on the stomach, he or she may lie on the side, as shown in Figure 9.69, while the therapy is applied. During this 15-minute period, moist heat is applied to the low back and pelvis into the thigh over the course of the painful sciatic nerve (Figure 9.70). Following the heat application, remove the heat and place cold packs over the same area, as shown in Figure 9.71. Often, if severe spasm of the paravertebral muscles is found, tetanizing current is applied to the muscles while the ice is applied for relief of spasm and swelling.

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BRACING

Bracing is recommended in patients with herniated lumbar discs with radiculopathy, instability (defined in the chapter on facet syndrome), degenerative spondylolisthesis, and severe low back pain. As the patient attains 50% relief of leg pain and low back pain brace use is reduced to 50% of the time and discontinued when another 50% relief is attained. During brace use, the patient performs Cox exercises at home and the patient is encouraged to actively participate in rehabilitation, both in the clinic and at home. Recommended is the use of the wobble board, extension exercises on office machines, and other exercises to gain flexibility and strength as relief allows.

Clinical Bracing Principles

Proposed Clinical Objectives of Spinal Braces (1)

- Protection of injured tissues to improve healing
- Protection of spinal cord and nerve roots after unstable fracture or dislocation
- Decrease pain
- Prophylaxis against reinjury or new injury
- Correction of spinal deformity
- Facilitate early reactivation after back injury

Mechanical Principles of Spinal Orthotics

- Three-point fixation with sensory feedback
- Indirect transfer of load
- Direct transfer of load
- Insulation
- Stored energy

Orthotics Effects on Spinal Biomechanics

- Restriction of motion (gross motion versus intersegmental) (1, 2)
- Influence posture
- Redistribution of load within a segment
- Decrease total spinal load
- Change of temperature of the superficial structures of the trunk
- Decrease work of trunk muscles

Ninety-nine percent of 3410 orthopedic surgeons surveyed in the United States reported prescribing spinal orthoses. Patient acceptance and symptom improvement are seen in 30 to 80% of cases (3). The lower lumbar region is of particular interest because most disorders occur in one or both of the lower two segments. Placing a back support on a patient often assists him or her in early return to full function, and it helps avoid the well-documented harmful effects of prolonged immobilization and inactivity (3).

Indications for Lumbosacral Supports and Braces (3)

- Anyone needing to avoid compressing forces on the spine
- Pain, muscle guarding, and spasm
- Acute sprains and strains
- Congenital or traumatic joint instability (the patient should be advised to use the support only when needing the protection)
- Herniated disc protrusion (spinal supports have been

shown to reduce the intradiscal pressure in the lumbar disc by 25% in both the sitting and the standing positions)
- Postural backache
- Degenerative joint or disc disease
- Preventive measure

Types of Lumbosacral Supports and Braces (1, 3)

Lumbosacral Corset

Lumbosacral corsets are usually sized according to hip measurement; they are higher in the back than front, usually made of canvas, and they produce a semirigid cylindrical three-point fixation (Fig. 9.76).

Semirigid Lumbosacral Brace

The chairback brace consists of two upright, pelvic and thoracic bands posteriorly, and a pie pan abdominal support anteriorly. These braces effectively restrict movement in the thoracolumbar region, but are not necessarily effective in restricting mobility in the lumbosacral area (Fig. 9.77).

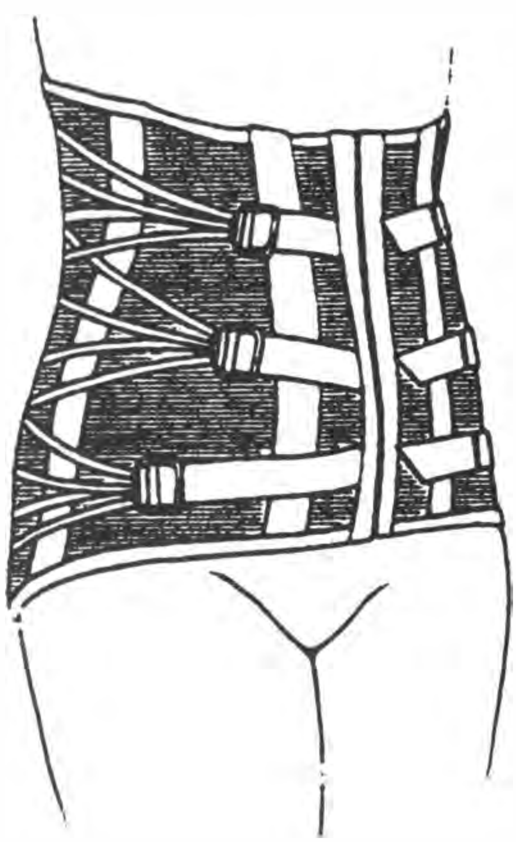

Figure 9.76. Lumbosacral corset. (Reprinted from Goldish GD. Introduction: Lumbar spinal orthotics. Journal of Back and Musculoskeletal Rehabilitation 1993;3(3):1–11; with permission from Elsevier Science Ireland Ltd., Clare, Ireland.)

Lumbar Support or Belt

Most lumbar supports or belts are made of fabric and elastic with Velcro and buckle closures. They are fitted by waist size. Their main benefit is to remind the patient to practice proper posture and body mechanics while increasing intra-abdominal pressure. See Figure 9.78.

Thoracolumbosacral Orthosis

A thoracolumbosacral orthosis (TLSO) has straps that extend around the shoulders from the corset which covers the thoracic spine to the sacrum. Figure 9.79 through 9.82 show various types of TLSOs. The Taylor brace (Fig. 9.80) is a chairback brace converted to a TLSO. The Jewett brace (Fig. 9.81) has sternal and pelvic pads anteriorly and posteriorly at

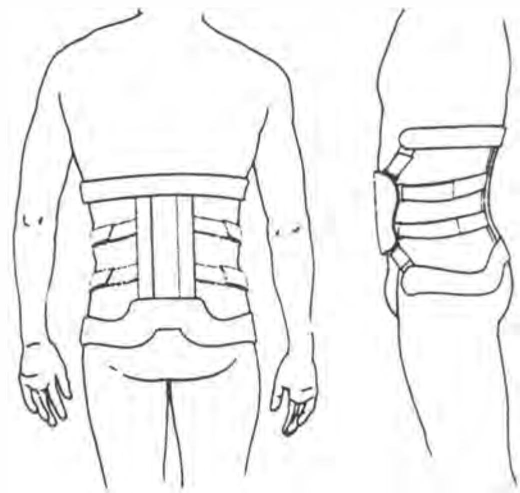

Figure 9.77. Rigid Chairback brace lumbosacral orthosis. (Reprinted from Goldish GD. Introduction: Lumbar spinal orthotics. Journal of Back and Musculoskeletal Rehabilitation 1993;3(3):1–11; with permission from Elsevier Science Ireland Ltd., Clare, Ireland.)

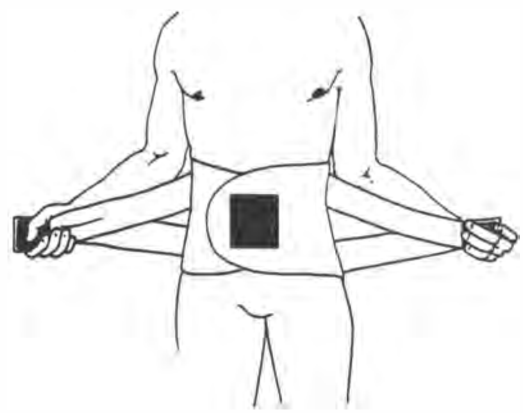

Figure 9.78. Lumbosacral flexible binder. (Reprinted from Goldish GD. Introduction: Lumbar spinal orthotics. Journal of Back and Musculoskeletal Rehabilitation 1993;3(3):1–11; with permission from Elsevier Science Ireland Ltd., Clare, Ireland.)

Figure 9.79. Thoracolumbosacral orthosis corset. (Reprinted from Goldish GD. Introduction: Lumbar spinal orthotics. Journal of Back and Musculoskeletal Rehabilitation 1993;3(3):1–11; with permission from Elsevier Science Ireland Ltd., Clare, Ireland.)

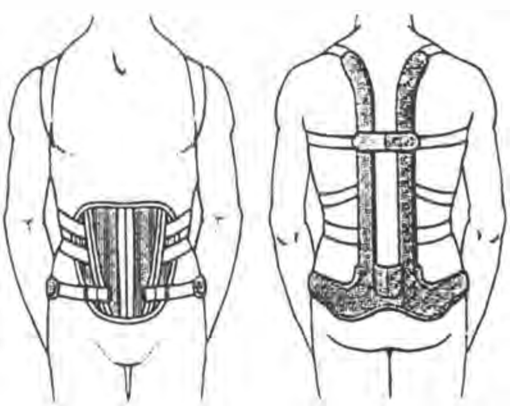

Figure 9.80. Taylor brace. (Reprinted from Goldish GD. Introduction: Lumbar spinal orthotics. Journal of Back and Musculoskeletal Rehabilitation 1993;3(3):1–11; with permission from Elsevier Science Ireland Ltd., Clare, Ireland.)

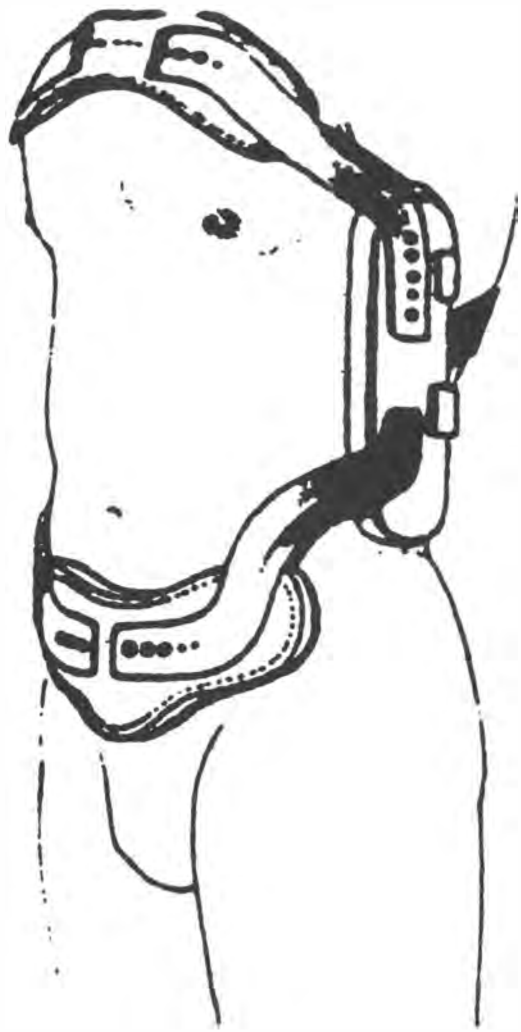

Figure 9.81. Jewett thoracolumbosacral orthosis. (Reprinted from Goldish GD. Introduction: Lumbar spinal orthotics. Journal of Back and Musculoskeletal Rehabilitation 1993;3(3):1–11; with permission from Elsevier Science Ireland Ltd., Clare, Ireland.)

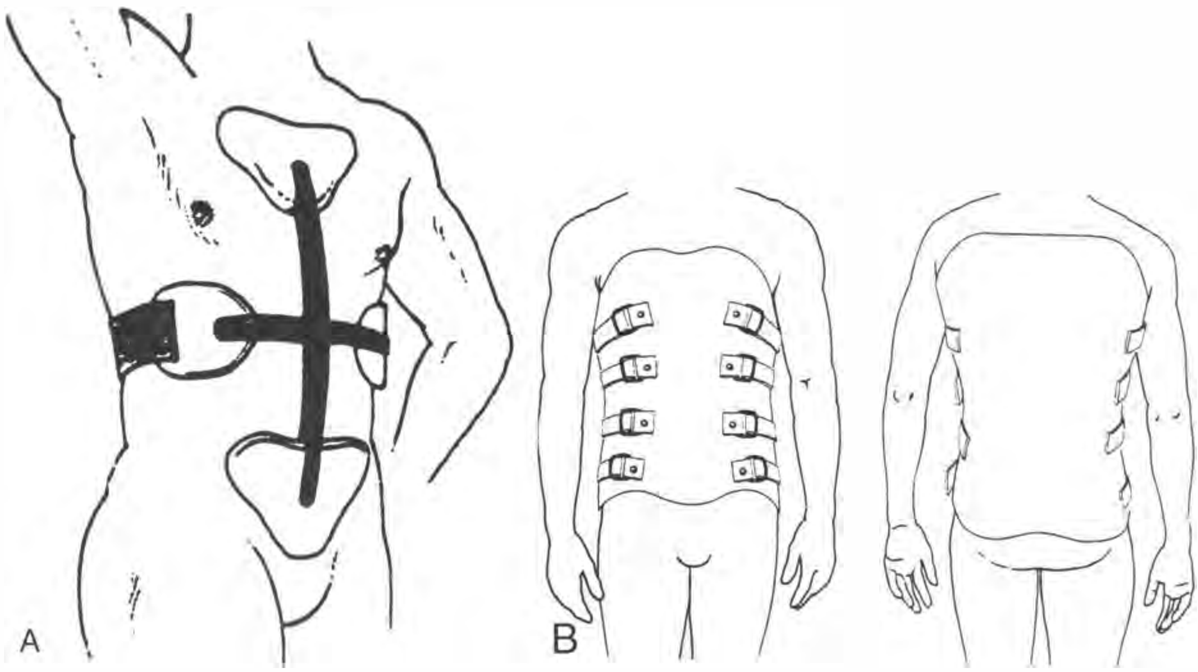

Figure 9.82. **A.** CASH (cruciform brace) thoracolumbosacral orthosis. **B.** Custom molded plastic thoracolumbosacral orthosis (TLSO). (Reprinted from Goldish GD. Introduction: Lumbar spinal orthotics. Journal of Back and Musculoskeletal Rehabilitation 1993;3(3):1–11; with permission from Elsevier Science Ireland Ltd., Clare, Ireland.)

the thoracolumbar junction pad that create a three-point fixation. The CASH (cruciform) brace (Fig. 9.82*A*) utilizes anterior sternal and suprapubic pads, which form an effective deterant to motion, but cause discomfort for the patient. Figure 9.82*B* is a plaster-fabricated TLSO body jacket. Lumbosacral orthosis have a positive effect by restricting gross motions of the trunk rather than intervertebral mobility in the lumbar spine (2).

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Bracing

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NUTRITION AND DRUG EFFECTS WITH LOW BACK PAIN PATIENTS

Medications Used for Back Pain and Their Effects (1)

Steroids (Adrenocorticosteroids)

A. Anti-inflammatory agents
B. Two categories:
   1. Mineralocorticoids (based on their sodium retention)
   2. Glucocorticoids (based on their glycogen deposition) (cortisol is the most significant glucocorticoid secreted by the adrenal medulla)
C. Act by regulating protein synthesis and controlling RNA
D. Other effects: stimulates hypertension, produces muscle wasting, and induces behavioral changes ranging from euphoria to depression and ulcers of the stomach
E. Anti-inflammatory effects are vasoconstriction to drive out inflammatory transudate and recruit white blood cells to the area of inflammation
F. Toxicity results in hypertension, Cushing's syndrome, cataracts, myopathy, ecchymosis, acne, hirsutism, avascular necrosis, infection, hyperglycemia, electrolyte disturbances
G. Contraindications: heart disease, congestive heart failure, hypertension, infections, diabetes, glaucoma, osteoporosis, psychoses, herpes simplex infection, and ulcer history

Nonsteroidal Anti-Inflammatory Medicines (NSAIDs) (1)

A. Mechanism of action: suppresses bradykinin release, alters lymphocyte response, decreases granulocyte and monocyte migration and phagocytosis
B. Indications: reduces inflammation and thereby pain
C. Classes of NSAIDs:
   1. Carboxylic acids
      a. Salicylates (acetylsalicylic acid, diflunisal)
      b. Acetic acids (indomethacin, tolmetin)

c. Propionic acids (ibuprofen, naproxen)
d. Fenamates (mefenamic acid, clonixin)
2. Pyrazoles (phenylbutazone, apazone)
3. Oxicams (piroxicam, isoxicam, tenoxicam)

D. Adverse Side Effects
1. Renal catastrophes: acute interstitial nephritis, acute tubular necrosis, nephrotic syndrome
2. Hypertension
3. Liver effects
a. Up to 5% of patients
b. Monitor liver function tests at 6 and 12 weeks
4. Bleeding due to platelet effect. Must be off NSAIDs prior to surgery
5. Gastrointestinal effects
a. 10 to 20% of patients on chronic use have ulcers
b. Hemorrhage four times greater likelihood to cause death in elderly
6. Skin rash, anaphylaxis, tinnitus, headache, confusion, agranulocytosis, asthma

Antidepressants (Tricyclic Antidepressants) (1)

A. Commonly used: imipramine, amitriptyline, doxepin, desipramine, nortriptyline, protriptyline

B. Mechanism of action: blocks the amine reuptake pump of the amine neurotransmission to potentiate the action of biogenic amines in the central nervous system. This allows longer duration of active amines in the receptor site (amine pump theory). This is felt to increase the postsynaptic response in the deficient central nervous system of patients with depression.

C. Side effects:
1. Anticholingeric effects are mydriasis, flushed dry skin, dry mucosa, absent bowel sounds, urinary retention.
2. Cardiac side effects are tachycardia and complex supraventricular tachyarrhythmias with high output cardiac failure.
3. Psychiatric disorders include delirium, anxiety, hallucinations, disorientation, seizures.

Muscle Relaxants

A. Two categories
1. Blocking the neuromuscular junction
2. Acting on the central nervous system, including carisoprodol compound (Soma Compound), Maolaate (chlorphenesin carbamate), Paraflex (chlorzoxazone), Skelaxin (metaxalone), Robaxin (methocarbamol), Norflex (orphenadrine citrate). These agents depress transmission through spinal and supraspinal polysynaptic pathways.

B. Adverse reactions: lightheadedness, dizziness, drowsiness, nausea, headache, allergic reaction. Overdose can have gastrointestinal effects such as nausea and diarrhea and vomiting.

C. Valium is a muscle relaxer that acts on the reticular neuronal mechanism controlling muscle tone. Danger exists in taking this drug with alcohol as it can be lethal if severe respiratory and neurologic depression occurs.

D. Flexeril (cyclobenzaprine) is a muscle relaxant whose action is not known. It is to be avoided in patients with heart arrhythmias or heart block because of its cardiotoxic actions (1).

Opioids

A. Opioids, including morphine, meperidine (Demerol), methadone, pentazocine (Talwin), oxymorphone (Numorphan), oxycodine and aspirin (Percodan), propoxyphene (Darvon), propoxyphene with acetaminophen (Darvocet) oxycodone and acetaminophen (Percocet), Tylenol with codeine, hydrocodone (Vicodin), and Darvon with acetylsalicylic acid

B. Opioids lessen pain without loss of consciousness

C. Central nervous system (CNS) effects: drowsiness, mood alteration, mental clouding, analgesia

D. Death occurs because of respiratory depression with overdose

E. Overdose triad: coma, pinpoint pupils, decreased respiration

Sir William Osler stated: "Imperative drugging—the ordering of medicine in any and every malady—is no longer regarded as the chief function of the doctor" (1).

Anti-inflammatory and pain-relieving drugs are needed because patients demand them. However, reducing dependency on them and striving to improve the patient's health nutritionally without them is a goal of the good doctor. The side effects of joint pain medications will be discussed in the next section.

SUMMARIES OF SPECIFIC DRUG SIDE EFFECTS ON BONE AND CARTILAGE

Prednisone

Prednisone, when taken by rheumatoid arthritis patients, had a 34% probability of causing a fracture in 5 years (2). Improvement on the natural history of sciatica with prednisone use has not been shown, and the side-effects can be substantial (3). Tetanus immunization can trigger rheumatoid arthritis in some individuals (4).

Side Effects of NSAIDs

Nonsteroidal anti-inflammatory drugs are the most frequently prescribed class of medications and one of the most common drug groups associated with serious adverse events (5). They can be extremely dangerous. When used on a chronic basis—not occasionally for pain relief—they cause bleeding from the gastrointestinal tract in approximately 25,000 people annually. Less well known is that they block the body's ability to produce cartilage and can actually cause cartilage destruction. Thus, they accelerate the destructive nature of the disease (6).

Indomethacin (Indocin) has been found to lead to more rapid destruction of the hip joint than any other NSAID (6).

A history of inflammatory bowel disease or diverticular disease should be considered a contraindication to NSAIDs use as they can cause ulcerative disease (7).

End-Stage Renal Disease

People who take acetaminophen or NSAIDs frequently have an increased risk of end-stage renal disease (ESRD), but this risk is not found in those who take aspirin frequently. Both heavy average intake (more than one pill per day) and medium-to-high cumulative intake (1000 or more pills in a lifetime) of acetaminophen appear to double the odds of ESRD.

A 41-year-old woman took 1200 to 1600 mg of ibuprofen for low back pain and developed renal insufficiency. NSAIDs can be associated with nephrotoxicity (proteinuria and renal failure). Diuretics and chronic volume depletion enhance this pathology (8).

Acetaminophen consumption may cause up to 10% of the overall incidence of end-stage kidney disease. High doses of acetaminophen can lead to liver damage, and massive single doses sometimes lead to fatal hepatic necrosis (9, 10).

NSAIDs Impair Nociceptive Input

Nonsteroidal anti-inflammatory drugs have been shown to inhibit prostaglandin (PG) synthesis and affect the synthesis and activity of other neuroactive substances believed to have key roles in processing nociceptive input within the dorsal horn (11).

NSAIDs May Inhibit Spinal Fusion Healing

Forty-five percent of control animals achieved fusion versus 10% of indomethacin-treated animals. Spinal fusion is a process that occurs via osteogenesis, which is affected by NSAIDs. Clinically, the widespread use of NSAIDs in the postoperative period after spinal fusion may need to be avoided (12).

Gastrointestinal Complications of NSAIDs

Increased preoperative bleeding and blood transfusion requirements have been associated with NSAID use (13). Gastrointestinal complications account for most of NSAID-related adverse effects. Almost all NSAIDs cause microscopic blood loss from the gastrointestinal (GI) tract secondary to a direct mucosal toxic effect. A single aspirin pill typically causes 3 mL of fecal blood loss daily (3).

Gastrointestinal-related hospitalizations were six times more frequent in patients with rheumatoid arthritis who were taking NSAIDs than in those who were not, and deaths from GI causes occurred approximately twice as frequently in rheumatoid arthritis patients as in the general population (3).

Unlike short-term NSAID use, long-term therapy with these agents can lead to gastroduodenal ulceration and associated serious complications—hemorrhage, perforation, and death. Mucosal damage is seen in 50 to 70% of arthritis patients treated with long-term NSAID therapy.

A 10.6 times increase in GI ulcers is seen in patients taking combined NSAIDs and corticosteroids. Relative risks for ulcer formation exist with use of several NSAIDs from a low dose of 2.3 mg of ibuprofen to a high of 8.7 mg of meclofenamate. The greatest risk of complications is with piroxicam (Feldene), with progressively lower risk ratios for indomethacin, aspirin, naproxen, and ibuprofen (3).

Use of NSAIDs may promote both ulcerous and nonulcerous lesions of both the upper and lower GI tract; delay healing of peptic ulcers, even to the extent of intractibility; and may cause ulcer recurrence after gastric surgery. Prevention of side effects of NSAIDs is unresolvable (14).

Estimated incidence and relative risk for children with arthritis using NSAIDs is comparable to the rates found in adults with arthritis taking NSAIDs (15).

NSAID-Induced Esophageal Injury

Esophageal injury occurs at sites of anatomic narrowing, such as the midesophagus at the level of the aortic arch and left atrium. Most of these patients present with symptoms of odynophagia, dysphagia, and heartburn. All patients should be advised to take NSAIDs while in the upright position, with sufficient quantities of liquid. They should not take them immediately prior to bedtime when recumbency, reduced salivation, and swallowing can lead to impaired esophageal clearance (3).

Enteropathy and Colopathy With NSAID Use

Sixty to seventy percent of patients on long-term NSAID therapy may develop an asymptomatic enteropathy, associated with low-grade blood and protein losses. The amount of blood loss in most cases has generally been mild, ranging from 1 mL to 10 mL per day, a value similar to the amount of intestinal blood loss in patients with colorectal cancer (5).

Colonic injury, termed "colopathy," can be caused by NSAID use. The spectrum of injury varies from colitis resembling inflammatory bowel disease to an increased rate of colonic perforation, bleeding, or complicated diverticular and appendiceal disease. Rectal administration of NSAIDs has also been associated with proctitis. NSAIDs have been linked to the development of collagenous colitis, a diarrheal disorder characterized pathologically by collagen deposition beneath the surface epithelium, with associated lymphocytic inflammation in the lamina propria. NSAIDs have also been associated with serious complications of diverticular disease, including perforation and fistula formation (5).

Anemia

Small intestine inflammation and bleeding (enteropathy) caused by NSAID uses must be considered in the evaluation of anemia in patients with arthritis (16).

Blood Pressure Monitoring with NSAID Therapy

Many antihypertensive agents are less effective during concurrent therapy with the more potent NSAIDs. Blood pressure must be closely monitored on initiation of NSAID treatment.

Central Nervous System Effects

Aseptic meningitis (most commonly caused by ibuprofen), acute psychoses, and memory dysfunction have been reported, most commonly in the elderly (5).

Liver Function Tests Needed in Patients on NSAIDs

Most NSAIDs and aspirin can cause minor, reversible elevations in liver chemistry values, but only rarely do they cause serious liver injury, which in some cases has been fatal. Serum liver function should be monitored when initiating NSAID therapy, and the medication should be discontinued if levels progressively increase or clinical signs or symptoms of liver disease develop (17).

The relative risk for serious injury is elevated approximately threefold among NSAID users, and may be even higher in the elderly, those with prior ulcer disease, patients who take concomitant corticosteroids, and those taking high-dose or multiple NSAIDs.

Elevations in liver function tests have been attributed to diclofenac (17). Chemical hepatitis can occur with other NSAIDs. Idiosyncratic aplastic anemia is, fortunately, a rare complication of NSAID therapy (5, 17).

Helicobacter pylori Infection and NSAID Use

The relationship between *Helicobacter pylori* infection and NSAID use, and whether the two act synergistically in the pathogenesis of gastroduodenal ulceration, is not known. Both independently impair mucosal defense and are contributing factors in the formation of ulcers. *H. pylori* induces an acute and chronic inflammatory infiltrate in the gastric mucosa termed "chronic active gastritis," whereas pure NSAID ulcers occur in the background of normal mucosa (5).

Withdrawal Reactions from NSAID Therapy

Optimal treatment to promote gastroduodenal ulcer healing during continued NSAID therapy has not been well defined. Whenever possible, NSAIDs should be discontinued to promote more rapid ulcer healing.

Misoprostol is the only unequivocally effective agent for the prevention of NSAID-induced gastroduodenal ulcers. However, the use of this drug is expensive and despite the encouraging preliminary findings, the drug has not proved to reduce complications, such as bleeding, perforation, or death, during long-term NSAID use (5).

Elderly Show Specific Health Problems with Certain Drugs for Back Pain

Several commonly prescribed medications for back pain—including indomethacin (Indocin), cyclobenzaprine (Flexeril), amitriptyline (Elavil), and diazepam (Valium)—could cause health problems in some patients over the age of 65 and they should be avoided (18).

Outcome Results in NSAID Use

A total of 395 male infantry recruits were evaluated in a prospective study of possible risk factors for overexertional back pain and the efficacy of drug treatment regimens for this syndrome. No difference was recorded between the piroxicam-treated group and the control group regarding the presence of pain in the back and leg and functional ability. Nor was any difference found in the need for additional analgesics (19). Little indication is found for the use of NSAIDs in acute cases of sciatica (20). Evidence that NSAIDs accelerate articular cartilage degeneration in patients with osteoarthritis is somewhat contradictory. Although widely prescribed for osteoarthritis, little evidence is found that NSAIDs are better than simple analgesics for managing symptomatic osteoarthritis (21).

Antidepressants Increase Risk of Hip Fracture

Antidepressants, which have sedative and autonomic effects, increase the risk of hip fracture by 60% in the geriatric population (22).

Epidural Lipomatosis Following Steroid Use

Epidural lipomatosis is a condition in which excess adipose tissue is deposited circumferentially about the spinal cord in the epidural space. Most frequently seen in patients on chronic steroid treatment for a variety of medical problems, epidural lipomatosis can present as nonspecific back pain, radiculopathy, or frank spinal cord compression (23).

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NUTRITIONAL APPROACHES TO TREATING DISC DEGENERATION AND OSTEOPOROSIS

Proteoglycan Loss Precedes Disc Degeneration and Arthritis

Connective tissues are composed chiefly of collagen, water, and large glycoproteins called "proteoglycans." Collagen provides the tissue with tensile strength, whereas the proteoglycans, through their large density of negative charge, imbibe water and produce a high swelling pressure within the tissue (1).

Loss of proteoglycan from these tissues is a central event in the development of disk degeneration and osteoarthritis. Studies of human lumbar spines have suggested that loss of proteoglycan can predispose the disk to degeneration and that degeneration may be associated with specific changes in proteoglycan subspecies (1).

Small proteoglycans are actively involved in osteoarthritic processes. They contribute to the deterioration of the articular cartilage and ultimately interfere with the repair processes in arthritic cartilage (2).

End Plate Loss of Proteoglycans Promotes Nuclear Proteoglycan Loss

It has been shown previously that removal of proteoglycans from the end plate accelerates the loss of proteoglycans from the nucleus. Hence, a major function of the cartilage end plate may be to prevent fragments of osmotically active proteoglycans from leaving the disc (3).

Perna Canaliculus as a Source of Chondroitin Sulfate

Perna canaliculus is rated the best preparation ever encountered for the treatment of arthritis, superior to NSAIDs. One of the most popular and effective substances used by doctors in Europe for arthritis is chrondroitin sulfate A (CSA). CSA is naturally found in bones, cartilage, tendons, ligaments, vertebral discs, and in many plants.

Two groups of patients were tested, one with NSAIDs and the other with CSA. Findings were that 77% of those taking CSA had reduced inflammation versus 42% of those taking NSAIDs.

Degraded bones began to repair through the ability of CSA to increase calcium absorption and replacement. CSA diminishes the disease and begins to rebuild the damaged area with none of the health risks of NSAIDs.

Perna canaliculus extract has proved to be the single most effective preparation ever encountered for the treatment of osteo and rheumatoid arthritis (4). Perna canaliculus extract has genuine anti-inflammatory effects (4).

Nutritional Home Care of the Intervertebral Disc Patient

Cole et al. (5) reported that Arteparon, a polysulphated polysaccharide, was administered systemically to mature beagle dogs over a 26-week period. At necropsy, disc proteoglycans were isolated, purified, and analyzed. Their findings were the first report that a systemically administered drug could influence the disc proteoglycans, and they suggested that Arteparon might be of value in the management of degenerative disc disease.

Lowther (6) reported 50% loss of proteoglycan from the cartilage of the rabbit articular cartilage when arthritis of the joint was present. This loss caused the cartilage matrix to be less capable of restoring the proteoglycan content of the cartilage and resulted in loss of joint stiffness and resistance to compression.

Wilhelmi and Maier (7) found, in rabbits with osteoarthrosis of the knee, that injection of sulphated glycosaminoglycans (GAG) inhibited enzymes that destroy cartilage and promoted repair of the defects. GAG has been found to increase proliferation of hyaline cartilage of the hip joint in mice and the femoral

condyles, femur, and tibia of rabbits. Puhl and Dustmann were reported (7) to have induced regeneration of damaged cartilage in rabbits with glycosamine sulfate.

Discat Supplement in Treatment of Disc Degeneration

Discat is a nutritional formula I have used for patients with disc degeneration or disc protrusion. Discat Plus contains 210 mg of manganese sulfate, 160 mg of calcium, 55 mg of potassium, 80 mg of magnesium, 12 mg of zinc, 100 mg of perna canaliculus (glycosaminoglycan, chondroitin sulfates), and 500 mg of glucosamine sulfate. For the first 3 months of care, the patient is told to take four tablets daily, after which a maintenance dose of two tablets a day is prescribed.

Disc Imbibition of Nutrients

Direct vascular contact (vascular buds) exists between the marrow spaces of the vertebral body and the hyaline cartilage of the end plates of the vertebra, which is important for the nutrition of the disc (8). Until a person reaches the early 20s, the intervertebral disc receives nutrients via the epiphyseal end plates. Following their closure, however, the hyaline cartilage between the nucleus and the vertebral body thins and an ingrowth of granulation tissue, which becomes important in the nutrition of the disc, occurs. Diffusion of solutes occurs, both from the cancellous bone of the vertebral body into the nucleus through the end plate and from the anterior and posterior anulus fibrosus. Oxygen and glucose enter primarily through the end plate route, whereas the sulfate radical enters primarily through the anterior and posterior anulus fibrosis.

According to Naylor et al. (9), studies of the components of the disc by chemical analysis, radiograph crystallography, and electron microscopy have shown that in disc degeneration a fall in the total sulfate (both chondroitin sulfate and keratin sulfate) occurs with age. Happey et al. (10) have shown that a gradual diminution of the sulfate content of the disc occurs with aging and degeneration and that the prolapsed nucleus pulposus usually contains less than half the sulfate values of the normal disc. Keep in mind that the posterior anular fibers have the poorest nutrition, although they are subjected to the greatest strain by a bulging turgor-filled nucleus pulposus.

Robles (11), in an extensive study of disc nutrition, used electron microscopy and atomic spectrometry to measure the mineral salts and water content of the anulus fibrosus and nucleus pulposus. He found that the disc, which is deprived of vessels, receives nutrients by the diffusion of plasma filtrates from the surrounding structures. The intervertebral disc is supplied by the vertebral epiphysis until a person reaches the age of approximately 25 years. After fusion of the epiphysis, the vessels join those of the vertebral bodies. Certain vascular loops reach the cartilaginous structures of the vertebral plates and the area above where the disc tissue is formed. It has been suggested that, by diffusion, these loops form nutrient channels from the cancellous bone of the vertebral body into the adjacent disc.

The nucleus pulposus demonstrates a *hydrophilia,* which is an osmotic force that brings about a diffusion of fluid from the vertebral body into the nucleus. The nucleus pulposus demonstrates twice the hydrophilic capacity of the remaining disc. Nutrient channels are formed from the vertebral bone into the disc, and a high rate of mineral salt flow is noted within these channels.

It has been demonstrated by the use of atomic spectrometry that five mineral constituents (potassium, calcium, magnesium, iron, and sodium) flow into the disc. Only one of these elements, sodium, is found in increased concentration within the nucleus. Robles determined the flow of nutrients from the vertebral body into the disc by injecting dye into the nucleus and observing it flowing through the nutrient channels.

Urban et al. (12) found diffusion to be the main mechanism of transport of small solutes into the intervertebral disc. About 40% of the end-plate area was found to be permeable to small solutes in experiments on dogs. The amount of solute entering via the end plate was shown to be less for negatively charged solutes (e.g., sulfate ion) than for the neutral solutes (e.g., glucose) because of altered charge exclusion in the region of the nucleus.

This nutritional route is important, as many authors believe that a correlation exists between the impermeability of the central region of the end plate and disc degeneration. The only solute whose metabolism has been studied is the sulfate ion. A turnover of sulfate occurs in the nucleus pulposus in about 500 days. Consequently, Nachemson (8) believes ruptured discs take a long time, if ever, to heal.

The in vivo procedure used to study sulfate metabolism was performed on dogs, who were anesthetized and given injections of radioactive sulfate tracers. Blood samples were collected at regular intervals until the dogs were killed at intervals of 1 hour to 6 hours after the initial injection. The spines were dissected as quickly as possible, usually within 5 to 10 minutes after death, and plunged into liquid nitrogen. Liquid nitrogen was poured onto the discs to stop diffusion from occurring during the measuring and cutting operations that followed. Urban et al. (12) report that the cell density in the peripheral regions of the anulus and near the end plate is about three to four times higher than that in the rest of the disc. From the values determined in their study, it appears that the cells in the periphery of the disc are taking up sulfate and, hence, producing proteoglycan less actively than those in the center of the disc. The cells in the surface layers of the articular cartilage likewise are less active in producing proteoglycans than are those in the deeper zones.

Sulfate is lost from discs as they undergo degenerative change. It has been postulated that nutritional deficiencies could lead to disc degeneration. If the end plate were blocked, waste products could build up or a nutritional deficiency could predispose the disc to degenerative change (13). Consequently, we use Discat, which incorporates manganese sulfate along with five other minerals, in the treatment of low back pain. The benefits of glucosamine sulfate and glycosaminoglycan are discussed on the following page. Exercises have been reported to improve the delivery of nutrients to the spinal discs, perhaps delaying the deterioration that eventually afflicts all backs (14).

Influence of Exercise on Nutrient Imbibition

Lowther (6) found that, with exercise, synovial lined joints stimulated penetration of nutrients into the cartilage. Kramer (15) states that a continuous well-balanced metabolism is necessary in the disc to maintain the synthesis and depolymerization of the extracellular components. Cells lacking satisfactory nutrition produce macromolecules inferior in quality and quantity.

Holm and Nachemson (16) reported that the free sulfate concentration for exercised canines was higher than among those not exercising. Improved delivery of nutrients by exercise might delay the deterioration that eventually affects all backs (14). Ogota and Whiteside (13) state that nutritional deficiency can lead to disc degeneration because a block of the end plate creates a buildup of waste products or a nutritional deficiency that may predispose the disc to degenerative change.

Finally, Eismont (17) showed in rabbit models that circulation into the disc occurs. More than 50% of the serum level remained 8 hours following antibiotic intramuscular injection with antibiotic present, in the nucleus pulposus. The possible nutritional causes of discal degeneration are only beginning to be understood.

GLUCOSAMINE SULFATE AND GLYCOSAMINOGLYCAN BENEFITS AND CLINICAL OUTCOME STUDIES

Glucosamine sulfate and chondroitin sulfate (glycosaminoglycan) are naturally occurring substances essential for cartilage maintenance and regeneration. Together, they help chondrocytes within cartilage to form new cartilage. The amount of proteoglycans formed depends on the amount of glucosamine present. *The more glucosamine available, the more proteoglycans can be made* (18).

Glucosamine sulfate (GS) is a molecule of sulfur and an amine group bound to glucose, and it serves as the precursor of glycosaminoglycan (GAG) synthesis. Glucosamine stimulates synthesis of GAG, inhibits its degradation, and is anti-inflammatory. Animal studies show 98% absorption, primarily in the small intestine, of glucosamine sulfate after oral administration. Articular cartilage shows high uptake of GS (19).

In a double-blind study of 20 people with osteoarthritis of the knees 10 were given 500 mg of glucosamine three times a day and 10 were given a placebo. Results indicated reduced pain, joint tenderness, and swelling within 6 to 8 weeks in the group given glucosamine (20).

In a study testing glucosamine versus ibuprofen in 100 participants, side effects were much higher in the ibuprofen group (21). A study in Portugal involved 1208 patients given 1.5 g glucosamine in three daily doses over a period of 30 days. Symptoms such as pain at rest, on standing, and on exercise improved steadily throughout the trial period. The improvement lasted for a period of 6 to 12 weeks after the treatment ended (22, 23). Chondroitins protect existing cartilage from premature breakdown by inhibiting the action of certain "cartilage chewing" enzymes (24).

Daily glucosamine by lavage into rat knee joints that had been damaged with pain injection showed an increase of GAG and its synthesis in the cartilage. The treated rats showed decreased pathologic alteration of the cartilage (25).

Chondroitins interfere with other enzymes that attempt to "starve" cartilage by cutting off the transport of nutrients and stimulating the production of proteoglycans, glycosaminoglycans, and collagen—the cartilage matrix molecules that serve as building blocks for healthy new cartilage (26).

Chondroitins work synergistically with glucosamine. Administration of supplemental chondroitin sulfates acts as naturally occurring chondroitins found in cartilage, protecting the cartilage of premature breakdown. Chondroitins are nontoxic (27).

A study done in France followed 50 patients with osteoarthritis of the knee who were given oral administration of either 800 to 1200 mg of chondroitin sulfates or 500 mg of a pain medication. Cartilage tissue samples were taken at the beginning of the study and after 3 months of therapy. Results showed that the cartilage in the chondroitin group was repaired significantly (28).

In a double-blind, random study comparing the effectiveness of a pain killer with chondroitins, 120 patients with osteoarthritis of the knees and hips were given either oral chondroitin sulfates or a placebo. After 3 months, the group given the oral chondroitins reported a reduction in pain and pain movement and no side effects. In addition, a 60-day carry-over effect was seen when administration was stopped. Therefore, the combination of glucosamine and chondroitin sulfate appears to be powerful therapy, working together to help synthesize new cartilage (29).

Glucosamine Sulfate Is the Superior Form of Glucosamine

Glucosamine sulfate (GS), the preferred form of glucosamine, is used in treating osteoarthritis in more than 70 countries. It has been used by millions of people; more than 300 scientific investigations and more than 20 double-blind studies have been done on its use (30). The sulfate compound in GS is an essential nutrient for joint tissue, as it functions in the stabilization of the connective tissue matrix of cartilage, tendon, and ligaments. Arthritis victims are deficient in sulfate and restoring its level brings about significant benefits (31).

Sulfation of Proteoglycans Is Important

Previous studies have presented evidence that an underlying cause of intervertebral disc degeneration is related to changes in proteoglycan sulfation. Chondroitin sulfation as assessed by discogram in cadaveric lumbar IVDs, at two different stages of degeneration, was analyzed. Findings graded 14 of 28 lumbar discs 2 and the other 14 were graded 4 (i.e., more degenerated). The major differences in sulfation of the chondroitin between the grade 2 and grade 4 discs only occurred in the pos-

terior central anulus and nucleus segments. The chondroitin in the posterior central and nucleus segments of the grade 2 and grade 4 IVDs were undersulfated as compared with the other segments, and the differences between these segments and the others were more accentuated in the grade 4 than in the grade 2 IVDs (32).

Sulfur, also important in the manufacture of GAG, inhibits the enzymes that lead to cartilage destruction in osteoarthritis (e.g. collagenase, elastases, and hyaluronidase) (33, 34).

Proteoglycans Determine Imbibition of Fluids Into the Disc

After 2 days of weightlessness astronauts gained up to 60 mm in height. Similarly, during bed rest, when osmotic pressure within discs is greater than hydrostatic pressure from compressive loads of standing, discs imbibe fluid, causing spine lengthening and higher intraspinal stresses on rising (35).

Large Molecules Are Absorbed from the Gut

The question of the GI tract's ability to absorb the molecular formula is often raised. Chichoke (36) found that macromolecules have been recognized as being absorbed through the gut wall in more than immunologically relevant quantities in humans and adult animals. Enteral uptake has not merely been demonstrated for proteins and polysaccharides, but also for larger foreign body particles, such as iron filings, particles of plastic, and so forth. The term "perabsorption" has been coined to describe the absorption of larger particles such as these.

Type II Collagen Inhibits Rheumatoid Arthritis

A collagen solution made from chicken cartilage and swirled into patients' morning orange juice appears to arrest the progress of rheumatoid arthritis in a small group studied. The technique, called "oral tolerization," seems to "teach" the body's immune system to stop inflaming the tissue around joints and re-instructs the body to cease the attack on its own joints. All 28 patients taking the collagen during the 3-month trial got relief from their disease and 4 went into remission; disease worsened in the 31 patients who received a placebo (37).

Dr. Arthur Grayzel, Senior Vice President for Medical Affairs of the Arthritis Foundation, said he was encouraged by the study and believes oral tolerization techniques have the potential to halt rheumatoid arthritis. It was clear that the drug was beneficial (37).

Blood Brain Barrier Does Not Extend to Spinal Nerve Roots

Spinal nerve roots appear to be located outside the blood brain barrier because of their greater vascular permeability to labeled plasma proteins creating a possibility for nutrition by diffusion, which might be one of the nutritional pathways to spinal nerve roots (38).

OTHER SIGNIFICANT RESEARCH IN NUTRITIONAL TREATMENT OF DISEASE

Vitamin D_3 in the Treatment of Osteosarcoma

Vitamin D_3 metabolites have an antitumor and differentiating effect on human osteosarcoma cells in vitro and in athymic mice. Vitamin D_3 should be examined further to discover whether it could be a useful drug in hormonal treatment for human osteosarcomas. Tsuchiya et al. (39) documented the inhibitory and differentiating effects of vitamin D_3 on human osteosarcoma cells. Vitamin D_3, which has been clinically used in treating bone marrow diseases such as myelofibrosis, myeloblastic syndrome, and acute myeloblastic leukemia, should be the subject of further experimental study and clinical trials for the treatment of human osteosarcomas (39).

Smoking Causes Disc Malnutrition

Smoking leads to disc malnutrition, which in turn renders the disc more vulnerable to mechanical stress. Malnutrition can be brought about by carboxyhemoglobin formation, nicotine-induced vasoconstriction, arteriosclerotic vessel wall changes, impairment of fibrinolytic activity, and changes in the flow properties of blood (40, 41).

Other Medical Nutritional Advances

Treatment with black current seed oil resulted in reduction in signs and symptoms of disease activity in patients with rheumatoid arthritis (42). High doses of vitamins A, B_6, C, and E reduce the risk of recurrence of disease in patients with transitional cell carcinoma of the bladder (43).

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OSTEOPOROSIS

Osteoporosis, a decrease in bone density and weight, affects 20 to 25 million US residents, and it is present in about one of four women over the age of 65 (1).

Bone Composition

Bone is made up of three components: matrix (50%), mineral (45%), and cells. Nearly 100% of total body calcium is located in bone with minerals consisting of hydroxyapatite crystals containing calcium, sodium, potassium, magnesium, and carbonate (2).

By the fourth decade of life, the peak bone mass has been reached and a slow loss of both cancellous and cortical bone begins. Osteoporosis affects women more severely than men because, after menopause, women experience an accelerated loss of cancellous bone (1). Another study, however, shows peak bone mass is obtained by the middle of the third decade, and the greater the peak bone mass achieved, the better the chance of avoiding osteoporosis later in life (3).

Five factors determine the risk of developing osteoporosis: age, initial bone density, menopause, bioavailability of calcium, and sporadic factors such as low weight, smoking, alcohol intake, and physical activity. One of three women will have a vertebral fracture after age 65 years and a hip fracture in extreme old age (4).

Two Types of Osteoporosis

1. Postmenopausal or idiopathic osteoporosis occurs most commonly in women between the ages of 50 and 70 years, and it is associated with rapid loss of trabecular bone and

vertebral fractures. This is the accelerated type of osteoporosis, with a loss of up to 10% a year.

2. Involutional or senile osteoporosis occurs equally in men *and women between the ages of 70 and 90 years, and it is* associated with loss of both cortical and trabecular bone with hip fractures (2).

Other Risk Factors for Osteoporosis

1. Heavy alcohol intake and smoking affect osteoblast activity (5, 6).
2. Hypogonadal men and women treated with glucocorticoids are at markedly increased risk for spine fracture (5).
3. Vasectomy. Eight of twenty-four men who developed osteoporosis at a mean age of 52.1 years had vasectomies between 5 and 15 years prior to diagnosis of their osteoporosis (6).
4. Vibration. Chain-saw operators whose both upper extremities had been exposed to the impact of vibrations exceeding the threshold limit showed a statistically significant difference in the mineralization of their clavicles when compared with a control group (7).
5. Menopause, exercise deficiency, and genetics. Early natural or operatively induced menopause, prolonged periods of amenorrhea, poor nutrition, history of limited exercise, genetic factors (a positive family history), and a history of excessive alcohol intake or smoking were cited as risk factors (3).

Causes

The most important nutritional risk factor is inadequate calcium intake (1). Approximately 25% of the dietary dose of calcium is absorbed primarily in the upper part of the gut. Normal absorption of calcium by the gut requires an appropriate gastric pH level, an adequate serum level of 1,25-dihydroxyvitamin D, and an appropriate dietary calcium:phosphate ratio (3).

Other Mineral Deficiencies Involved with Osteoporosis

Magnesium: The typical US diet is low in magnesium, which suggests a possible widespread deficiency. Surveys of the diet in 1985 show that 80 to 85% of US women consume less than the US Recommended Daily Allowance (USRDA) of magnesium. A second survey suggests intake is only about two thirds of the USRDA (8–13).

Manganese: Manganese is essential for the formation of the bone's organic matrix and for the synthesis of connective tissue in bone and cartilage. Serum manganese levels have been found to correlate with osteoporosis (8–13).

Copper: In the United States, adults typically consume approximately one half of the USRDA for copper (8–13).

Zinc: Most US adults typically do not consume sufficient zinc. A dietary survey showed that 68% of adults consumed less than two thirds of the USRDA of zinc. Zinc deficiency causes reduced osteoblast activity, collagen and chondroitin sulfate synthesis, and alkaline phosphatase activity (8–13).

Caffeine: A threefold increase in the risk of hip fracture was found in women who consumed large amounts of caffeine (more than 4 cups of coffee per day) (8–13).

Imaging Diagnosis

Both inter and intraobserver perception of osteopenia on lumbar radiographs shows good agreement (14). Up to 40% of bone loss will have occurred by the time plain radiographs detect osteopenia (15).

Dual-energy radiograph absorptiometry, commonly called "DEXA," has a high rate of precision, and it subjects the patient to only a low dose of radiation. DEXA is currently the most frequently used method of evaluating bone density in clinical practice (3).

Radius Diagnosis of Osteoporosis

Subjects with severe osteoporosis in the distal radius suffer severe degenerative changes in the discs and the facets; those with mild osteoporosis in the distal radius show a tendency to have a lesser degree of degenerative changes (16).

Height Loss Suggests Osteoporosis

Vertebral collapses are found in 35.4% of women who have lost more than 3 cm or 1.18 inches in height. Height measurement in adults could be a simple and inexpensive method to detect spine lesions, and particularly osteoporosis, even in asymptomatic subjects (17).

Treatment

Renal calculi are not a contraindication to increased calcium intake: *high dietary calcium intake reduces kidney stones.*

A prospective study was done of the relationship between dietary calcium intake and the risk of symptomatic kidney stones in a cohort of 45,619 men, aged 40 to 75 years with no history of kidney stones. After adjustment for age, dietary calcium intake was inversely associated with the risk of kidney stones; high dietary calcium intake decreased the risk of symptomatic kidney stones. *There is no support for the belief that a diet low in calcium reduces the risk of kidney stones* (18). Another study of 91,731 women aged 34 to 59 years with no history of kidney stones showed that the risk for stone formation varied inversely with intake of dietary calcium, and that supplemental calcium was positively associated with kidney stone formation. Dietary calcium reduces the absorption of oxalate, and the apparent different effects caused by the type of calcium may be associated with the timing of calcium ingestion relative to the amount of oxalate consumed (19).

Intuition has linked stone formation to levels of urinary calcium, but women consuming low calcium diets seem more at risk for developing kidney stones than those with high calcium intakes. Calcium intake of 800 mg per day or more should be ingested by those with a tendency to form stones who also take thiazide daily. A low calcium diet does not increase urine supersaturation (19).

Calcium and Vitamin D Dosage in Osteoporosis Prevention and Treatment

In the elderly, 800 U of vitamin D per day is recommended. Women at risk for osteoporosis should maintain a daily intake of 1500 mg of calcium. Calcium supplements are best absorbed when taken with meals and in an acidic environment (1).

The US Food and Drug Administration and the National Institutes of Health Consensus Conference on Osteoporosis have published a recommended daily allowance for calcium: 1000 mg for estrogen-normal women and 1500 mg for estrogen-deprived women. Middle-aged and elderly women have an average intake of calcium of only 550 mg per day, and women with osteoporosis often consume less. The calcium requirement of premenopausal women is 1000 mg, whereas for postmenopausal women it is 1500 mg (4, 20). Women need this amount of calcium for the reasons listed below.

1. Middle-aged women cannot achieve calcium balance at intakes of less than 1000 mg per day (21).
2. Calcium absorption efficiency declines with age (22).
3. Estrogen hormone deficiency leads to decreased calcium absorption and decreased retention of absorbed calcium (23).

Men also develop osteoporosis, although less commonly than women. It occurs in men due to lowered testosterone hormone (24).

CALCIUM AND VITAMIN D_3 TREATMENT RESULTS

Forty-five osteoporotic patients medicated for 1 to 13 years with α_1-hydroxyvitamin D_3 with calcium supplement (treated group) and 11 osteoporotic patients with no medication for 1 to 3 years (control group) were compared. The bone mineral density (BMD) of the treated group remained unchanged for the first 4 and 6 years, followed by significant decreases, whereas that of the control group decreased significantly at the second and third year. The vertebral fracture rate of the treated group was significantly less than that in the control group at the third year. Thus, α_1-hydroxyvitamin D_3 with calcium supplement can be considered a safe and effective agent for long-term use in osteoporotic patients (15).

Calcium supplementation of 1000 mg per day significantly slows bone loss by 43% in the spine, hips, and extremities. A postmenopausal woman needs 1500 mg of calcium to keep her calcium balance (17).

Children receiving supplemental calcium exhibited significantly enhanced gain in bone mass relative to those not receiving supplements. Complete cessation of age-related bone loss occurred at an average calcium intake of less than 900 mg per day (9).

Calcium Prevents Pre-eclampsia

Calcium supplementation results in an important reduction in systolic and diastolic blood pressure and pre-eclampsia in pregnant women (8), although this finding is controversial.

Calcitonin

The well-demonstrated effects of nasal calcitonin permit it to be considered as a highly rational solution for the prevention and the treatment of postmenopausal osteoporosis (10).

Testosterone

Testosterone replacement therapy is available for hypogonadal men. Calcitonin also suppresses osteoclast activity. Calcium, vitamin D, estrogen, calcitonin, biphosphonates, and fluoride are recommended in necessary cases (3). Drug treatment of osteoporosis is not covered in this text.

For premenopausal women, I prescribe 500 mg; for postmenopausal women, 1000 mg of nonphosphorous calcium citrate daily to supplement their dietary intake. Men are prescribed 500 mg daily.

Osteoporosis Compression Fracture: Diagnosis and Treatment

Incidence

Each year, about 1.3 million bone fractures related to osteoporosis occur (12 to 20% being hip fractures), and the resulting cost is estimated to be between $7 and $10 billion (1). The incidence of compression fracture from osteoporosis is from 26 to 35%, even in those with no injury or prior knowledge of fracture (17).

Twenty-six percent of persons over 50 show compression fracture on radiograph and only 8% seek medical care for them (15). **More women die from osteoporosis-related fracture than combined breast and ovary cancer,** and 1 in 4 women over 50 are affected (11). Twenty percent of women and 34% of men with hip fracture die in less than a year; osteoporosis is usually asymptomatic until a fracture occurs. The RDA of calcium is 1000 to 1500 mg (12).

Fracture Sites

The most common fracture site for women is L1, and in men it is T12 (25). There are three types of fractures:

1. Compression fractures with loss of the entire vertebral height.
2. Anterior wedge fractures with posterior height maintained.
3. Biconcave collapse of end plates, known as "picture framing."

Most fractures consistently occur in three anatomic sites:

1. Apex of the thoracic kyphus.
2. The transitional thoracolumbar zone.
3. Apex of the lumbar scoliotic curve.

Differential Diagnosis of Osteoporotic Versus Tumor Fracture

The MRI characteristics that differentiate osteoporotic from tumor compression fractures or vertebral lesions are as fol-

lows: (*a*) Decreased signals of T1-weighted images and increased signals on T2-weighted images are sensitive but not specific for tumor involvement. (*b*) Normal marrow preservation of the compressed vertebral body or lesion on T1-weighted images almost completely rules out a tumor fracture or lesion. (*c*) Pedicle involvement or an associated soft tissue mass are fairly specific for a tumor compression fracture or lesion (26).

Causes of Compression Fracture

Bending, lifting, stepping from a curb, pulling on a wrench, a minor fall, stooping, or such a common event as bending forward at the waist can cause collapse of an osteoporotic vertebral body. Sneezing or coughing have been forceful enough to cause fracture. Even a minor fall can cause an acute vertebral compression fracture (VCF) (17).

Pain Description

Usually, pain is sharp and localized to the affected vertebral level, aggravated with spine loading activity and flexion, and relieved in a neutral spine position.

Patients with less severe acute pain may have a delayed onset of visible radiographic fracture up to 3 months after the initial onset of acute pain. After 2 to 3 months, a compression fracture should have healed so it is no longer a source of local pain (17). Moderate levels of back pain can persist for several years after the fracture. Not every woman who has a fracture develops back pain. In one study only 46% of women who developed a new fracture reported increased frequency of back pain. The other 54% of women with new fractures either did not experience significant back pain or forgot about the back pain at follow-up (27).

Rehabilitation

Acute osteoporosis fracture treatment involves the following:

1. One to two weeks of bed rest. Some patients may sit up with support at 3 to 4 days. To maintain a neutral spine curvature and reduce kyphotic tendency, a thin pillow is placed under the patient's head and a regular pillow under the knees as the patient lies supine in bed. Physical modalities such as infrared heat lamp and gentle stroking massage can help relieve muscle spasm.
2. Adequate pain management and institution of hormonal and calcium supplementation are crucial in the complete care of these patients. Calcitonin has been shown to stabilize bone mass in hyper-resorptive states and to decrease acute biomechanical pain (17). Vitamin D deficiency is a common finding (28).

Modest increases in physical activity and calcium intake (even in the third decade) might result in significant reductions in fracture risk later in life (29).

Calcium Types for Treatment of Bone Intake

Studies favor the more soluble forms of calcium such as citrate, citrate-malate, and hydroxyapatite. Citrate absorption is 20 to 66% greater than carbonate, and also greater than carbonate in achlorhydrics and normochlorhydrics. Citrate absorbs twice as well as hydroxyapatite (8, 13).

Orthoses Use

The purpose of an orthoses in vertebral compression fracture is to maintain spinal alignment and relieve pain. Figures 9.80 (Taylor brace), 9.81 (Jewett brace), and 9.82 (Cash brace) shown earlier in this chapter render pain relief as custom-molded orthoses (30).

In trauma to the upper thoracic spine, a cervicothoracolumbosacral orthosis stabilizes the upper thoracic and lower cervical spine. Jewett orthosis or a Knight-Taylor brace with pectoral extensions is used to stabilize upper thoracic fractures and to effectively limit flexion in this region. Prolonged rigid fixation is not recommended.

Exercise Program for the Disc Lesion Patient

The patient is started on the first three of the Cox exercises (Fig. 9.83) at the outset of treatment, regardless of pain severity. Following relief of Déjérine's triad (i.e., relief of pain in the low back on coughing, sneezing, and straining at the stool), the patient is prescribed the remainder of the exercises. These exercises must be chosen carefully by the doctor with regard to the patient's condition. The exercises are on a videotape, which is given to patients to assist them in performing them properly.

Home Care for the Disc Lesion Patient

Following examination and diagnosis of the patient's condition, the radiographs are shown to the patient and the condition is explained. It is important that the patient understand his or her problem as fully as possible in order to participate in care and recovery. A copy of the book *Low Back Pain: What It Is and How It Is Treated* (Fig.9.84) is given to the patient. Figure 9.85 is the index of the book. The patient's diagnosis and treatment procedures, along with instructions on what to do at home, are written down for the patient to study and follow. This forces the patient to become personally involved with care. Figure 9.86 shows the instructions for the patient to follow at home; the appropriate instructions are checked for each patient.

COX LOW BACK WELLNESS SCHOOL

Every patient is invited to attend low back wellness school, which is a 2-hour class that teaches the patient how to control a low back problem so that the problem does not control the patient. The school consists of three parts: first is a 25-minute seg-

THE COX EXERCISES

TO ACCOMPANY CHIROPRACTIC MANAGEMENT OF LOW BACK PAIN

Exercises for the acute severe low back pain patient.

Exercise 1.

Lie on your back with your knees flexed and your feet flat on the floor as close to the buttocks as possible. Keep the knees together. Tighten the muscles of the lower abdoman and buttocks so as to flatten your low back against the floor. Slowly raise your hips up from the floor and hold for slow count of 8. Repeat this exercise 4 times. If you cannot raise your hips from the floor, merely tighten the belly, the abdominal and buttock muscles and wait until you can raise the hips.

Exercise 2.

Lie on back and draw the right knee up to the chest and pull the knee down upon chest while attempting to touch the chin to the knee. Do this for a slow count of 8 and repeat 4 times. Repeat the same exercise with left knee brought to the chest. Relax between each session. Repeat with both knees brought up to the chest.

Exercise 3.

While standing or lying tighten the abdominal and buttock muscles so as to flatten your back. Repeat this several times throughout the day. Contract the muscles and relax the approximately 8 times at each session.

Exercises after the acute pain has diminished. Do the following exercises if you feel no pain in your low back upon coughing, sneezing, or straining to move the bowel.

Exercise 4.

Repeat #1 exercise above but be sure to hold the knees firmly together.

Exercise 5.

Lie flat on your back and raise the right leg straight upward without bending the knee. Place your hands behind the knee while keeping the knee straight, pull the leg straight up so as to stretch the muscles behind your thigh. Repeat this 8 times on the right leg and then do it on the left. Relax your low back muscles following this exercise.

Exercise 6.

Lie on stomach and raise the right leg off of floor while keeping the knee straight. Hold the leg up in this position for a count of 4 and slowly let it down. Repeat this 4 times. Repeat the same exercise with the opposite leg. Relax following this exercise.

General Instruction

Do Not Sit when you have low back pain. This increases the pressure within the disc and the joint of your spine. If your doctor prescribes a belt to wear, remove it to do these exercises. If your doctor agrees, it is good to alternate hot and cold on your low back before doing these exercises. This is done by applying moist heat in the form of a hot towel for 10 minutes followed by 5 minutes of ice therapy in which a moist cool towel is placed on the skin with an ice bag on top of it. Place the heat on the back 4 times and ice on the back 3 times beginning and ending with heat.

If your doctor suggests nutritional supplementation, be sure to follow it closely.

Do these exercise on a firm surface such as the floor or a mat. Do not be alarmed if discomfort is noted during exercise. If this pain is great, stop it and consult your doctor before continuing.

The Cox exercises are to be used in conjunction with your chiropractic care and should be discussed with the chiropractic physician before use.

Do the exercises marked (x) in numerical order ____________times a day.

Figure 9.83. Cox exercise program.

Exercise 7.

Lie flat on stomach with arms along side, palms down. Slowly raise chest from floor. Feel the muscles of the low back tighten. Hold the chest up from the floor for a slow count of 6 and slowly let it down. Rest between each session. Repeat this 6 times.

Exercise 8.

Sit on floor on your knees. Extend your right leg as far to the side as possible, keeping the knee straight and the arch of the foot on the floor. Slide your foot along the floor until you feel the stretch of the muscles inside your thigh. Do it slowly and hold for a count of 5. Repeat it 3 times on the right leg and then repeat with the left side. These muscles, which are tight at the beginning, will loosen and stretch with subsequent exercise sessions.

Exercise 9.

Abdominal Strengthening Exercises. Lie on Back with Knees bent and feet on floor. Bring chin to chest as shown. Now tighten the abdominal muscles so as to lift and curl the shoulders up to about 1 foot off the floor. Remember - curl up the spine from the neck downward to between the shoulder blade. Feel the abdominals tighten. Do this 10 to 30 times depending on your stamina.

Exercise 10.

Lie on side. Turn the toes inward on the right foot and lift leg upward. Repeat this 6 times on right and then 6 times on the left. You will feel pulling in the outer thigh and pelvis.

Exercise 11.

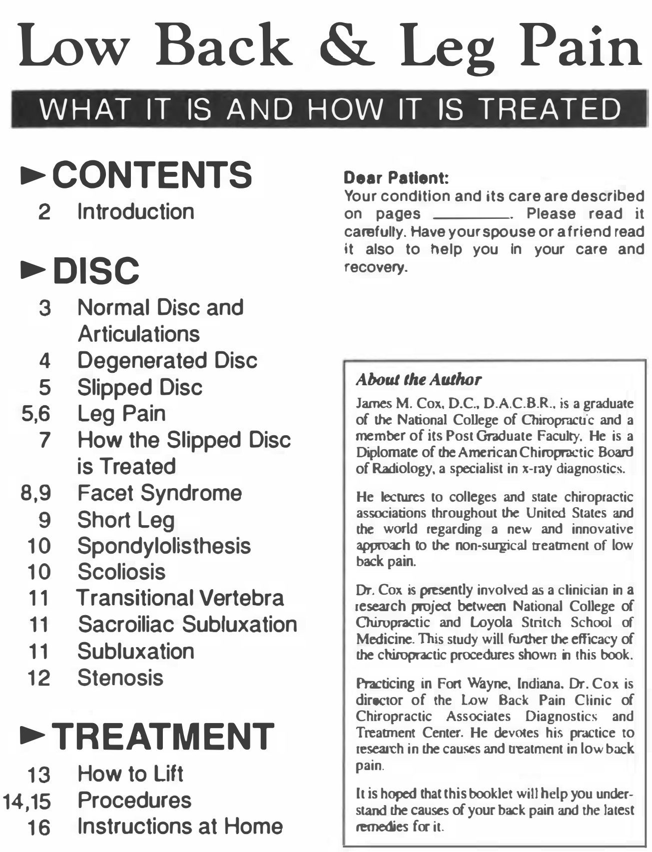
Lie on back and draw knees to chest, arms extended level with shoulders, roll hips to side in attempt to touch the knees to floor. Turn your head, in the opposite direction to which your knees are bending. Repeat this 4 times going first to the right and then to the left. This exercise brings all spinal movements together in a smooth forceful manipulation of the spinal articulations. Since the exercise involves rotation, it should only be done under physician instruction.

Exercise 12.

Lie on back. Bend knees and bring feet up to the buttocks. Now lift and straighten the legs so that the legs are at a right angle to the body. Raise the buttocks from the floor and place the hand beside the buttocks and support your pelvis as you raise the pelvis from the floor. Allow the legs to go over the head with feet over the head and the legs parallel to the floor. Hold this position for 10 seconds and repeat 2 - 3 times. Slowly lower your pelvis and legs to the original starting position. This exercise shoulo only be used by those who have been working with the exercises for some time and have their low back pain under control.

Figure 9.83. *continued*

Figure 9.84. This book contains a simple explanation of various problems occurring in the low back, such as disc degeneration, disc protrusion, spondylolisthesis, transitional segment, subluxations, facet syndrome, short leg, stenosis, and scoliosis. It helps the patient understand his or her problem.



Low Back & Leg Pain

WHAT IT IS AND HOW IT IS TREATED

►CONTENTS

2 Introduction

►DISC

3 Normal Disc and Articulations
4 Degenerated Disc
5 Slipped Disc
5,6 Leg Pain
7 How the Slipped Disc is Treated
8,9 Facet Syndrome
9 Short Leg
10 Spondylolisthesis
10 Scoliosis
11 Transitional Vertebra
11 Sacroiliac Subluxation
11 Subluxation
12 Stenosis

►TREATMENT

13 How to Lift
14,15 Procedures
16 Instructions at Home

Dear Patient:
Your condition and its care are described on pages ________. Please read it carefully. Have your spouse or a friend read it also to help you in your care and recovery.

About the Author

James M. Cox, D.C., D.A.C.B.R., is a graduate of the National College of Chiropractic and a member of its Post Graduate Faculty. He is a Diplomate of the American Chiropractic Board of Radiology, a specialist in x-ray diagnostics.

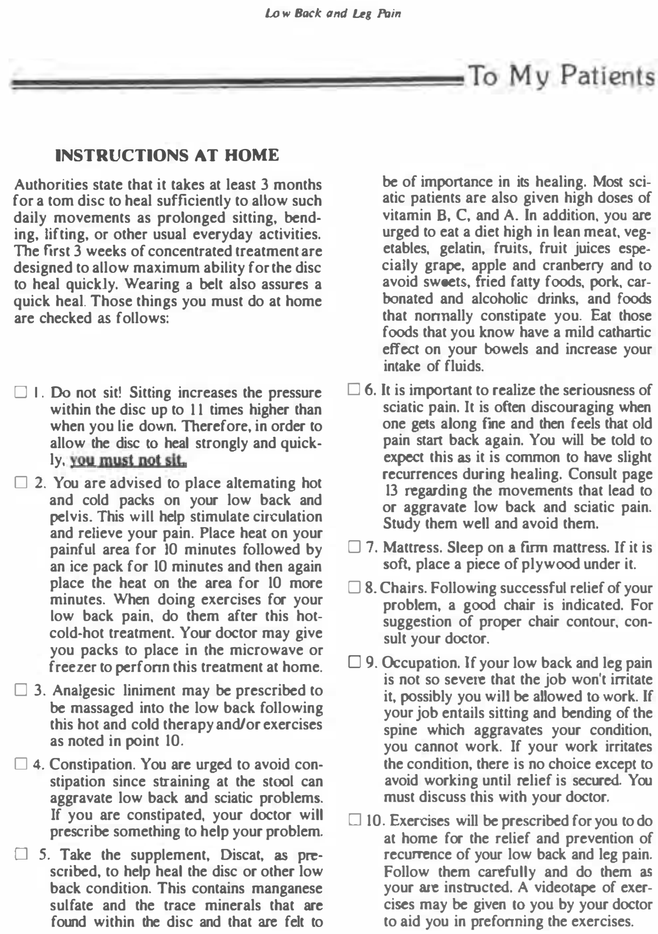
He lectures to colleges and state chiropractic associations throughout the United States and the world regarding a new and innovative approach to the non-surgical treatment of low back pain.

Dr. Cox is presently involved as a clinician in a research project between National College of Chiropractic and Loyola Stritch School of Medicine. This study will further the efficacy of the chiropractic procedures shown in this book.

Practicing in Fort Wayne, Indiana, Dr. Cox is director of the Low Back Pain Clinic of Chiropractic Associates Diagnostics and Treatment Center. He devotes his practice to research in the causes and treatment in low back pain.

It is hoped that this booklet will help you understand the causes of your back pain and the latest remedies for it.

Figure 9.85. Index of the book.



Low Back and Leg Pain

To My Patients

INSTRUCTIONS AT HOME

Authorities state that it takes at least 3 months for a torn disc to heal sufficiently to allow such daily movements as prolonged sitting, bending, lifting, or other usual everyday activities. The first 3 weeks of concentrated treatment are designed to allow maximum ability for the disc to heal quickly. Wearing a belt also assures a quick heal. Those things you must do at home are checked as follows:

☐ 1. Do not sit! Sitting increases the pressure within the disc up to 11 times higher than when you lie down. Therefore, in order to allow the disc to heal strongly and quickly, **you must not sit.**

☐ 2. You are advised to place alternating hot and cold packs on your low back and pelvis. This will help stimulate circulation and relieve your pain. Place heat on your painful area for 10 minutes followed by an ice pack for 10 minutes and then again place the heat on the area for 10 more minutes. When doing exercises for your low back pain, do them after this hot-cold-hot treatment. Your doctor may give you packs to place in the microwave or freezer to perform this treatment at home.

☐ 3. Analgesic liniment may be prescribed to be massaged into the low back following this hot and cold therapy and/or exercises as noted in point 10.

☐ 4. Constipation. You are urged to avoid constipation since straining at the stool can aggravate low back and sciatic problems. If you are constipated, your doctor will prescribe something to help your problem.

☐ 5. Take the supplement, Discat, as prescribed, to help heal the disc or other low back condition. This contains manganese sulfate and the trace minerals that are found within the disc and that are felt to be of importance in its healing. Most sciatic patients are also given high doses of vitamin B, C, and A. In addition, you are urged to eat a diet high in lean meat, vegetables, gelatin, fruits, fruit juices especially grape, apple and cranberry and to avoid sweets, fried fatty foods, pork, carbonated and alcoholic drinks, and foods that normally constipate you. Eat those foods that you know have a mild cathartic effect on your bowels and increase your intake of fluids.

☐ 6. It is important to realize the seriousness of sciatic pain. It is often discouraging when one gets along fine and then feels that old pain start back again. You will be told to expect this as it is common to have slight recurrences during healing. Consult page 13 regarding the movements that lead to or aggravate low back and sciatic pain. Study them well and avoid them.

☐ 7. Mattress. Sleep on a firm mattress. If it is soft, place a piece of plywood under it.

☐ 8. Chairs. Following successful relief of your problem, a good chair is indicated. For suggestion of proper chair contour, consult your doctor.

☐ 9. Occupation. If your low back and leg pain is not so severe that the job won't irritate it, possibly you will be allowed to work. If your job entails sitting and bending of the spine which aggravates your condition, you cannot work. If your work irritates the condition, there is no choice except to avoid working until relief is secured. You must discuss this with your doctor.

☐ 10. Exercises will be prescribed for you to do at home for the relief and prevention of recurrence of your low back and leg pain. Follow them carefully and do them as your are instructed. A videotape of exercises may be given to you by your doctor to aid you in preforming the exercises.

Figure 9.86. This page describes the patient's home instructions for care of the low back problem. The rules to be followed by the patient are indicated by placing a checkmark in the box in front of the number.

ment to teach the patient the causes of low back pain based on knowledge of the disc and facet joint as sources of pain. Next, ergonomic training is given, teaching the patient the proper low back motions to avoid disc damage; emphasis is placed on lifting, bending, and twisting as the causes of back pain, and on the combined stresses they create on the lumbar spine. Finally, the Cox exercise program is presented with attendees participating so that they learn how to do them properly. Questions and answers are shared in a mutually beneficial atmosphere. Once the patient has attended this 2-hour class, he or she understands two important facts: first, the patient is equally responsible with the doctor for his or her own care and relief, and, second, although a cure is not always possible, low back pain can be controlled. Patients learn that degenerative disc disease, spondylolisthesis, transitional vertebrae, scoliosis, and disc protrusions, although not curable conditions, can be controlled by proper understanding and use of the low back in daily living.

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LOW BACK WELLNESS SCHOOL

Principles and Benefits

Opposing opinions are found in the literature regarding the benefits of muscle strengthening exercises, lifting postures, return to work factors, and range of motion findings in persons with and without low back pain. Low back wellness school is a definite benefit for low back pain patients, their family, and the doctor. Once patients attend low back school, they are never the same patient again. They realize that there is no cure for low back pain, but there is control. Often, for the first time, patients learn that they are responsible for a major portion of their treatment. That is, they must build parameters into their lifestyle to allow maximal improvement to be attained and maintained. Discussed below are some studies that reflect the needs and shortcomings of low back school.

Texas Mandates Back School

Texas Worker's Compensation found that 37% of all compensable injuries are related to the back and pelvis. The average cost of these injuries per case is $18,725.00. In response to these expenses for back injured workers, Texas is mandating back school in an attempt to reduce costs (1).

Back School Lowers Cost of Back Care

After back school 42% fewer consultations, 59% fewer physical therapy referrals, and 33% fewer imaging procedures were ordered. Consultations with back care physicians in medicine or family practice were ordered more frequently than were consultations with either neurologists or orthopaedists, which reduced the cost of back care. This change in pattern of care took place without significantly altering the clinical course (2).

A study of a standardized functional restoration program that included 11 centers in seven states, involving 303 patients in the treatment group and 94 patients in the comparison group, found that patients demonstrated improved return to work rates and work retention after both surgical and nonsurgical patients participated in a functional restoration program (3).

Back School Relieves Back Pain

A six-session outpatient hospital program had a great immediate and 1 year follow-up impact on patients' actual physical

fitness and on their knowledge of correct body mechanics. Patients returned to work sooner, had shorter sick leave time, and learned that it is safe to move while regaining function (4). Intensive exercises, "work-hardening" exercises, or expensive equipment were not necessary to regain occupational function (5).

Psychological treatment added nothing to the effectiveness obtained by a standard exercise rehabilitation program devoid of psychological counseling (6).

Prevention of herniated lumbar discs through education in lifting techniques and evaluation through intervention studies should be extended beyond the workplace and into the home. Lifting 25 or more pounds with the knees bent and back straight and not extending with the arms or twisting during the lift are justifiable precautions (7).

The American Back School teaches students to maintain the lumbar lordosis while lifting. Cognitive learning strategies and practice in correct lifting were also taught with significant differences between the back school group on the cognitive, psychomotor, and affective measures. Results indicate that the back school is an effective tool for influencing lifting posture and conveying information regarding spinal mechanics and lifting technique. Video programs and control groups did not do as well (8).

Back School Reduces Reinjury, Cost, and Time Lost

Back school participants had significant reduction of lost work time, lost time and medical costs, and number of injuries for 70 back-injured workers who participated in a 6-week back school when compared with 70 randomly selected back-injured city employees who did not participate in a back school. Actual dollars saved in lost time and medical costs between the groups was of practical value to the city (9).

Benefit for Chronic Low Back Pain Disability

Eighty-one patients with chronic low back pain were randomly allocated to a fitness program or control group. Significant differences between the groups were shown in the changes before and after treatment in scores on the Oswestry low back pain index, pain report, self-efficacy report, and walking distance (10). After completing an intensive rehabilitation program, 71% of patients reported significant improvements on pain severity and interference, depressed mood, and perceived disability. They also significantly improved their execution of repetitive dynamic lifting (11).

Patients Not Expecting a Cure Have Better Results

In a study, 71 back pain patients and their therapists (6 chiropractors and 6 rheumatologists) showed that congruent patients seem to accept living with their back problems, whereas noncongruent patients do not seem to share this conception of back pain. Congruence mainly reflects an agreement on the treatment goal being the management of a long-term condition rather than at the resolution of the back pain problem. Noncongruent patients responded less favorably to treatment and the patient–therapist relationship seemed more difficult (12).

RETURN TO WORK PRINCIPLES

The goal of back school is to assist return to work for the patient; therefore, a discussion of return to work principles is in order.

Without Restriction Is Best

The probability of failure increases significantly with the recommendation of restricted return to work. The success rate for the return to work without restrictions group was 84%, compared with only 47% for the restricted group. The recommendation to limited capacity can become a self-fulfilling prophecy and patients see themselves as no longer able to perform their normal work-related duties. The recommendation to return to work unrestricted doubled the number of people who went back to full duty (13). The best way a general practitioner can play a role in reducing sickness absence is by encouraging an early return to work (14).

Return to Work Index (15, 16)

Patients ($n = 134$) who returned to work had fewer job, personal, or family-related problems. No significant differences were found between patients who returned to work and those who did not when comparing myelograms, computed tomographic scans, or radiographs. Patients who did not return to work had a statistically higher incidence rate of muscle atrophy. For patients off for fewer than 6 months, important predictors were a high Oswestry score, history of leg pain, family relocation, short tenure on the job, verbal magnification of pain, reports of moderate to severe pain on superficial palpation, and a positive reaction to a "sham" sciatic tension test (15, 16).

Final Predictive Indices

Figure 9.87 shows the factors included in the predictive indices of Lancourt and Kettlehut (15) for return to work. Range of the index for the sample was 0 to 15, the actual minimal score for any patient was 0, the maximal score was 10, and the mean was 4.8. The average score of patients returning to work was 3.9; for those not returning it was 7.2, and the difference was significant ($P \geq 0.01$).

Figure 9.88 compares the differences in return to work and those not returning to work based on the index score. For the total sample, the probability of return shifted at the index score of 4. Patients scoring more than 4 were not likely to return, and those scoring less were likely to return to work (15).

Younger Disabled Patients More Difficult to Rehabilitate

Younger patients with longer duration of work disability who report lower return to work expectations and higher levels of perceived disability, pain severity, and focus on bodily sensations may experience compliance problems during active rehabilitation (17).

Factor	Response*	Q#†	Total Sample	Time Off Work ≤6 Months	Time Off Work ≥6 Months
Personal factors/history					
Prior workman's comp injury	Yes	1	1	—	1
Oswestry score	≥55	2	1	1	—
History of leg pain	Yes	3	1	1	—
Length off work	>6 months	4	1	NA	NA
Family factors					
Living arrangement	Other than single or married	5	1	1	1
Length of living arrangement	≥7 years	6	1	—	1
Relocation	Due to problems	7	1	1	—
Employment information					
Employees less than 26 weeks	Yes	8	—	1	—
Fired or terminated	Yes	9	1	—	
General stress indicators					
Financial difficulty	Yes	10	—	1	1
General coping	Problems	11	1	—	—
Non-organic physical signs					
Verbal magnification	Present	12	1	1	—
Superficial palpation	Moderate/severe tenderness	13	1	1	—
Sciatic tension (pf)	Non-negative	14	1	1	—
Mixed organic/non-organic signs					
Supine straight leg raise (right or left)	<90 degrees, either leg	15	1	—	1
Lateral bending (right or left)	Decreased ≥25%	16	—	—	1
Gait	Uneven or assisted	17	1	—	1
Ease of forward flexion	Slow or difficult	18	—	1	1
Deep palpation	Any tenderness	19	—	1	1
Organic physical signs					
Muscle atrophy	≥1/4 inch	20	1	1	—
Sitting straight leg raise	Inconsistent	21	—	—	1
Diagnostic findings by groups					
None found		NA	—	—	—
Possible index values			0 to 15	0 to 12	0 to 10
Actual index minimum value			0	0	2
Actual index maximum value			10	10	9
Actual index average			4.8	4.8	5.8
Value for workers returning to work‡			3.9	3.5	4.3
Value for those not returning			7.2	6.4	6.2

*Selection points for reponses based upon 2 × 2 analysis using Fishers Exact Statistic All 2 × 2 analysis significant at $p \leq 01$

†Q# refers to Appendix 1 list of questions

‡Differences between each group significant (t-test, $p \leq 01$)

Figure 9.87. Factors included in the predictive indices. (Reprinted with permission from Lancourt J, Kettlehut M. Predicting return to work for lower back pain patients receiving worker's compensation. Spine 1992;17(6):629–640. Copyright 1992, Lippincott-Raven.)

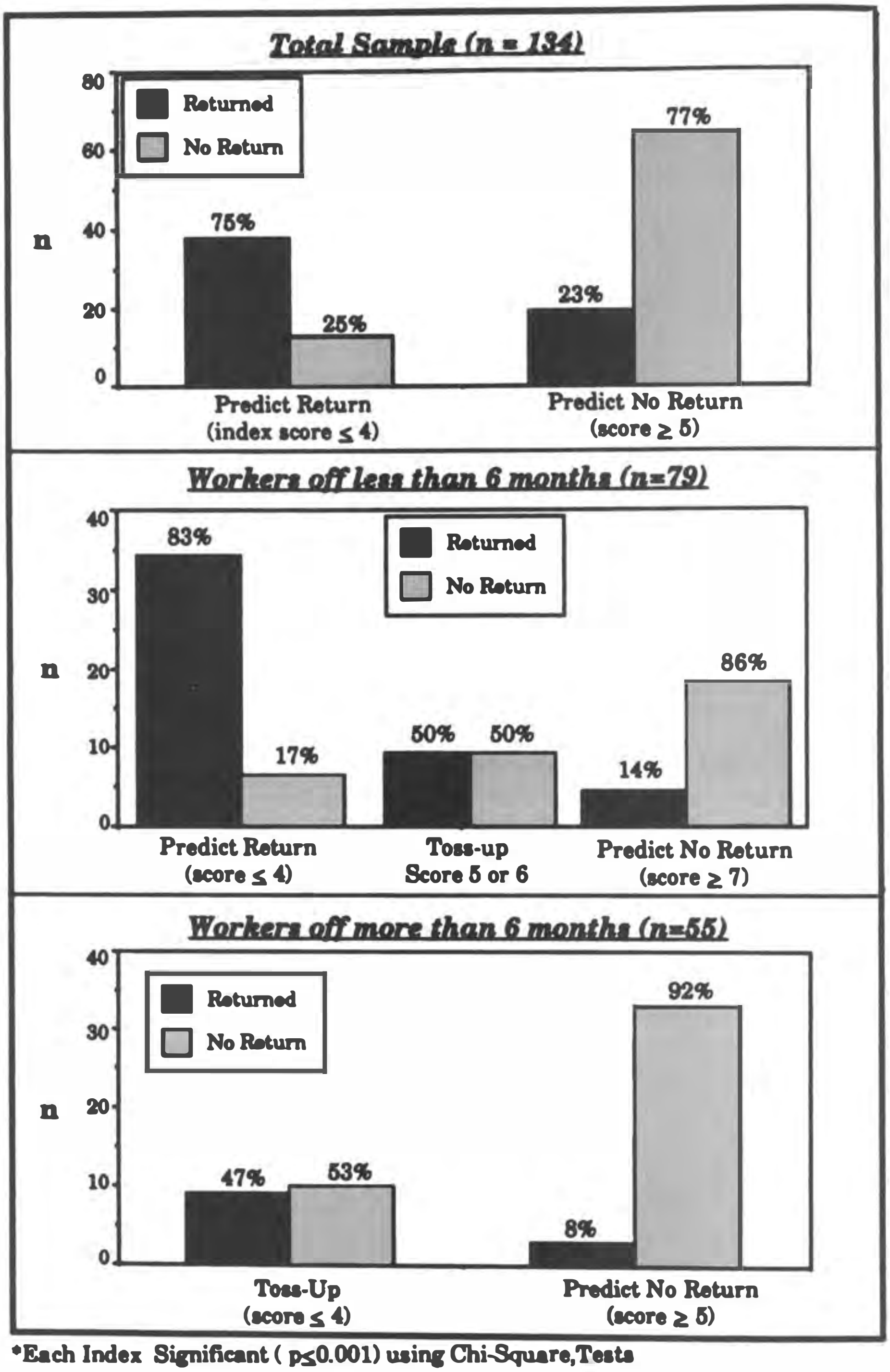

Figure 9.88. Comparison of actual return with predicted return to work. Percentages indicate ratio for each category. (Reprinted with permission of Lancourt J, Kettlehut M. Predicting return to work for lower back pain patients receiving worker's compensation. Spine 1992;17(6):629–640. Copyright 1992, Lippincott-Raven.)

Worker's Compensation Patients Have Less Relief

At the time of discharge, 9 of 10 patients with mechanical low back pain syndrome were working in some capacity compared with 5 of 11 patients with herniated lumbar disc disease (18).

Differing Opinion on the Value of Back School

A randomized, placebo-controlled trial study of 473 patients in 40 general practices assessed the efficacy of exercise therapy for acute low back pain. Patients received either exercise instruction with advice for activities of daily living by a physiotherapist, placebo ultrasound therapy by a physiotherapist, or usual care by the general practitioner. All patients received analgesic agents and information on low back pain before randomization. It was concluded that exercise therapy for patients with acute low back pain had no advantage over usual care from the general practitioner (19).

For acute work-related low back pain, back school did not reduce the time to return to work or the number or duration of recurrent episodes of low back pain requiring compensation over 1 year compared with identical treatment without back school. In addition, no differences were seen between these groups for the level of pain, functional status, and spinal mobility in the year after study enrollment (20).

Satisfaction with Back School Cited

I have developed and taught a 2-hour low back wellness school with patient-involved exercises for the last 20 years. All new low back pain patients are invited and urged to attend as soon as possible after starting care. The class is held every 2 weeks, and the spouse, family members, and friends of the patient are invited to attend. Three parts of the school are:

1. Causes of low back pain: Biomechanics of the lumbar spine and pelvis is covered with an explanation of the pain-generating tissues in the spine (e.g., disc, facet capsule, ligaments, and so forth), along with an explanation of the degenerative and congenital defects that occur for which no cure exists. The patients are told that, although no cure exists for low back pain, control is taught in this class. Lumbar disc herniation is explained and the course of conservative chiropractic care is described together with an explanation of the course to be taken if chiropractic care is not successful. Home care of the back problem is stressed, which includes instruction on intradiscal pressure changes by various postures, cautionary lifting and bending, home exercises, hot and cold therapy, massage, diet changes and nutritional supplementation, bracing, and occupational changes. The monetary cost of low back pain to industry, society, and individual suffering is taught. Education and qualifications of chiropractic physicians in today's academic world is covered so the patient gains confidence in the doctor's care and ability.
2. Ergonomics: The principle, "There is no cure for back pain but there is control," is stressed. The patient is taught proper daily lifting habits with emphasis on specific actions that require repetitive rotation actions (e.g., hoeing, shoveling, starting gasoline engines with pull starters, making a bed, changing a tire).
3. Exercise: Low back pain exercises are performed at the end of class so that the patient and accompanying significant others are made aware of the proper execution and importance of each exercise.
4. The family members or friends are taught how to apply hot and/or cold therapy and liniment massage to the trigger points of the lumbar spine and pelvis, which helps increase improvement of the patient condition.
5. Return to normal activities of daily living are encouraged as soon as pain allows. I do not urge prolonged rest, but rather ambulation as improvement occurs and increased exercise as the patient is capable.

A positive acceptance of this low back wellness training is evidenced by 95% of the 61 patients feeling it was worth their time to attend and that they had learned something to prevent reinjury in daily living; 100% felt it worth the doctor's time to present the class. Low back wellness school is a positive program from both the patient's and the doctor's viewpoints (21).

PATHOPHYSIOLOGIC FINDINGS IN BACK PAIN PATIENTS

Disc degeneration was seen more frequently in 48 low back pain patients than in the healthy volunteers. Psoas and back muscles (erector spinae and multifidus) of the patients were smaller than those of the volunteers. Patients had also more fat deposits in the back muscles than controls. The maximal isometric strength of trunk muscles of the patients was on average weaker than that of the volunteers (22).

Weak Paraspinal Muscles

The lumbar back muscles exert a net posterior shear force on segments L1 to L4, but exert an anterior shear force on L5. Collectively, all the back muscles exert great compression forces on all segments (23). In low back pain patients the paraspinal muscles demonstrate excess fatigability (24). Weakness in both the low back and lower extremity muscles suggests that low back pain cannot be explained by selective trunk muscle atrophy. Generalized muscle weakness, from disuse atrophy or poorly developed musculature, or from psychological factors (e.g., fear of injury) or malingering may cause weak paraspinal muscles (25, 26).

Thirty-six men, aged 45 to 55 years, with healthy low backs were studied with respect to body composition, isokinetic and isometric trunk strength, trunk muscle endurance, and cross-sectional area and radiologic density of erector spinae muscles. Results were compared with those of men in the same age group with intermittent low back pain and with chronic low back pain. Those in the group with a healthy back were significantly stronger and had longer trunk muscle endurance times than men

with chronic low back pain. Men with intermittent low back pain had strength and endurance values in between the healthy back and chronic groups. No significant differences were found between any of the groups with respect to body composition and cross-sectional area of the erector spinae muscles. Radiologic density for erector spinae muscles was significantly decreased in the chronic low back pain group compared with the healthy back and intermittent low back pain groups (27).

Muscle Strengthening Benefits Chronic Low Back Pain Patients

Trunk muscle strength was evaluated in 123 patients with chronic low back pain and 126 healthy individuals without low back pain (control group). Patients were further divided into two groups—those in group 1 had detectable organic lumbar lesions and those in group 2 had no detectable organic lesions.

Trunk muscle exercises reduced low back pain in both groups, but they were more effective in group 2 than in group 1.

The exercise-associated increase in trunk muscle strength did not completely eliminate the low back pain induced by the organic lumbar lesions in group 1. However, increasing trunk muscle strength was effective in patients in group 2, in whom decreased trunk muscle strength was a major factor in chronic low back pain (28).

Symptomatic geriatric women can increase their strength with progressive resistance exercise, which leads to a decrease in low back pain (29).

Deconditioning

The most common physical finding in patients chronically disabled with low back pain is deconditioning. Rehabilitation starts, from week 0 to 6, by telling the patient that it is safe to move with acute low back pain. In subacute low back pain, weeks 6 to 12, active exercise treatment results in 30% fewer work days, and cost of care 25% less at 9-month follow-up compared with the conventionally treated patients. Transcutaneous electrical neuromuscular stimulation (TENS) units added no advantage to exercise.

Manipulation in patients with 14 to 28 days of low back pain produced statistically significant improvements in pain and function and did not improve outcome compared with no treatment in patients with symptoms of less than 14 days or more than 28 days (30).

A 12-week rehabilitation program that addressed physical deficits and function, not pain complaints, significantly reduced self-reported pain and disability and significantly improved return to work compared with controls (30).

Patients with low back pain (n = 40) exhibited significantly less upper torso and pelvic motion and lateral trunk flexion than those in a control group (n = 40) (31).

The anterior and posterior neck muscles in patients with osteoarthritis of the cervical spine fatigue faster than those of normal subjects (32).

Muscle Atrophy Relates to Spinal Disability

The fat content of the lumbar back extensor muscles, assessed from computed tomograms, showed a positive relationship between the fat content of the lumbar paraspinal muscles at the lumbosacral level and self-reported disability in men. The relationship was weaker in women, and at higher lumbar levels it was not found in either sex. CT scan atrophy, an objective clinical measure, seems to correlate with subjective disability (33).

MRI Detects Muscle Injury

Magnetic resonance imaging can detect evidence of acute and chronic muscle injury (of psoas, multifidus, and longissimus/iliocostalis) with increased signal intensity in injured muscle persisting for as long as several months after the initial injury (34).

The left sciatic nerve was sectioned in seven of nine rats. Axial MRI with and without fat suppression were obtained 1, 8, 15, and 29 days postsurgery. Electromyographic (EMG) studies were performed on two additional nerve-sectioned rats. The T2 of denervated muscles become significantly elevated at 15 days apparently because of an increase in water content. MRI could be used as a painless, noninvasive technique to reveal the pattern of denervation in human muscles. EMG can detect denervation slightly earlier (35).

Magnetic resonance imaging of the rectus capitis major and minor muscles in subjects with chronic pain found dead suboccipital skeletal muscle was replaced with fatty tissue. This infiltration was not observed in control subjects. Reduction in proprioceptive afferent activity in affected muscles may cause increased facilitation of neural activity that is perceived as pain (36)

Muscle Degeneration As Common As Disc Degeneration

Lumbar intervertebral discs and paraspinal muscles in 74 healthy volunteers ranging in age from 19 to 74 years were evaluated with MRI, and the occurrence of degeneration was correlated to age and body mass. Degenerated back muscles were small and contained fat deposits. By contrast, the psoas muscles never showed gross fat deposits. Degeneration of both the lumbar discs and muscles increased with age. No correlation was found between muscle and overweight. *Muscle degeneration is as common as disc degeneration in the lumbar area* (37).

Two signs of muscle degeneration, easily detected on MRI are (*a*) atrophy, which is histologically seen in early and advanced stages of muscle degeneration; and (*b*) deposits of fat and connective tissue, which are characteristics of advanced degeneration (37).

MUSCLE STRENGTH CAN BE REGAINED THROUGH EXERCISE

Isometric lumbar extension strength can be maintained for up to 12 weeks with a reduced frequency of training to as low as once every 4 weeks when the intensity and the volume of ex-

ercise are maintained (38). Six weeks of extension exercises increased extensor lumbar muscle strength by 22% (39).

Lumbar extension exercises resulted in decreased pain and improved perception of physical and psychosocial functioning in chronic back pain patients (n = 54) who had been assigned to a 10-week exercise program or a wait list control group. Women were found to respond better to intensive back exercises than men, whereas men responded best to physiotherapy (40).

Stabilization Procedure for Low Back Pain Patients (41)

Active lumbar stabilization (ALS) is divided into four stages representing muscle re-education, static stabilization, dynamic stabilization, and functional activities.

Stage 1: Oblique abdominal, transversus abdominis, and multifidus are facilitated.
Stage 2: Lumbar spine is held in mid range while exercising, an alignment termed the "neutral position."
Stage 3: Restoration of correct pelvic tilting.
Stage 4: Proprioceptive training and stabilization activities using a 65-cm gymnastic ball.

Multifidus Muscle Atrophy Can Be Reversed

Maximal or submaximal effort can reverse the selective atrophy of type 2 fibers in the multifidus muscles in men. Some studies (42) have found little or no association between muscle strength and the development of low back symptoms. Other studies have shown that trunk muscles of patients with chronic low back pain are much weaker than those of healthy individuals, the difference being greater in trunk extension than in flexion (42).

In a porcine model, stimulation of the disc and the facet joint capsule produced contractions in multifidus fascicles. Interactive responses may occur between injured or diseased structures (e.g., disc or facet joints) and the paraspinal musculature. Activation of the multifidus muscles may have a stabilizing effect, constraining the motion of the lumbar spine. Long-standing musculature contraction may produce ischemic conditions and may be a potential source of pain (43).

Graded Activity Reduced Sick Leave and Hastened Return to Work

Blue-collar workers who were on sick leave for 8 weeks because of subacute, nonspecific, mechanical low back pain underwent a graded activity program consisting of four parts: (*a*) measurements of functional capacity; (*b*) a work-place visit; (*c*) back school education; and (*d*) an individual, submaximal, gradually increased exercise program, with an operant-conditioning behavioral approach, based on the results of the tests and the demands of the patient's work. The patients in the activity group returned to work significantly earlier than did the patients in the control group. The patients in the graded activity program learned that it is important to move while regaining function. Intensive exercises, "work-hardening" exercises, or expensive equipment were not necessary to regain occupational function (44).

Coordination training for patients with chronic low back pain is equally as effective as endurance training (45).

Exercise May Influence Nutrition to the Disc

Improved nutrition of the intervertebral disc induced by motion and partially by release of endorphins that modify the perception of pain might explain exercise improvement in low back pain patients (45).

No Correlation of Radiograph to Pain Severity

No correlation of lumbar spine radiographs with back pain severity and/or training effect is found (45).

Thixotropy

Thixotropy is the phenomenon of connective tissue becoming more fluid when it is stirred up and more solid when it sits without being disturbed. No way is known to prevent the eventual drying and stiffening of connective tissues, a process which eventually produces wrinkled skin and cranky joints of old age. Poor nutrition and sedentary habits weaken all the connective tissues of the body, stiffen them, and significantly accelerate their biologic aging, even in a young adult (8).

Trauma causes loss of movement and vitality with loss of the vigor required to keep the connective tissue warm, moist, and resilient. Manipulation to an anatomic part can raise metabolic rate and restore some fluidity to connective tissue. Pressure and stretching and the friction they generate can raise the temperature and energy level of the tissue to promote more connective tissue ground substance, which is more soluble and ductile.

Excessive deposits of connective tissue can be palpated as thick, lumpy bandaging around joints, as fibrous tissue throughout an entire area, or as tough fibrotic ropes and cysts in the muscle bellies. It is this thickening, shortening, and gluing that eventually prevents erect posture and graceful motion (8).

No Relationship Between Weight Loss and Low Back Pain Risk

No apparent evidence in the current literature supports the recommendation of weight loss as a treatment for low back pain in any patients with a body mass index less than 29.0. Little evidence is found that correlates low back pain with obesity in a body mass index greater than 29.0. No apparent connection is seen between fat mass changes and low back pain risk, with the possible exception of weight loss in the severely obese by means of surgical intervention (46).

No Difference in Outcomes Between Flexion and Extension Exercises

Flexion and extension exercise groups did not differ in any outcome over 8 weeks. After 1 week, both exercise groups had reduced disability scores, a higher proportion returning to work, and fewer subjects with a positive straight-leg raise compared

with the control group. No difference was seen among groups regarding recurrence of low back pain after 6 to 12 months of exercise. *However, exercise was slightly more effective than no exercise when patients with acute low back pain were treated* (47).

Muscle Stretching

Exercise causes significant increases in the static strength of back extensor muscles and in back muscle myoelectric signal. Pain decreases significantly after 2 weeks of continuous treatment (48).

Hamstring Muscle Principles

Short hamstrings limit pelvic flexion during forward bending with straight knees, but lumbar flexion range of motion is not influenced significantly. People with short hamstrings could be more susceptible to low-back injury than those who have normal-sized hamstrings. After maximal pelvic flexion during forward bending, further effort could increase the lengthening stress on lumbar spine tissues (49, 50). Altered or faulty motion patterns during forward bending in subjects with a history of low back pain may contribute to high rate of pain recurrence. Subjects with a history of low back pain tend toward tighter hamstrings. Improving hamstring flexibility in subjects with a history of low back pain may allow greater hip motion and less stress on the lumbar spine during forward bending (51).

A 3-week program of hamstring muscle stretching (*a*) will not alter standing lumbar and pelvic postures, (*b*) will produce greater forward bending as a result of increased motion at the hips, and (*c*) may alter the pattern of lumbar and hip motion during forward bending (52).

Time and Heat Effect on Hamstring Muscles

Stretching for 30 seconds is effective in enhancing the flexibility of the hamstring muscles. No increase in their flexibility occurred by increasing the duration of stretching from 30 to 60 seconds. However, 15 seconds of stretching was no more effective than no stretching (53). Application of a superficial heating or cooling modality to the hamstring muscles did not improve the efficacy of static stretching (54).

No Difference in Fitness for Those With or Without Low Back Pain

Range of motion, symptoms, straight leg raise, strength, physical ability, reduced propensity for low back impairment, pain episodes, and grip strength were found no different between controls and back-injured persons. Both groups were classified as deconditioned by fitness testing (55).

No differences were reported in low back pain episodes, abdominal muscle strength, and grip strength between experimental and control groups (56). No evidence indicates that preplacement back strength testing would predict workplace claims of injury (57). Among patients with acute low back pain, continuing ordinary activities within the limits permitted by the pain leads to more rapid recovery than either bed rest or back-mobilizing exercises (58). Posture training supports for osteoporosis patients (n = 45) showed that back extensor strength can increase in patients who comply with its use and a postural exercise program (59).

LOW BACK BELT SUPPORTS

Controversy exists regarding whether low back belt supports are helpful. As large companies such as Coca-Cola and Wal-Mart adopt the use of back belts to reduce injuries, attention has focused on arguments in favor of using back belts. The truth is somewhat different (32). Low back belt use can cause a lack of neuromuscular coordination of the abdominal muscles (60).

American Airlines, in a study of 642 baggage handlers, found no significant differences in rates of total lumbar injury incidents, lost workdays and restricted workdays, and Workers' Compensation claims. Participants who wore the belt for a while and then discontinued its use had a higher lost day case injury incident rate than the control group workers who did not wear the belts (60). Compliance was a major problem, with the overriding complaint about the belt being it is too hot. Other complains are sore backs, shortness of breath, stomach pains, numbness, and upper back strain. Only 5 of the 642 employees reported that the belts reminded them of proper lifting techniques.

Weight-lifting belts are not the solution in reducing injuries. Injury rate was significantly higher in employees who used the belt and then discontinued use. Belts lead to weakening of the abdominal muscles and are clearly not recommended (60). Workplace modifications, optional belt wearing, employee education, and exercise to counter weakened abdominal muscles are recommended (60).

Back Belts Do Not Reduce Risk of Back Injury

Use of a lumbar belt does not enhance isometric lumbar muscle strength or dynamic lifting capacity (61). The magnitude of increased lifting ability in women, although statistically significant, is not sufficient to advocate the use of lumbosacral support belts to increase lifting capacity (62).

No evidence indicates that wearing back belts reduces the risk of back injury among otherwise healthy workers; workers wearing back belts can increase their risk of injury by attempting "to lift even more with the belt than they would have without it." Little evidence is found that lumbar belts are any more capable of strengthening the spine or preventing back injuries than a cummerbund on a tuxedo (63).

Lumbar belts do not prevent back injuries or lost work time. In fact, the longest study to date suggests that wearing a lumbar belt and then discontinuing its use may increase injury rates. Costs for injuries incurred while wearing a belt were significantly higher than injuries sustained without a belt (64).

The National Institute of Occupational Safety and Health (NIOSH) states that the value of back belts in the workplace is as yet unproved, and that they may slightly increase the chance of back injuries. A company policy regarding back belts should make wearing one voluntary, not mandatory (65).

The value of wearing a back belt in the workplace is unproved, and their wear may slightly increase the chance of back injury. Wearing them should be voluntary (66). Evidence suggests that the use of weight belts changed performance or affected strength and endurance in persons without back problems. No difference in back injury was seen in an 8-month period between airline baggage handlers who were randomized to wearing or not wearing back supports (67).

Some workers consider the belt beneficial, and others consider it an impediment. It really comes down to who is made happy when a belt is worn—the employer or the worker (68).

Lumbar Belt Wearers Need to Be Screened for Cardiovascular Disease

Workers who perceive a benefit to belt wearing should wear a belt on a trial basis if they satisfy the following criteria:

1. Given the concerns regarding increased blood pressure and heart rate while wearing a belt, all candidates for lumbar belts should be screened for cardiovascular risk by medical personnel.
2. Because of the concern that belt wearing provides a false sense of security about lifting ability, all belt users must be educated on lifting mechanics.
3. Belts should be not prescribed until a full ergonomic assessment has been made of a worker's job.
4. Belts should not be considered for long-term use (69).

A study found that back support devices reduced low back injuries by about one third in a study of 36,000 Home Depot Inc. employees between 1989–1994 (70).

WHAT IS THE BEST LIFTING POSTURE?

Back Muscle Forces in Flexion Similar to Upright Posture

Compression forces and moments exerted by the back muscles in full flexion are not significantly different from those produced in the upright posture (71).

Anterior Pelvis Suggested As Safest Lift Posture

The effect of two different alignments of the pelvis and three different loads on electromyographic activity of the erector spinae and oblique abdominal muscles during squat lifting and lowering was studied. Each of 15 healthy subjects lifted and lowered loads using two different techniques: the pelvis aligned in an anterior tilt and in a posterior tilt. The results suggest that the greater trunk muscle activity occurring with the anterior tilt position may ensure optimal muscular support for the spine while handling loads, thereby reducing the risk for low back injury (72).

Lumbar Lordosis Is Best Lift Posture

Seven muscles in 17 healthy men were analyzed: rectus abdominis, abdominal obliques, erector spinae, latissimus dorsi, gluteus maximus, biceps femoris, and semitendinosus during three squat lifts with a 157-N crate with the spine in both a lordotic and kyphotic posture. The increased erector spinae EMG activity seen in the lordotic lift in the first quarter of the lift suggests its greater involvement in lumbar spine support. Lifting with the lumbar spine in lordosis is felt to be advantageous (58).

Safest Lift Is with a Flexed Spine

Lifting a moderate or heavy load with a flexed spine does not unduly stress the lumbar spine. Mayer teaches his patients that the natural lifting style is with slightly bent knees and flexed spine and hips. He sees no concrete evidence that this technique developed through thousands of years of evolution should be changed by the limited understanding of back pain gained in the last 50 years. He notes that back school trained persons invariably return to this traditional style of lifting when not supervised (73).

The L2–L3 and L4–L5 motion segments were studied in cadavers by taking stress profiles of the segments with the spinal segments at 0 and 75% of full flexion after loading the spines with 500 to 2000 N of force. A minimal force of 500 N showed spinal flexion to increase both pressure in the nucleus and stress in the front of the disc anulus. At first this bolstered the idea of a lordotic lift posture. But when 2000 N of force was applied to the motion segments, flexion no longer increased nuclear pressure and the *peak stress on the posterior anulus was reduced.*

Flexed spinal sections are slightly stronger than lordotic posture segments (73) and:

1. When lifting heavy objects, a flexed posture does not greatly raise pressure within the disc.
2. At high load levels, the anterior anulus "stress shields" the nucleus in flexed postures.
3. Lordotic postures do not strengthen the spine, but expose the posterior structure of the spine to excessively high stress loads.
4. Intradiscal pressure only increased in low load levels, and high load levels showed no advantage to lordotic postures when lifting.
5. A lordotic posture should not be advocated during manual labor. Avoid lordosis in bending and working continuously.
6. Moderate or high compressive loads on the lumbar spine will produce high stresses on the apophyseal joints when lordosis is maintained while lifting.

   Note: One point of flexion-distraction manipulation is to unload the apophysial joints and reduce stenotic factors in the posterior arch of the motion unit, while allowing intradiscal pressure to drop and disc protrusion to reduce. This study by Adams (73) does a lot to confirm my past concepts. Other studies show similar findings (74–76).
7. Workers can lift safely in postures that are within the normal range of flexion if they observe good body mechanics.

8. Bogduk (73) states that the flexed spine is not a weakened or particularly vulnerable structure. It is protected from injury during flexion by the posterior ligaments, the intervertebral discs, and the back muscles. Normal discs suffer acute herniations only with severe hyperflexion injuries involving forces and ranges of motion well outside the normal range of activities of daily living.

Flat Lumbar Lordosis During Lifting to Avoid Injury

Lumbar "motion segments," consisting of two vertebrae and the intervening disc and ligaments in cadavers were compressed while positioned in various angles of flexion and extension. Extension caused the apophyseal joints to become load-bearing, and damage occurred at compressive loads as low as 500 N. Flexion angles greater than 75% of the full range of flexion (as defined by the posterior ligaments) generated high tensile forces in these ligaments and caused substantial increases in intradiscal pressure. The optimal range for resisting compression, therefore, appeared to be 0 to 75% flexion (77). At 0% flexion high stress concentrations occur in the posterior anulus of many discs, whereas an even distribution of stress was usually found at 75% flexion. No significant difference is seen in the compressive strength of motion segments positioned in 0 and 75% flexion. Moderate flexion is preferred when the lumbar spine is subjected to high compressive forces, and normal lumbar lordosis should be flattened during manual handling to avoid injury to the osseoligamentous lumbar spine (77).

Farfan (78) wrote that in the flexed position, compression is increased in the disc. The facet joints are jammed into close contact, and torque transmission is increased. Both ligament systems are tightened and backed up by all the abdominal muscles. In this position, the spine is in its most rigid mode and in the best position to avoid damage. It is in the best position to be adopted for peak performance at all times.

Leg Lifting Is Not Always Better Than Back Lifting Posture

It appears that lifting with the back rather than the legs minimizes the energy required to move the body and load mass combination. Although it was commonly believed that "back lifting" was more stressful than "leg lifting," this is not always the case (79).

Minimizing the distance of the load from the lumbar spine is the most important principle when lifting loads. A heavy load held close to the body when lifted is much less hazardous to the back than when held away from the body. As the horizontal distance at the start of a lift increases, the peak moment acting on the lumbar spine also increases, but the increase is nonlinear. The moment magnitude influence of horizontal distance of the object is such that as the distance changes from 20 to 40 cm, the distance-related rate of increase is approximately one half of that occurring with a distance change from 40 to 60 cm (80).

Flexed Knee, Straight Back Is *Not* Best Lifting Technique

The *cumulative trauma model* attributes low back pain to a combination of gradual spinal degeneration and prolonged exposure to compressive loads (81).

The B-200, a triaxial dynamometer, was used to determine the effects of standing postures at upright (0°) and at 15° and 35° of trunk-flexed positions on the triaxial torque-generating capabilities of the trunk and EMG activities of selected trunk muscles (erector spinae, latissimus dorsi, oblique and rectus abdominis muscles) during maximal and submaximal isometric trunk extension.

As the trunk was flexed from 0° to 15° to 35°, the erector spinae and latissimus dorsi muscles showed significantly increasing EMG activity. For the abdominal oblique muscles, however, the EMG activity decreased significantly as the trunk-flexion angle increased. In all tests, the rectus abdominus muscles were silent.

Trunk-flexed posture up to 35° was measured because most trunk activities (e.g., manual material handling and lifting) occur in the sagittal plane with the trunk slightly flexed to approximately 36°. Also, prolonged standing and prolonged stooping had been shown to be associated with low back pain (81).

Neuromuscular Efficiency Ratio

When the neuromuscular efficiency ratio (NMER) was analyzed during 100% of maximal voluntary exertion (MVE) only, both the erector spinae and the latissimus dorsi muscle groups showed significant increase in NMER as the trunk was flexed from 0° to 35°.

The findings that NMER and torque-generating capacities of the trunk are both increased at higher trunk-flexion angle does not seem to support the conventional wisdom of recommending lifting with straight back and bent knees. In many workplaces, the straight-back and bent-knees lifting technique does not seem to be realistic. In fact, as the load increased during repetitive freestyle lifting, a tendency was seen to lift more with back muscles and less with leg muscles (i.e., with straight legs) to reduce metabolic costs (81).

Static Posture Promotes Muscular Ischemia

Prolonged static posture fosters continuous muscular contractions creating muscular ischemia. Local muscular ischemia is believed to cause disorders at the insertion site of tendons, ligaments, and articular capsules. Static spine postures also restrict the fluid flow and the nutrition of the intervertebral disc (79).

Chronic Low Back Pain Patients Do Not Have Restricted Lumbar Flexion

Lumbar flexion was not reduced in chronic low back pain patients, which may explain some of the current thought casting doubt on the presence of any true anatomic or structural impairment in chronic low back pain patients (16).

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CARE OF SPECIFIC LOW BACK CONDITIONS

Nonoperative Adjustment Treatment of Adult Scoliosis

Always remember that scoliosis and pain do not necessarily occur together. They can be two totally different findings, with the pain coming from a cause other than the scoliosis. Therefore, diagnosis demands consideration of what these other causes might be.

Chiropractic adjustments can be applied to these patients with an expectation of pain relief and increased mobility. The techniques must be applied carefully, with complete awareness of patient discomfort, applying low-force levered manipulative adjustments to the intervertebral disc and facet joint spaces. Remember that the greater the arthrotic change present, the less force must be used in the application of the adjustment. The facets are tested individually as they are moved through their physiologic ranges of motion. The presence of pain during test-

ing precludes the use of that particular levered motion with the instrument. The application of such manipulative adjustive techniques will be described in the case discussed below.

Case 1

A 71-year-old white woman was seen complaining primarily of pain in the lumbar spine, with some radiating pain into the thoracic spine in the T6 to T12 area bilaterally. The chief pain was at the L3–L4 level, and it was more severe on the right side of the spine. To complicate the pain scenario, this patient had gallbladder dysfunction that caused her pain in the right abdomen and spine, for which she was under treatment. Her spinal pain had progressed to the point where it awakened her at night after approximately 4 hours of sleep. Examination revealed normal vital signs, normal urine analysis, the abdomen negative for masses or pain at the time of examination, and no other cause for her spinal discomfort aside from the degenerative scoliosis.

Radiographic examination (Figs. 9.89 and 9.90) showed a levorotatory degenerative scoliosis of the lumbar spine with the apex at the L3–L4 level and an L3–L4 vacuum phenomenon present. The lateral view (Fig. 9.90) reveals the extensive degenerative state of the L3–L4 disc. The extensive atherosclerosis of the abdominal aorta was noted, which creates an awareness of applying any pressure to this abdomen.

My impression was that a degenerative levoscoliosis of the lumbar spine was a major cause of this patient's pain. She remembered having been told of a minor curve in her younger years.

Again, I want to stress that total relief of this patient's pain is impossible. The goal was to attain some measure of relief and improved quality of life. To that end, the following chiropractic adjustments were applied.

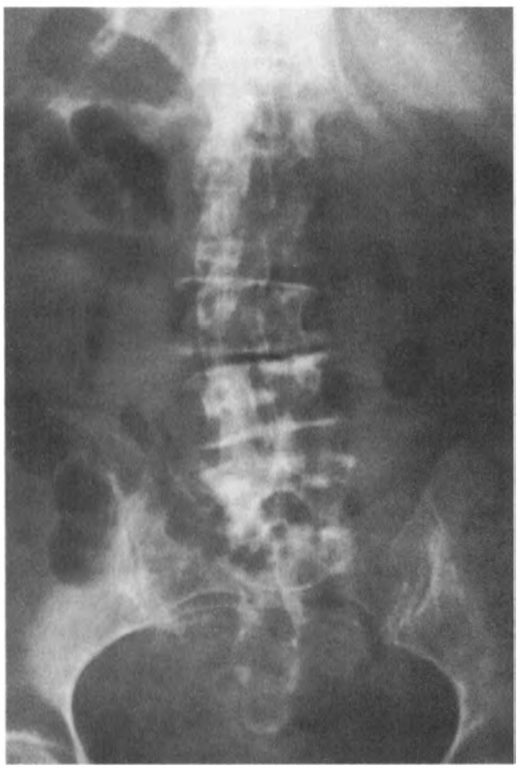

Figure 9.89. Degenerative levorotatory scoliosis of the lumbar spine with the apex at the L3–L4 level and osteochondrosis of this disc space noted. Atherosclerosis of the abdominal aorta is seen.

Figure 9.91 shows thoracic rotatory movement applied to the lumbar segments. Grasping the spinous process of the lumbar segment, the vertebral segment was gently placed into right and left rotation. More rotation to the left was used in an attempt to gently derotate the left posterior rotatory subluxation of the vertebral body. (We certainly realized that Wolff's law had altered structure so that no permanent correction of the subluxation was

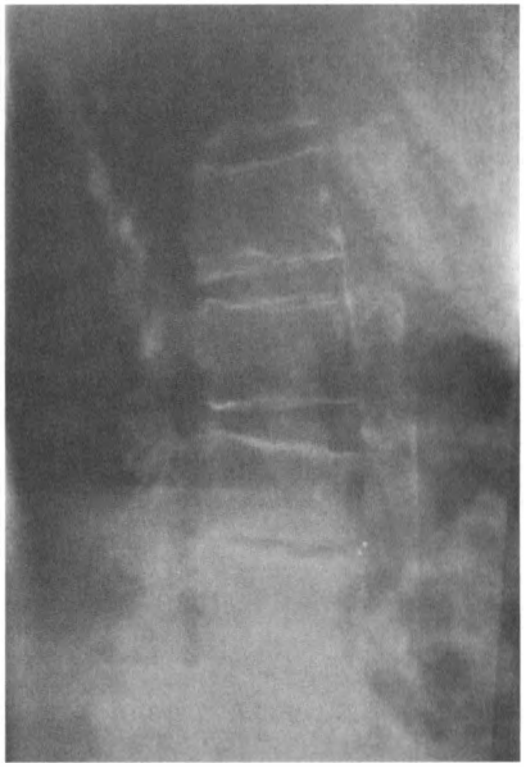

Figure 9.90. Lateral projection of the patient seen in Figure 9.89 shows the L3–L4 advanced degenerative disc disease with loss of lordotic curve. The intervertebral foramina at the midlumbar levels appear narrowed sagittally and vertically compared with the upper lumbar levels. The aortic arteriosclerosis is seen.

Figure 9.91. The left posterior lumbar vertebral body rotations are adjusted by rotating the thoracic section of the table to allow the left vertebral body rotation to rotate anteriorly as the spinous process of the segment being manipulated is held in the midline.

possible.) The goal was to restore maximal motion to this segment. This movement as shown in Figure 9.91 was repeated to each lumbar segment from L1 to L4.

Figure 9.92 shows maintenance of the left derotation movement of the thoracic section on which the lumbar segments had just been derotated. Contact was made well above the rotatory scoliosis, in this case at the lower thoracic segments, and gentle distraction applied to the spine by taking the caudal section of the table downward slowly while lifting the spinous process of the thoracic segment with thenar contact of the right hand. Moving down, one vertebra at a time, careful and gentle slight distraction was applied to the L4 segment.

Figure 9.93 shows the lateral flexion of the caudal section of the table to the left side to gently stretch the lumbar scoliosis into its left convexity. Here, therefore, three movements—flexion, left derotation, and left lateral flexion—were applied to the lumbar levoscoliotic curve. (Note: If the patient felt any discomfort to any such motion, the adjustment would be stopped and only motion applied that caused no discomfort.)

If the patient felt too much discomfort to lie prone, or if the treatment caused discomfort when lying on the abdomen, treatment was applied with the patient lying on the side.

Figure 9.94 shows the patient lying on the side with the convexity—in this case, the left side—down on the table. This was done to allow reduction of the levorotation component of the curve to a slight degree, and, depending on patient tolerance, lowering the caudal section of the table as shown in Figure 9.94. The spinous process of the lumbar vertebra was contacted , as shown in Figure 9.95, and the table brought into forward lateral motion to apply a mild flexion to the lumbar spine while palpating the interspinous space for fanning (opening of the interspinous space). Figure 9.96 shows the lumbar spine being placed into mild, carefully controlled extension while feeling the interspinous spacing for motion. The lumbar spine could also be placed in flexion or extension by moving the caudal section of the table laterally, and while in this position applying a downward motion with the caudal section to laterally flex and derotate the lumbar spine. *All such adjustments are done very slowly and carefully while monitoring patient comfort or complaint.*

Figure 9.97 shows the foramen magnum pump technique applied. Here the basiocciput is cradled in the hand and full spine

Figure 9.92. The left posterior lumbar vertebral body rotation subluxations are held in derotation by locking the midsection of the adjusting table in the position of derotation. Distraction is then applied by placing the thenar contact of the right hand under the spinous process of the vertebrae above the scoliotic curve. The caudal section of the table is then placed into downward distractive position. This figure shows a coupled adjustment of left derotation and traction being supplied.

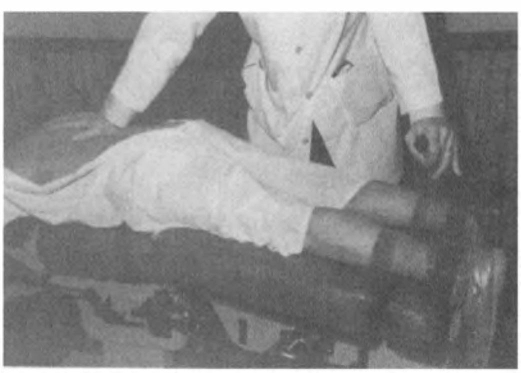

Figure 9.93. With left derotation and traction applied, gentle left lateral flexion of the spine is introduced by placing the caudal section of the instrument into left lateral flexion. This is done very gently, with patient comfort monitored at all times.

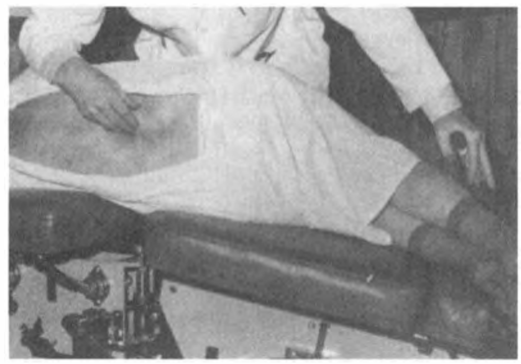

Figure 9.94. For patients who feel pain when treated lying on the abdomen, the adjustment can be delivered with the patient lying on the side, as shown here. When treating the spine shown in Figures 9.89 and 9.90, we would have the patient lie on the left side to allow the levorotation of the lumbar segments to be reduced by posture alone. A small pillow may also be placed under the lumbar spine to enhance the effect of reducing the left lumbar spinal curvature. *Note:* by placing the caudal section of the table into flexion, you can see that lateral flexion of the lumbar spine is applied.

Figure 9.95. Traction is applied by forward bending of the caudal section of the table to open the lumbar spinous processes. The doctor's right hand palpates the spinous processes to detect fanning (opening of the spinous processes) while this maneuver is carried out. *Note:* in patients with atherosclerosis of the abdominal aorta, this form of care prevents pressure on the arterial system.

Figure 9.96. Extension can also be applied by lateral bending of the caudal section posteriorly while again monitoring the interspinous spaces with the palpating hand.

Figure 9.97. Foramen magnum pump technique applied for full spine distraction adjustment. Here the basiocciput is cradled in the doctor's right hand. Downward caudal traction is applied while the occiput is gently lifted cephalad. Upper cervical tension, headaches, cervical muscle spasm, zygapophysial degeneration and subluxation, discal degeneration, and cephalic tension are helped by this technique.

Figure 9.98. The traction shown in Figure 9.97 is continued down the cervical spine by contacting the spinous processes and laminae of each cervical vertebra and repeating the distractive pull until separation of the interspinous space is felt. As you continue down the cervical spine, the thoracic spinous processes are felt to press into the web between the thumb and index finger. At that time firmly contact the thoracic spinous processes between the web of the thumb and index finger, and continue to apply the cephalad distraction throughout the thoracic spine. This can be carried out throughout the scoliotic curve. Always be mindful of patient comfort when applying the technique. Monitor patient comfort at all times.

distraction applied. This results in a mild full spinal tractive force and patients often state that they feel as though it would feel good if someone pulled them apart. I think they are stating that the effects of gravity in compression of the spine are painful to their disc, facets, and supporting elements. The foramen magnum pump is used in many conditions, one of which is degenerative scoliosis, but always slowly and gently, as patient tolerance allows. Figure 9.98 demonstrates how this distraction is continued down into the thoracic spine by tractioning the thoracic segment spinous process cephalad, grasping it in the web of the contact hand between the thumb and first finger. Downward distraction is applied gently with the caudal section of the table as the spinous contact is lifted cephalad.

Other treatment of this patient included home exercises consisting of *gentle* knee-chest procedures. The patient was told to precede these exercises by applying 15 minutes of heat to the low back, followed by 10 minutes of cold, followed again by 15 minutes of heat. This relaxes the spinal muscles and makes the exercises less irritating. In this type of case, knee-chest is the only maneuver we recommend for home exercise. Too many exercises tend to aggravate this type of spine. The chiropractic adjustment was followed with positive galvanic current into the L3–L4 area and then mild tetanizing currents to the paravertebral muscles from L1–L2 to L4–L5, with moist heat applied concurrently with 15 minutes of electrical stimulation. This patient attended low back wellness school to learn the proper ways to lift, bend, and twist in daily living, to reduce strain to her spine. She also was given 1000 mg of nonphosphorous calcium per day to take orally and encouraged to walk as much as her stamina allowed.

This combination of therapy was applied three times weekly for 3 weeks and then two times weekly for 2 weeks, with the result that the patient reported approximately 50% relief and certainly felt positive about having undergone this conservative approach to her problem. In the end, success is achieved when the patient feels that the relief obtained is greater than the expense or inconvenience of therapy.

Discogenic Spondyloarthrosis

The most common condition seen in a manipulative practice is probably the degenerative disc with resultant facet weight-bearing increase, which results in the clinical entities of facet arthrosis and disc spondylosis that result in a condition termed "discogenic spondyloarthrosis." Middle-aged to elderly people are susceptible to this condition as the nucleus pulposus dehydrates; the opposing vertebral body plates approximate one another, with loss of disc space height and subchondral end plate sclerosis. The person becomes shorter in stature and may become stooped if stenosis of the canal accompanies these changes. Such stooped posture affords a greater sagittal diameter of the vertebral canal. These patients often state that it would feel good if somehow they could be "pulled apart" or tractioned. This condition, therefore, can be effectively treated by flexion distraction adjustments while monitoring patient tolerance.

By working within patient tolerance, the doctor can distract the specific disc space and facet joints while placing the facet joints through their normal ranges of motion, which are *flexion, extension, lateral flexion, circumduction, and rotation.* A vertebra capable of performing its physiologic ranges of motion is less encumbered with subluxation and the resulting nerve root irritations accompanying it. This technique can increase the

Figure 9.99. Note the levorotation of the lumbar segments, the loss of disc space and hypertrophic changes of the anterior lateral body plates at L3–L4 and L4–L5, and the transitional changes of the L5 segment.

Testing for patient tolerance of traction, as demonstrated in Figure 9.103, is performed before the ankle cuffs are applied. This is done by grasping the ankle and applying traction while asking whether the patient feels any pain in the low back. Muscle resistance can be felt in patients who cannot tolerate traction. If no pain is felt, the cuffs are attached and flexion is applied as shown in Figure 9.102.

Lateral flexion, demonstrated in Figure 9.104, is performed by grasping the spinous process of each lumbar segment individually between the thumb and index finger (Figure 9.105). Motion palpation is elicited by testing the ability of the articular facets to bend laterally during movement of the caudal section of the table in lateral flexion. Hypomobility is evidenced by resistance to movement laterally, pain to the patient, or both.

Circumduction, which is a combination of lateral flexion and plain flexion, is demonstrated in Figure 9.106. This coupled movement of the table allows full range of motion of the facet and is effective in restoring mobility to the facet.

Rotation, as demonstrated in Figure 9.107, is applied by rotating the caudal section of the table while the vertebral segment is held in resistance. Traction can be applied prior to this movement and maintained during rotation by leaving the ankle cuffs on the patient and opening the caudal section of the table. Keep in mind that L4–L5 and L5–S1 have restricted range of motion of an articulation previously considered degenerated and nonmobile until the patient is pain-free, or at least in less pain, and is able to perform a range of motion not previously possible.

Figures 9.99–9.101 present a typical case seen in clinical practice almost daily. Figures 9.99 and 9.100 are the anteroposterior and lateral views showing degeneration of the lower three lumbar discs, with the oblique view in Figure 9.101 revealing the facet imbrication that follows disc degeneration and the resultant increased weightbearing on the facet. This causes the facet joint to imbricate upward into the intervertebral foramen, resulting in lateral recess stenosis. Note that the inferior facet of the L4 vertebra tends to contact the lamina of the L5 vertebra below, which results in periosteal reaction with sclerosis. This has been termed "facet-lamina syndrome" and is considered a source of pain. Also note how the superior facet tends to telescope upward to contact the pedicle of the vertebra above, resulting in periosteal sclerosis as well, which is shown in Figure 9.101.

Treatment

In Figure 9.102, flexion is being applied to each lumbar disc space and facet facing. By maintaining hand contact with the spinous process of each lumbar and thoracic vertebra, the downward pressure on the caudal section of the table allows stretching and spreading apart of each functional spinal unit.

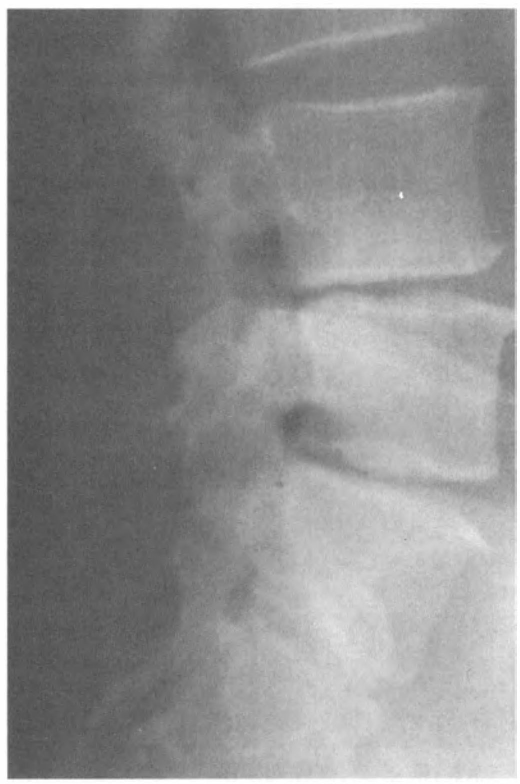

Figure 9.100. Lateral view of Figure 9.99 shows degenerative L3–L4 and L4–L5 disc disease with stenosis of the intervertebral foramina at these levels. The rudimentary disc of L5–S1 is seen at this level of transitional segment. This is Bertolotti's syndrome (i.e., a transitional L5 segment with degenerative disc disease at the disc level above).

Figure 9.101. Oblique views of the patient seen in Figures 9.99 and 9.100 show the L3–L4 and L4–L5 facet joints to have loss of joint space with subchondral sclerosis, and to imbricate superiorly into the intervertebral foramen to create stenosis of the osseoligamentous canal. With this imbrication, we find that the superior tip of the superior facet contacts the pedicle of the vertebra above and the inferior tip of the inferior facet contacts the lamina of the vertebra below. This creates some periosteal reaction, which could be a source of back pain. This is termed the facet-lamina or facet-pedicle syndrome.

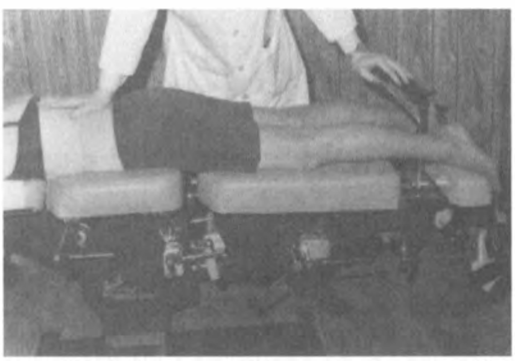

Figure 9.102. Flexion-distraction manipulation.

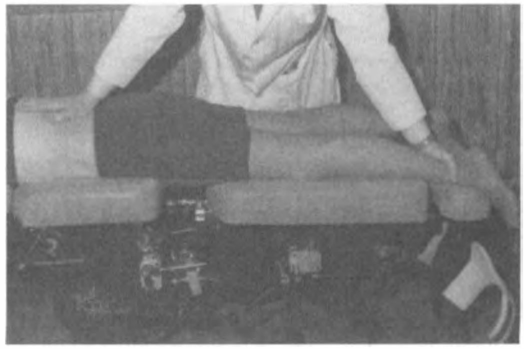

Figure 9.103. Testing patient tolerance to distraction before applying distraction cuffs.

Figure 9.104. Lateral flexion being applied to the articular facets.

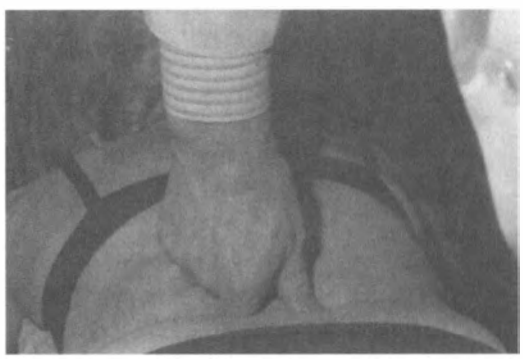

Figure 9.105. Grasping of the spinous process above the facets to be motion-palpated and manipulated.

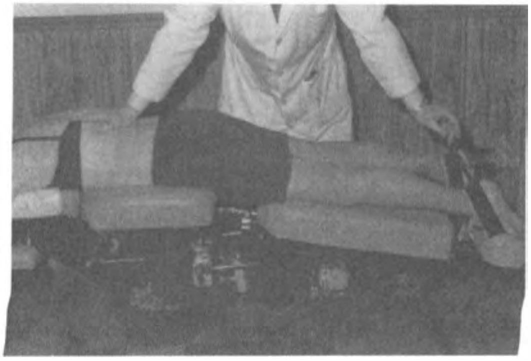

Figure 9.106. Circumduction manipulation.

Figure 9.107. Rotation being applied to the thoracolumbar spine.

ranges of motion in rotation and should not be forced into rotation. The upper lumbar and thoracic segments are capable of rotation.

Rotation and flexion as applied simultaneously to the upper lumbar and thoracic segments are demonstrated in Figure 9.108. This coupled mobilization is powerful and must be done to patient tolerance.

Goading of acupressure bladder meridian points B24 and B35, as demonstrated in Figure 9.109, is performed prior to and after distraction.

Deep pressure into the belly of the gluteus maximus muscle and bladder meridian point B49, as demonstrated in Figure 9.110, is used to relieve the pain of sciatica.

Pressure being applied to the adductor and gracilis tendons at their origin is demonstrated in Figure 9.111; pressure being applied to their insertions on the medial femur and medial condyle of the tibia is demonstrated in Figure 9.112.

Application of the "foramen magnum pump" is demonstrated in Figure 9.113. It is performed by grasping the occiput while applying traction to the full spine with caudal distraction.

The application of heat and sinusoidal muscle stimulation or ultrasound with sinusoidal currents, either before or after manipulation, also provides relief from pain for patients with discogenic spondyloarthrosis.

Other considerations important in the treatment of the patient with a degenerative low back include:

1. Nutrition. Osteoporosis is a common accompanying factor with the older spine. Therefore, amino acids are needed to build osteoid tissue and calcium is needed to aid in bone ossification. Supplements of these are recommended and prescribed. Manganese (500 to 800 mg/day), which is an ingredient of Discat, a nutritional supplement containing glucosaminoglycan, is also prescribed. Niacin (200 mg/day) and vitamin B_6 (150 mg/day) are also recommended. Bowel alkalinity depresses the absorption of calcium and, because *of the low output of HCl and enzymes in the elderly*, may cause osteoporosis and endocrine hyposecretion. Thus, digestive enzymes are also prescribed.
2. Exercise. Walking improves the circulation and increases the muscular activity of the paravertebral musculature, thereby enhancing the flow of nutrients to the bone tissue and the elimination of waste materials. Thus, exercise is recommended for patients with discogenic spondyloarthrosis.
3. The gracilis tendon should be tested and strengthened.
4. Low back wellness school is presented to these patients so that they learn the proper methods of lifting, bending, and twisting in daily living. They are shown how to perform the Cox exercises. If patients are not drilled on these exercises, or if they are merely given a sheet of exercises and told to do them, either they will not do them or, even worse, do them incorrectly. A videotape of the entire exercise program is given to the patient to follow and perform at home.

Compression Fracture of the Thoracic or Lumbar Vertebral Bodies

The treatment described here is intended for the compression-type fractures that result in trapezoid-shaped vertebral bodies. Pathologic compression fractures are not treated with a manipulative adjustment approach. Figures 9.114 and 9.115 show serial studies of a compression fracture of the ninth thoracic vertebral body following a fall. The radiograph in Figure 9.115 was taken 4 months following the radiograph in Figure 9.114, thus showing the progressive nature of the compression fracture. Remember that fracture severity can increase in the weeks following the initial injury and its discovery. This is especially true in the osteoporotic elderly female spine.

The treatment of the fracture seen in Figures 9.114 and 9.115 is shown in Figures 9.116–9.118. For further treatment technique for compression defects, see Figure 9.37.

Long-Term Results of Conservative Care of Thoracolumbar Fractures

A long-term study of 216 patients without neurologic complications who sustained thoracolumbar compression fractures was carried out for an average of 9 years. The functional results of single versus multiple fractures were no different, nor was the degree of spontaneous fusion found to cause any statistical difference in the functional outcome. No correlation was found between reduction in vertebral height, encroachment on the spinal canal, and persistent kyphotic deformities. It was concluded that nonoperative treatment of these fractures was a

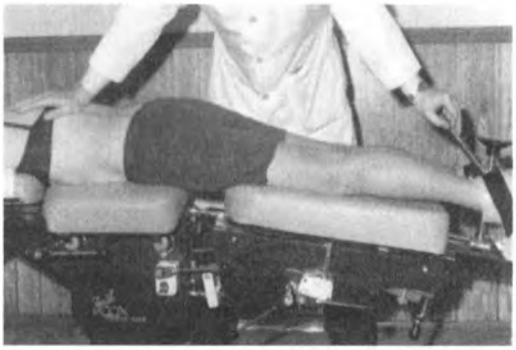

Figure 9.108. Rotation and flexion distraction being applied simultaneously.

Figure 9.109. Acupressure points B24 to B35 being goaded.

Figure 9.110. Acupressure being applied to the gluteus maximus and bladder meridian point B49.

Figure 9.111. Goading of the adductor and gracilis tendons at their origins.

Figure 9.112. Insertion of the gracilis tendon being goaded at the medial tibial condyle.

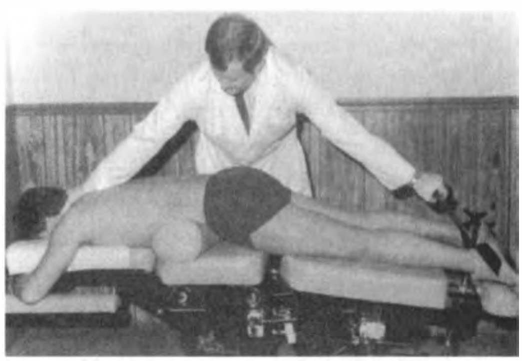

Figure 9.113. Application of the "foramen magnum pump" in full-spine occipital distraction.

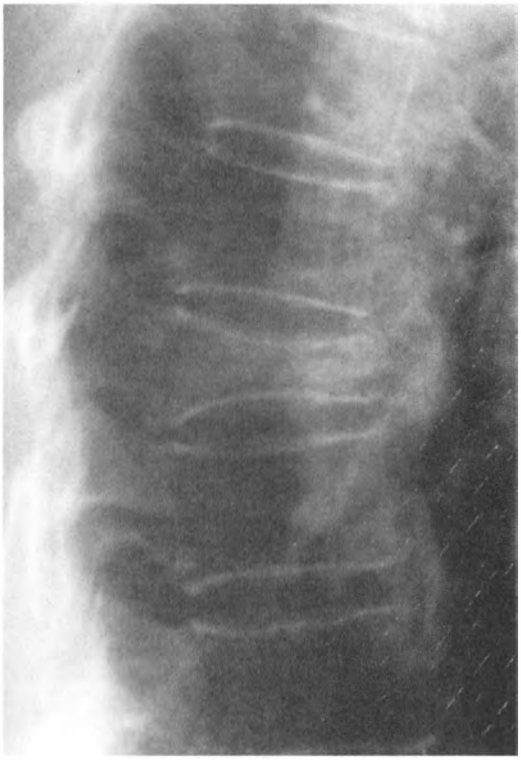

Figure 9.114. The ninth thoracic vertebra shows compression fracture and about 60% loss of the normal height. This is the result of a fall.

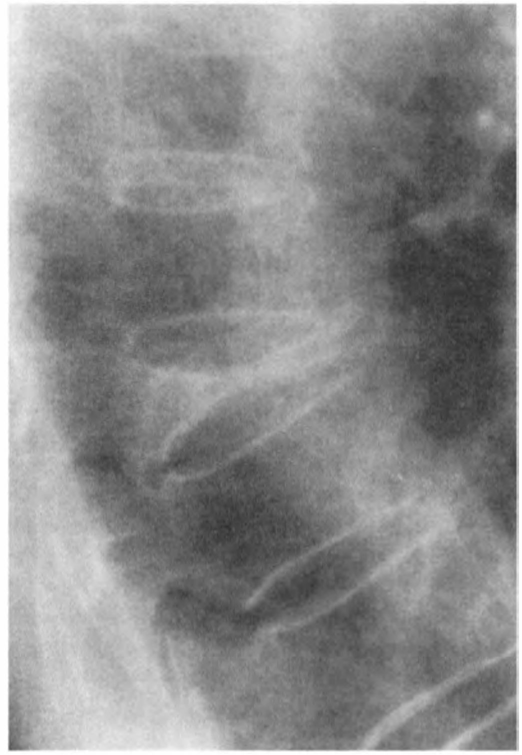

Figure 9.115. The fracture shown in Figure 9.114 again shown 4 months later; it now shows progressive trapezoid-shaped collapse, with about 90% loss of the normal vertebral body height.

sound method and that attempts at surgical reduction were not justifiable. None of the 216 patients required surgical reduction because of persistent symptoms (1).

Weinstein et al. (2) also found that nonoperative treatment of thoracolumbar burst fractures was a viable alternative to surgery in patients without neurologic deficit and that such conservative care resulted in acceptable long-term results.

Failed Back Surgery Syndrome

Recurrent herniated disc and symptomatic hypertrophic scar can produce similar low back symptoms and radiculopathy. Gradually escalating symptoms beginning a year or more after discectomy are considered more likely to be caused by scar radiculopathy, whereas a more abrupt onset at any interval after surgery is more likely caused by a recurrent herniated disc (3).

Failed back surgery syndrome is seen in 10 to 40% of patients who undergo back surgery. It is characterized by intractable pain and varying degrees of functional incapacitation occurring after spine surgery (4).

Epidural adhesions can occur with no previous treatment of low back pain or sciatica in some patients. Primary formation of epidural adhesions in the epidural space could explain why treatments sometimes fail and why surgery should be avoided in patients whose CT or myelograms are negative for nerve root compression (5).

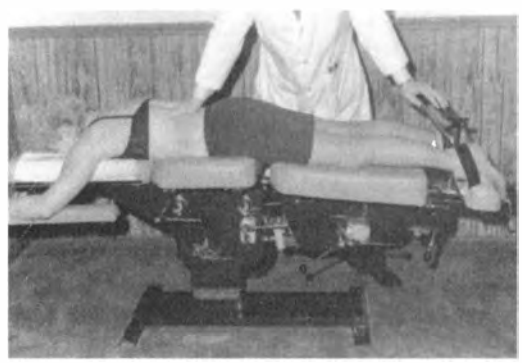

Figure 9.116. Mild flexion distraction being applied.

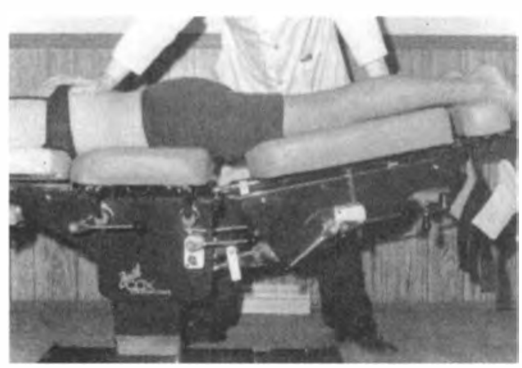

Figure 9.117. Mild extension manipulation being applied.

Figure 9.118. Treatment of the fracture seen in Figures 9.114 and 9.115 is shown here. The compression defect is placed over the split sections of the adjustment table. With a gentle anterior pressure applied to the spinous process of the compressed segment, the caudal section of the table is gently brought into extension. This places the flexion deformity created by the compression fracture to be brought into extension. This treatment is applied to patient tolerance and until the spine is felt to gain some measure of extension motion. This patient, as is true with all compression fractures, is advised to hold the spine in extension by wearing an extension support, lying over a small pillow under the thoracic segment, and performing extension exercises of the thoracic spine.

Differentiation of Recurrent Disc Herniation from Scar Formation

Epidural scarring and adhesions can be differentiated from recurrent disc herniation by intravenous contrast-enhanced CT scan of the postoperative spine (3). Gd-DTPA (gadolinium-diethylene-triaminepentaacetic acid/dimeglumine) enhanced MRI is also used (4). Scar tissue is enhanced by the contrast agent, whereas the disc material is not enhanced. A study shows that precontrast and early postcontrast T1-weighted spin-echo studies are highly accurate in separating epidural fibrosis from herniated disc (4).

Repetitive back surgery is the unfortunate consequence of persistent pain, although improvement from additional operations is very slight. De La Porte and Siegfried (6) state that Ohio Workmen's Compensation reported that no patients condition was cured by a second low back operation, 20% improved, 20% were made worse, and 60% were essentially unchanged. With additional operations the outcome worsens, and after four operations, 5% were improved and 50% were made worse.

The clinical features of lumbosacral spinal fibrosis are polymorphic. Lumbar pain and sciatica that become worse, even with minimal physical activities (seen in 60% of patients), are the main complaints. Nocturnal cramps and distal paresthesia are common. Twenty-five percent of patients have low back pain without radiculopathy. Ten percent show cauda equina syndrome with sphincter dysfunction and saddle hypesthesia. Lasègue's sign is positive only in 20% of the cases, but the absence of knee and ankle reflexes is frequent. The syndrome of spasm in the legs, muscular cramps, increasing radicular pain, elevated temperature, and shivering occur within the first 3 days following surgery, which may signify the first signs of spinal fibrosis (6).

In patients with epidural scar fibrosis, additional surgery can only magnify the scarring and resultant disability. One approach, used when all other conservative measures fail, is the application of epidural stimulation with an electrode lead wire anchored deeply into the epidural space. A percutaneous wire extends out through the skin and is attached to a small (pocket watch-sized) pulse generator implanted beneath the skin. A gentle buzzing sensation is imparted to the dorsal columns to produce a stimulus that acts as a signal jamming the chronic pain sensations that occur with nerve damage (7).

Postsurgical Failed Back

Case 2

A 43-year-old white single man was seen with the chief complaints of low back and right leg pain, and, occasionally, some pain into the left leg. The patient had back surgery performed twice, the first time in 1967 for a laminectomy and in 1968 for a spinal fusion. He noted that his back pain returned immediately following the surgeries. He had been seen at many clinics without relief of pain.

This patient also complained of neck pain and pain in the right shoulder, arm, and hand. Neck pain had started approximately 20 years previous, following an injury, at which time he was told he had a cervical disc problem.

Examination of the low back revealed marked restriction of range of motion, with flexion at 40°, extension at 5°, right and left lateral flexion at 10°, and rotation at 20°, all of which were accompanied by pain. Straight leg raising was bilaterally painful at 50°, creating leg pains. The muscle power of the lower extremities was grade 5 of 5 bilaterally. The right ankle reflex was absent, whereas the remaining deep reflexes of the lower extremities were +2 bilaterally. No sensory changes were noted on pinwheel examination. The circulation of the lower extremities was good.

Radiographic examination revealed the following: Figure 9.119 shows an extensive interlaminal fusion at the L4–S1 levels. Figure 9.120 is a lateral projection that reveals advanced degeneration of the L4–L5 and L5–S1 disc spaces with the posterior fusion in place. Figure 9.121 is an oblique projection, again outlining the bone fusion between the laminae at L4–L5 and the sacrum.

Figure 9.122 shows a lateral cervical radiograph of this patient, revealing extensive degenerative disc disease at the C5–C6 and C6–C7 levels. The oblique view in Figure 9.123 reveals the right C5–C6 intervertebral foramen to be somewhat narrowed because of degenerative disc disease at that level.

Examination of the cervical spine, physically, orthopedically, and neurologically, revealed reduction of ranges of motion on rotation to approximately 70°, with otherwise normal ranges of motion. Palpation revealed pain over the C4 through C7 levels bilaterally, with cervical compression being positive at the C5, C6, and C7 levels, radiating pain into the right shoulder and arm. No signs were evident of thoracic outlet syndrome. The deep reflexes of the upper extremities were +2 bilaterally, with no sensation changes to pinwheel examination. No motor weakness was noted in either upper extremity.

Our impressions of this case were as follows: (*a*) degenerative disc disease at the C5–C6 and C6–C7 levels, creating some foraminal stenosis and a resultant right brachial radiculopathy; (*b*) spinal fusion, interlaminar, at L4–L5 and the sacrum with advanced degenerative disc disease at the L4–L5 and L5–S1 levels; and (c) possible postsurgical stenosis at the L4–L5 and L5–L1 levels.

Figure 9.119. L4–S1 interlaminar spinal fusion, anteroposterior view.

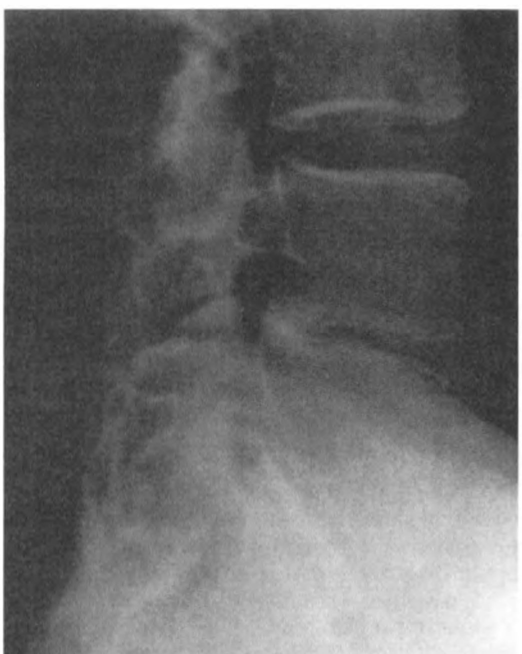

Figure 9.120. Lateral view of Figure 9.119 showing the spinal fusion with the extensive L4–L5 and L5–S1 discal degeneration.

Figure 9.121. Note the bone fusion on oblique view.

Figure 9.122. Lateral cervical spine radiograph of the patient in Figures 9.119 through 9.121. This figure shows C5–C6 and C6–C7 degenerative disc disease. I note that disc degenerative changes occur in those spinal segments where rotation is a minimal motion and flexion and extension are primary motions. Such areas occur at the L4–L5, L5–S1, C5–C6, and C6–C7 levels.

Figure 9.123. Oblique view of patient seen in Figure 9.122 does show some narrowing of the C5–C6 intervertebral foramen due to discal degeneration and loss of vertical height of the foramen.

This patient was given flexion distraction of the C5–C6 and C6–C7 levels, followed by ultrasound with mild tetanizing current.

The lumbar spine was treated by goading of acupressure points B22 through B49 and flexion distraction for the L3–L4 segment. The reason for this is that, with the fusion of L4 to the sacrum, all of the flexion, extension, and lateral bending motions have been transferred to the L3–L4 level. Maintaining complete ranges of motion with minimal stress can help to alleviate and prevent future degenerative change at the L3–L4 level. This will be the level of motion of this patient's spine for the rest of his life.

In addition, we used tetanizing current to the paravertebral muscles of the lumbar spine and pelvis, with alternating hot and cold packs. Treatment of postsurgical backs can be extremely difficult, especially when sciatic pains are present. In this case, the patient became discouraged by slow relief of pain and discontinued treatment before meaningful clinical treatment could be administered and underwent further surgical decompression and fusion.

Treatment of Failed Back Surgery Syndrome

Spinal manipulative therapy for the patient with the failed back surgery syndrome is applied under strict parameters:

1. Never is the caudal section of the table lowered more than 2 inches.
2. Rotation is never applied to the lower lumbar spine.
3. No electrical intermittent traction is used—only hand-controlled manual manipulation.
4. Any lateral flexion is restricted to facet capability; lateral bending should never be forced.
5. Flexion is the primary motion used.
6. Traction is applied above the fused segments.

Flexion of the spinous process is applied above the fusion, as is demonstrated in Figure 9.124. The rules for applying traction, which were given previously, are followed. In Figure 9.125, lateral flexion of the segments is demonstrated.

Sinusoidal currents are applied to the paravertebral muscles as demonstrated in Figure 9.126. Hydrocollator packs are applied over the sinusoidal current pads for 10 minutes (Fig. 9.127). Cold packs are then applied for 5 minutes (Fig. 9.128). Hot and cold packs, beginning and ending with heat, are applied alternately.

Figure 9.129 shows unilateral traction being applied without the use of ankle cuffs; the ankle is held while distraction is being applied. By holding each lower extremity, the facets can be more strongly tractioned unilaterally.

Distraction Arthroplasty Patients

Figures 9.130 and 9.131 are radiographs of a patient with hip arthroplasty. Commonly, these patients also have degenerative disc disease and are best treated unilaterally, as shown in Figure 9.129, to control traction on the involved replaced hip socket. Distraction can be used without cuffs on the patient (see Fig. 9.102).

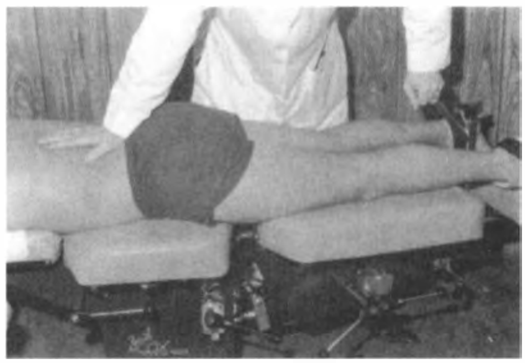

Figure 9.124. Contact is maintained on the spinous process above the surgical fusion shown in Figure 9.119.

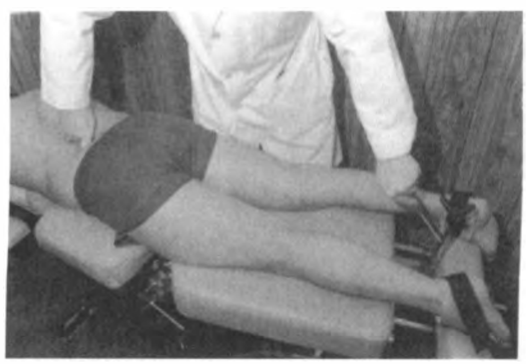

Figure 9.125. Lateral flexion being applied to the same patient shown in Figure 9.119.

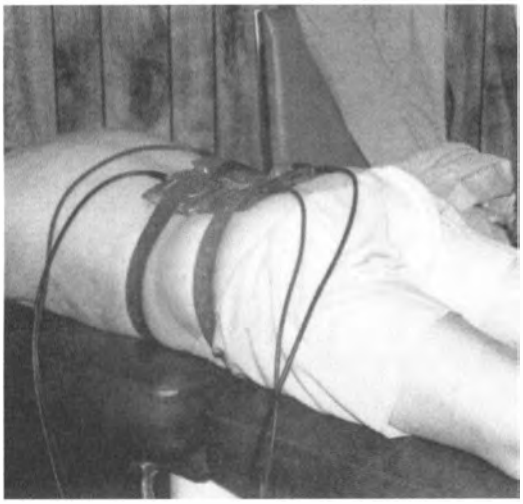

Figure 9.126. Sinusoidal current being applied.

Figure 9.127. Moist heat being applied.

Figure 9.128. Cold packs being applied to the low back and sciatic distribution.

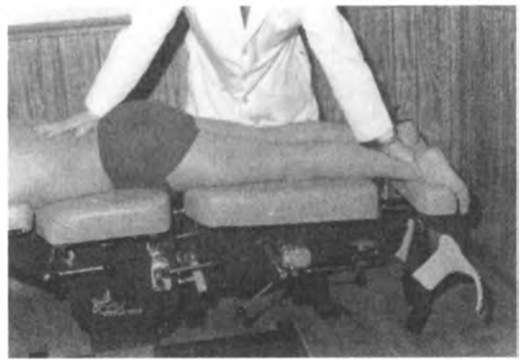

Figure 9.129. Unilateral distraction being applied.

Figure 9.130. Anteroposterior view of the lumbar spine and pelvis of a patient with hip arthroplasty.

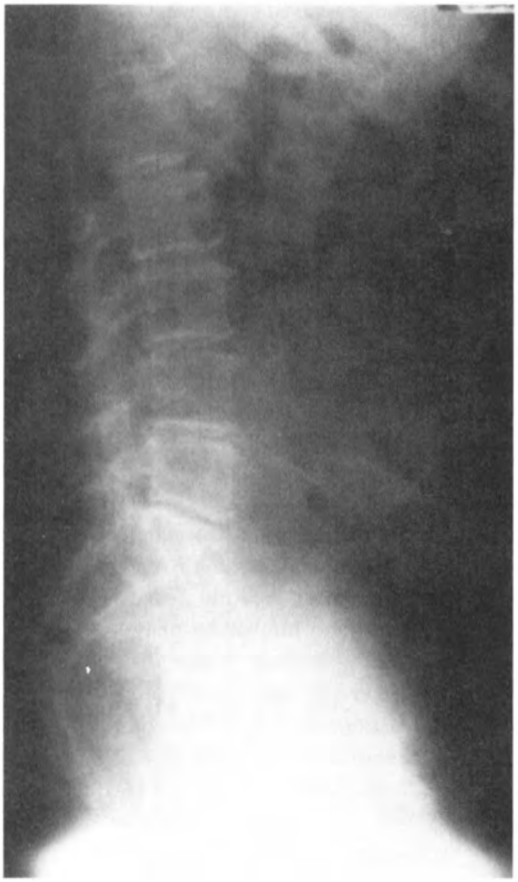

Figure 9.131. Lateral view of the same patient shown in Figure 9.130.

Figure 9.132. Shown is the correction of an anterior innominate subluxation. The patient's knee is flexed, and the doctor directs cephalad pressure against the popliteal space as an anterior thrust of the ischial spine of the ischium is given.

Figure 9.133. Patient with pes planus accompanying low back pain.

Figure 9.134. Medial view of patient in Figure 9.133.

EFFECTS OF CHRONIC LOW BACK PAIN ON FUNCTIONAL STATUS

Patients suffering from chronic low back pain have significant impairment in physical, psychosocial, work, and recreational activities. The greatest impairment is in the area of work, but disability ranges for recreation, home management, social interaction, emotional behavior, and sleep and rest are also comparatively high. In persons with chronic low back pain, the use of a sickness impact profile, which is a global measure of disability, is valid as a measure of functional status. The results of this test assist in the evaluation of the efficacy of multidisciplinary pain units (8).

TWO FINAL FACTORS IN TREATING LUMBAR DISC DISEASE

Anterior innominate subluxations are particularly problematic when they occur on the same side of disc lesion. I feel that the anterior ilium causes the sacrospinous ligament to traction the sciatic nerve over itself and to aggravate the leg pain. Therefore, when the patient has a longer leg on the side of sciatica, we adjust the innominate as shown in Figure 9.132. Note that rotation of the lumbar spine is avoided by flexing the hip joint, contacting the ischium, and delivering the corrective manipulation without inflicting any rotation to the lumbar spine which might tear the disc anular fibers.

Whenever dropped arches create pes planus deformity of the foot, as shown in Figures 9.133 and 9.134, we place orthotics made from foot casts into the shoes to correct this fault (Fig. 9.135).

Figure 9.135. The foot orthotic used to correct pes planus.

Chapter 15 by Scott Chapman, DC, DABCO details active rehabilitation procedures for the low back pain patient. It should be read and implemented with this chapter.

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CHIROPRACTIC SPECIALIZATION IN LOW BACK PAIN IS BECOMING A REALITY

When discussing training of chiropractic doctors in the specialized field of low back pain, I think of Crockard (1) who called for the next generation of neurosurgeons and orthopaedic surgeons to generate the spinal surgeon, the surgeon who embraces only the spine as a specialty as the hand surgeon or maxillofacial surgeon specializes. To paraphrase Saint Augustine on chastity: these groups want spinal surgery, but not pure spinal surgery yet. No surgeon can be expected to clip a cerebral aneurysm, remove a meniscus through an arthroscope, and perform pedicle screw fixation of the lumbar spine with equal facility.

Ben Eliyahu (2) reported treating 27 MRI documented and symptomatic cervical and lumbar herniated disc cases with a course of care including traction, flexion distraction, spinal manipulation, physiotherapy, and rehabilitative exercises. Clinically, 80% of the patients had a good outcome with postcare visual analogue scores under 2 and resolution of abnormal clinical examination findings. Repeat MRI showed 63% of the patients had a reduced size or completely resorbed disc herniation. Seventy-eight percent of the patients returned to work at their predisability occupations.

The severe low back pain patient can demand skill and ability of a chiropractor trained in specific clinical protocols. Thus the creation of the specialist in distraction adjusting of the low back exists by the certification course fostered and nurtured between myself and the National College of Chiropractic in 1991. In excess of 1000 chiropractic physicians now are certified and National College maintains a list of chiropractic physicians who are certified in these procedures.

It is incumbent on chiropractors to rely on meaningful patient outcomes such as the 1000 case study and the algorithms of decision-making in diagnosis and treatment presented here to determine the patients' clinical health disposition. Cox distraction-adjusting and diagnostic protocol is an important treatment technique and an important component in the care of the low back pain patient.

Diagnosis of the Low Back and Leg Pain Patient

James M. Cox, DC, DACBR

chapter 10

The quality of a person's life is in direct proportion to their commitment to excellence, regardless of their chosen field of endeavor.

—Vince Lombardi

LOW BACK PAIN TERMS

Definitions

Transient back pain: An episode in which back pain is present on no more than 90 consecutive days and does not recur over a 12-month observation period.

Recurrent back pain: Back pain present on less than half the days in a 12-month period, occurring in multiple episodes over the year.

Chronic back pain: Back pain present on at least half the days in a 12-month period in single or multiple episodes.

Acute back pain: Pain that is not recurrent or chronic (as defined above) and whose onset is recent and sudden.

First onset: An episode of back pain that is the first occurrence of back pain in a person's lifetime.

Flare-up: A phase of pain superimposed on a recurrent or chronic course. A flare-up refers to a period (usually a week or less) when back pain is markedly more severe than is usual for the patient (1).

Classification of Spinal Pain

Acute pain: Immediate onset, with a duration of 0 to 3 months.

Subacute pain: Slow onset, with a duration of 0 to 3 months.

Chronic pain: Duration is longer than 3 months, regardless of onset.

Recurrent pain: Intervals during which no symptoms are present, but pain reappears (2).

Lumbar Spine Pain Classified by Location and Distribution

Local pain: lower lumbar or lumbosacral pain (lumbago).

Referred pain: Pain experienced at the area that shares a common embryologic origin with the region involved. It is usually located to the inguinal or buttock region or the anterior, lateral, or posterior thigh. In some cases, however, it might be distributed even below the knee.

Radicular pain: Pain distributed along the dermatomal distribution of a spinal nerve root and is caused by a direct affection of the nerve tissue (Fig. 10.1). It is most commonly experienced along the course of the sciatic nerve, depending on the spinal level of the involved nerve root.

Sciatica: Sciatica literally means "related to the hip." The first time this term was seen in the literature was not in a scientific paper, but in a play by William Shakespeare entitled "Timons of Athens" where the character Timons cries out, "Thou could sciatica, cripples our senators as lamely as their manners." The first pathoanatomic definition of sciatica was in 1576 by Domenico Cortugno who stated sciatica was a local affection of the sciatic nerve in the thigh (2).

Malignancy-induced Low Back Pain

I give this problem a major classification because chiropractic doctors are presented with metastatic and primary tumor-induced low back pain.

Malignancy involves the lumbar spine much more often than the cervical spine. At least two thirds of spinal malignancies are metastatic rather than primary, usually spreading from a tumor in the breast, lung, prostate, or kidney.

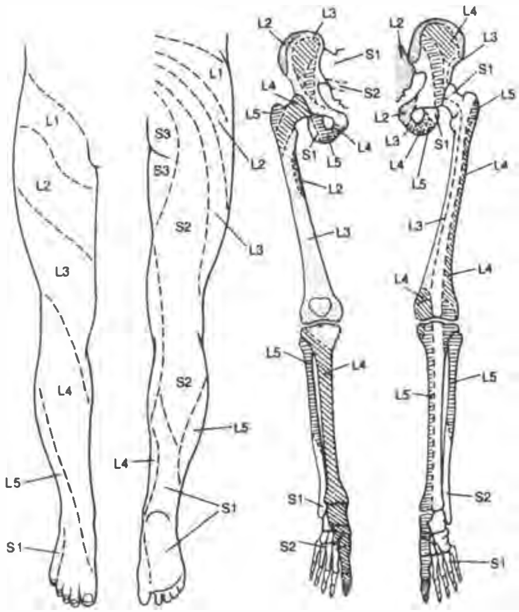

Figure 10.1. To the **left** is the dermatomal distribution of the innervation by each nerve root level demonstrated. To the **right** is the corresponding sclerotomal innervation. (Reprinted with permission from: Olmarker K, Hasue M. Classification and pathophysiology of spinal pain syndromes. In: Weinstein JN, Rydevik ABL, Sonntag VKH, eds. Essentials of the Spine. New York: Raven Press, 1995:12–24.)

All patients with a history of malignancy (excluding basal cell skin cancers) require screening with appropriate laboratory and radiographic studies to rule out metastatic disease. Patients with malignant pain characteristically describe constant pain that is present at night and disturbs sleep and which is unrelieved by positional change or rest (3).

Multiple myeloma is the most common "primary" malignancy involving the spine, and often results in diffuse osteoporosis. Serum protein electrophoresis is the initial screening test for multiple myeloma. About 75% of patients with myeloma have an "M-spike"—a monoclonal peak in the gamma region. The diagnosis of myeloma can be confirmed by a urine protein immunoelectrophoresis that shows excess light-chain proteinuria.

A Westergren erythrocyte sedimentation rate (ESR) of greater than 20 mm/h is present in 78 to 94% of patients with back pain who are found to have cancer. Forty to fifty percent of patients have an elevated serum calcium level, whereas 50 to 75% will have an elevation in alkaline phosphatase.

Back pain secondary to vertebral metastasis is the most common symptom in men with disseminated prostatic cancer. Prostate-specific antigen (PSA) levels rise in proportion to the clinical stage and volume of cancer.

Plain lumbar radiographs are probably indicated in all patients over 50 years of age undergoing evaluation for back pain, whether malignancy is suspected. Because plain x-ray films are only 65% sensitive for detecting malignancy, however, computed tomography (CT), magnetic resonance imaging (MRI), or possibly bone scanning are indicated in the presence of a suspicious preliminary workup.

The sensitivity of radionuclide imaging (bone scan) for malignancy is approaching 99%. An important exception to this finding occurs in patients with multiple myeloma (3).

Demographic and Other Factors in Lumbar Spine Pain

In the United States, 6.8% of the adult population has been found to have back pain at any given time. Twelve percent of those with low back pain will have sciatica. The prevalence of low back pain rises after age 25 to a peak in the 55- to 64-year age range, with a falling prevalence after age 65. For sciatica-like pain, the prevalence peaks at the 45- to 54-year age range. Consideration of the specific age of onset shows that 11% of persons are afflicted at less than 20 years of age; 28% at 20 to 29 years, 25% at 30 to 39 years, 20% at 40 to 49 years, 11% at 50 to 59 years, and 5% at more than 60 years of age (4).

The demographic prevalence shows regionally that the northeastern United States has a 38% higher rate of low back pain than the western states. Men and women are afflicted similarly, with white men having the highest prevalence and black men the lowest. Less educated persons have a 50% increased incidence over better-educated persons (4).

Genetics

Disc degeneration has been demonstrated more prominently on MRI in families of patients with it than in controls. A 5.6 times greater risk of lumbar disc herniation was seen in persons aged 18 years or younger whose immediate family showed a history of disc herniation than in control subjects. This strongly suggests that lumbar disc herniation in patients aged 18 years or younger shows familial predisposition and clustering. Familial clustering, however, does not immediately corroborate the presence of a genetic factor and further study is needed to answer this question (5).

Hanrats (6) states that the occurrence of herniated nucleus pulposus in male members of the same family seems to point to a possible hereditary or congenital association, but he has not found a tendency for disc protrusions to occur in the presence of congenital vertebral anomalies.

Childhood Incidence

Herniated intervertebral discs (IVDs) are infrequent in children and adolescents (constituting approximately 1% of patients undergoing surgery) (7).

The incidence of surgically proved lumbar disc prolapse in children varies from 0.8 to 3.2%. Trauma is not a significant causative factor, but a high familial incidence of back pain in affected children is found. Neurologic signs are not as prevalent in children as in adults. About 40% respond to conservative care, and the best surgical results are found in those with brief histories of sciatica (8).

Herniated Disc Presentation in Children

Under age 20, children may present with lumbar disc protrusions with only low back pain and no sciatica. Painless sciatic

scoliosis may be present and the absence of sciatica caused by a central lumbar disc protrusion can be missed. Lumbar disc disease in the first two decades of life may be missed because of the absence of sciatica. Lumbar discectomy in children under age 15 years is safe in all cases and known to be successful in 88% of cases. Initial symptoms are either back pain only or an almost painless kyphoscoliosis in 80%. Only 20% initially complain of sciatic pain (9).

In children with symptoms suggesting nerve root entrapment, the chief concerns are neoplasm, infection, and spondylolisthesis. The possibility of disc herniation often is not suspected because of its infrequency among children. A 10-year-old girl with a herniated disc has been documented with severe scoliosis and vertebral rotation (7).

In children, back pain, radicular pain, and tension signs are common, but neurologic signs are less frequent (10). A study of 1755 children aged 8 to 16 years showed a parental history of treated low back pain, competitive sports activity, and time spent watching television to have significantly increased the risk for low back pain (11).

Smoking

The relationship between cigarette smoking and the development of surgical disc disease was shown with the following speculations: (*a*) The association between cigarette smoking and intervertebral disc disease is more significant in surgical patients than in nonsurgical patients; (*b*) continued cigarette smoking can aggravate discogenic or radicular symptoms in patients with IVD disease; and (*c*) stopping cigarette smoking may have beneficial effects as no significant differences are found between exsmokers versus nonsmokers (12).

SOURCES OF LOW BACK PAIN COMPLAINTS

As with all human disease, the diagnosis and treatment of low back problems begin with the history, followed by the clinical workup, selected imaging modalities for confirmation, and a treatment protocol. Questioning the patient allows concepts to form regarding the involved anatomy. For example, low back pain alone is more common in anular tears and in facet degenerative and subluxation syndromes, whereas sciatica points to disc protrusion or stenosis within the vertebral canal. Serious disc lesions are preceded by numerous and worsening bouts of low back pain. Low back pain that suddenly is transformed into only leg pain probably represents a contained disc that has become a noncontained disc.

Five common causes of sciatica have been suggested (13):

1. Herniated disc
2. Anular tears
3. Myogenic, or muscle-related, disease
4. Spinal stenosis
5. Facet joint arthropathy

Table 10.1 outlines the key differential diagnostic points of these five common causes of sciatica.

Does Anular Tearing Cause Low Back or Leg Pain?

Devanny (14) states that the classic low back syndrome referred to as "muscle spasm" or a "strained back" usually has the disc as the source of pain. If back pain occurs without leg pain, most likely a weakened anulus fibrosus with a disc bulge, not a disc herniation, is causing the pain.

Macnab (15), by placing a catheter under inflamed nerve roots at laminectomy and inflating them later, found that previously irritated nerve roots reproduced the patients' sciatic symptoms. Normal nerve roots only produced feelings of numbness. He deduced that both chemical and mechanical irritation of the nerve root causing pain was analogous to the pain produced by a sunburn of the skin—there might be sunburn, but pain is produced only if the sunburned skin is touched. Similarly, a nerve root might be chemically inflamed but only painful on mechanical compression.

Vanharanta et al. (16) found, in 225 discs injected for discography, that the painful discs had higher degeneration and disruption scores than painless discs. The anular disruption was likely to be the source of exact pain production. The pain was not always similar to the patient's clinical back pain, but exact reproduction or similar pain was found to increase consistently with the amount of disc deterioration. These results suggested that increasing deterioration of lumbar discs was associated with increasing clinical pain. Even small degrees of deterioration can cause a disc to be painful on discography.

Saal (17) reported that anatomic studies have demonstrated the presence of nociceptive nerve endings in the anulus fibrosus of the lumbar discs and that anular tears can therefore cause pain referral of purely discogenic origin into the low back, buttock, sacroiliac joint region, and lower extremities even in the absence of neural compression.

Marshall and Trethewie (18) found that extract of glycoprotein from the human nucleus pulposus releases considerable quantities of histamine, another protein, and amine components that they considered a local irritant of the nerve root, producing edema and pain.

"Internal disc disruption" was described (13) as being the anular fiber tearing and probably also another discogenic cause of sciatic pain. Sciatica results when a tear in the anulus fibrosus leaks nuclear material posteriorly, and the escaped nuclear material irritates the dural sac and nerve sleeves.

Rothman and Simeone (19) state that radiating cracks in the anulus fibrosus develop in the most centrally situated lamellae and extend outward to the periphery. These radiating clefts in the anulus weaken its resistance to nuclear herniation. Herniation is a greater threat to a younger individual between the ages of 30 and 50 having good nuclear turgor than it is to the elderly in whom the nucleus is fibrotic. Falconer et al. (20) state that myelographic defects are seen unchanged after successful conservative treatment of sciatica, not because of mechanical factors but because of clinical nerve root symptoms created by the biochemical irritation of the nerve root degeneration and its resultant irritants on the nerve root. Rothman and Simeone (19) discuss variations of

Table 10.1

Key Diagnostic Tips for Distinguishing Among Five Causes of Sciatica

Herniated nucleus pulposus
- History of specific trauma
- Leg pain greater than back pain
- Neurologic deficit present; nerve tension signs present
- Pain increases with sitting and leaning forward, coughing, sneezing, and straining; pain reproduced with ipsilateral straight leg raising and sciatic stretch tests; contralateral straight leg raising test may also reproduce pain
- Radiologic evidence of nerve root impingement (metrizamide myelography, CT)

Anular tears
- History of significant trauma
- Back pain is usually greater than leg pain; leg pain bilateral or unilateral
- Nerve tension signs present (but no radiologic evidence of impingement)
- Pain increases with sitting and leaning forward, coughing, sneezing, and straining
- Back pain is exacerbated with straight leg raising and sciatic stretch tests (perform straight leg raising test bilaterally)
- Discography is diagnostic (neither CT scan nor myelogram show abnormality)

Myogenic or muscle-related disease
- History of injury to muscle, recurrent pain symptoms related to its use
- Lumbar paravertebral myositis produces back pain; gluteus maximus myositis causes buttock and thigh pain
- Pain is unilateral or bilateral, rather than midline; does not extend past knee
- Soreness or stiffness present on rising in the morning and after resting; is worse when muscles are chilled or when the weather changes (arthritis-like symptoms)
- Pain increases with prolonged muscle use; is most intense after cessation of muscle use (directly afterward and on following day)
- Symptom intensity reflects daily cumulative muscle use
- Local tenderness palpable in the belly of the involved muscle
- Pain reproduced with sustained muscle contraction against resistance, and by passive stretch of the muscle
- Contralateral pain present with sidebending
- No radiologic evidence

Spinal stenosis
- Back and/or leg (bilateral or unilateral) pain develops after patient walks a limited distance; symptoms worsen with continued walking
- Leg weakness or numbness present, with or without sciatica
- Flexion relieves symptoms
- No neurologic deficit present
- Pain not reproduced on straight leg raising; pain reproduced with prolonged spinal extension and relieved afterward when spine flexed
- Radiologic evidence: Hypertrophic changes, disc narrowing, interlaminar space narrowing, facet hypertrophy, degenerative spondylolisthesis (L4–L5)

Facet-joint arthropathy
- History of injury
- Localized tenderness present unilaterally over joint
- Pain occurs immediately on spinal extension
- Pain is exacerbated with ipsilateral sidebending
- Pain blocked by intrajoint injection of local anesthetic or corticosteroid

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the spinal canal in detail. The trefoil canal, which Finneson (21) discussed also, is common at the L4 and L5 level. The trefoil canal has lateral recesses that render it narrower and thereby more vulnerable to compression by extruded disc material. Radiographic findings have shown underdeveloped pedicles which would result in a decreased anteroposterior measurement of the vertebral canal and thus create a stenotic vertebral canal. This would result in more pronounced symptoms of disc protrusion. The combination of a trefoil canal with lateral recesses, underdeveloped pedicles, and articular facet degenerative arthrosis, all of which narrow the vertebral canal and, when coupled with disc protrusion, would result in an exceptionally painful condition. The lumbar nerve roots lie in the superior part of the intervertebral foramen in a relatively protected position, and it is only in disc

narrowing that the superior articular facet of the vertebra below might subluxate in a position to create nerve root pressure. Rothman and Simeone (19) also state that a small nuclear herniation of only 1 to 2 mm in height can cause marked nerve root compression in a patient with a small lumbar spinal canal, and particularly with a narrow lateral recess that makes the patient susceptible to degenerative changes of the intervertebral disc.

How Does Nuclear Degeneration Start and Progress?

Figure 10.2 shows a classification scheme for degenerative disc disease (22). Anular disruption, "leaking-protrusion-anular fissuring," is graphically shown in Figure 10.2. A numerical code indicates how far the contrast material has escaped into the periphery through tears in the anulus: *0* represents a normal disc, and *1* to *3* represent progression of contrast medium into the anulus fibrosus. As the contrast medium advances into the anular periphery, the pain response of the patient is recorded. Table 10.2 shows a code for classifying a pain response according to whether the patient described it as similar or dissimilar to the pain experienced prior to the examination.

Figure 10.3 shows the Videman et al. (23) classification of discographic appearances, from the normal contained disc to the bulging contained disc and leaking, noncontained fragmentation of the disc.

Pathway of Nuclear Entrance Into the Vertebral Canal

The nuclear disc posterolateral prolapse, as shown in Figure 10.4, is commonly recognized, but it must be realized that the nuclear material may find its way into the lateral recesses and vertebral canal through a lateral route to "enter through the side door" into the canal. This is vividly shown in Figure 10.5.

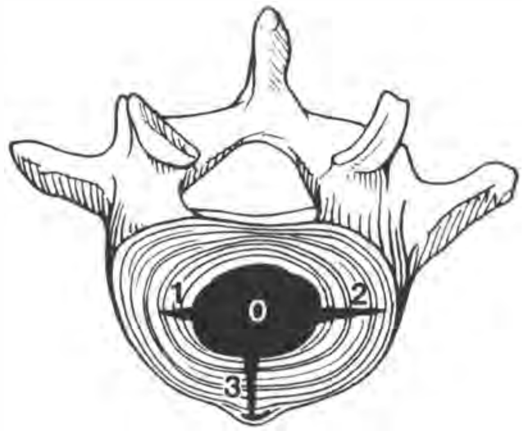


Figure 10.2. The concentric circumferential areas of the anulus used to grade anular disruption as contrast material progressively moves away from the center of the nuclear injection. Areas *0, 1, 2,* and *3* are noted. (Reprinted with permission from Sachs BL, Vanharanta H, Spivey MA, et al. Dallas discogram description: a new classification of CT/discography in low-back disorders. Spine 1987;12(3):288.)

Table 10.2

Dallas Discogram Description

Degeneration (Anulus)	Anular Disruption (Contrast Extension)	Pain
0—No change	0—None	P—Pressure
1—Local (<10%)	1—Into inner anulus	D—Dissimilar
2—Partial (<50%)	2—Into outer anulus	S—Similar
3—Total (>50%)	3—Beyond outer anulus	R—Exact reproduction

Reprinted with permission from Sachs BL, Vanharanta H, Spivey MA, et al. Dallas discogram description: a new classification of CT/discography in low-back disorders. Spine 1987;12(3):287.

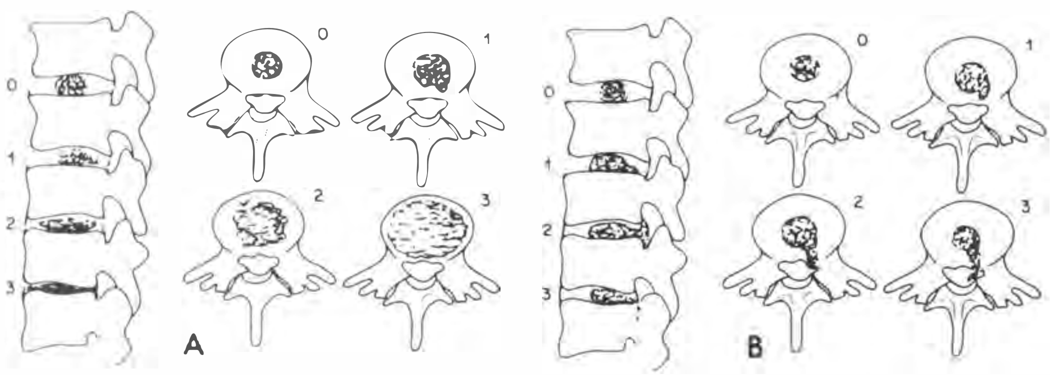


Figure 10.3. **A.** The general appearance of discograms was classified using the following scale: 0, normal; 1, slight; 2, moderate; and 3, severe degeneration. **B.** The anular ruptures were classified using the following scale: 0, none; 1, anular fissure, where dye goes through anulus but is not outside the contour of the normal disc; 2, protrusion, where dye can be seen bulging outside the contour of the normal disc; and 3, leaking, where dye can be seen in the spinal canal coming through the anulus. (Reprinted with permission from Videman T, Malmivaara A, Mooney V. The value of the axial view in assessing discograms: an experimental study with cadavers. Spine 1987;12(3):300.)

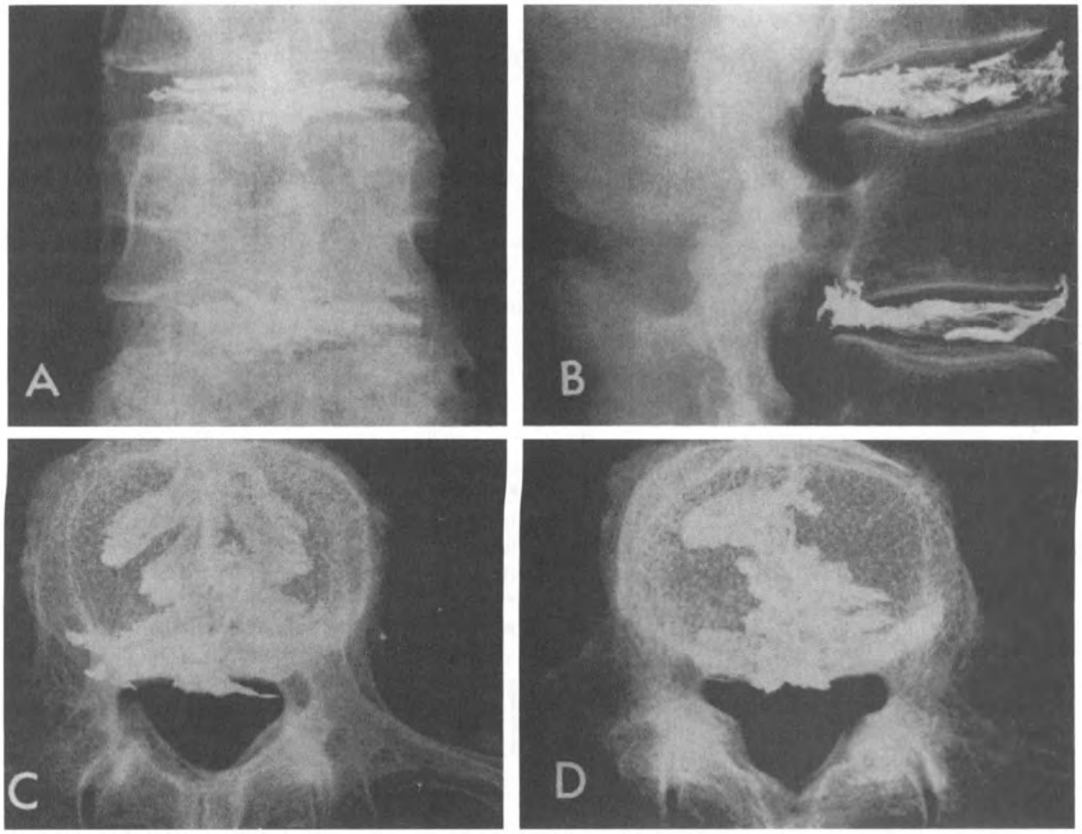


Figure 10.4. **A.** View of two lumbar discograms from levels L3–L4 and L4–L5 using barium sulfate. General degeneration is severe with associated protrusions. **B.** Lateral view shows moderate degeneration. **C.** Axial view at level L3–L4, the nature of degeneration is clear and two separate protrusions can be seen. **D.** Axial view at level L4–L5; an anterior anular fissure can be seen. (Reprinted with permission from Videman T, Malmivaara A, Mooney V. The value of the axial view in assessing discograms: an experimental study with cadavers. Spine 1987;12(3):302.)

The value of discography with CT is shown in Figure 10.6, in which a large free fragment of disc is not seen on a myelographically enhanced CT scan, but it is seen on a discographically enhanced CT scan.

Another interesting study of 441 surgical and autopsy specimens of disc tissue found that the anulus fibrosus was more commonly degenerated than the nuclear material, suggesting that the pathomechanism of disc protrusion is predominantly one of anular protrusion as opposed to nuclear protrusion (24).

DIAGNOSTIC BIOMECHANICS

The most important spinal component is the intervertebral disc. It is the key structure in the movable segment (or, as Schmorl calls it, the "motor segment"), and its lesions (tears, prolapses, and degeneration) affect the rest of the movable segment (25). The axis of sagittal movement of the spine passes through the middle to the posterior portion of the disc, and as the axis pivots around the nucleus pulposus, which acts as a fulcrum, it can shift slightly. In horizontally rotatory movement, the anular fibers in the lumbar region undergo shearing stress leading to tears or rupture, even in younger people, because the vertical axis of rotation is posterior to the vertebral bodies.

Rupture of anular fibers or the dissecting prolapse of the nucleus pulposus through the anulus fibrosus, and fracture and destruction of the basal cartilaginous and bony apophyseal plate may allow prolapse of the nucleus pulposus. This happens especially in young people with high intradiscal pressures sustained on loading in flexion and on high shearing stress in rotation, either into the posterior lateral extradural space (with the middle being protected somewhat by the posterior longitudinal ligament in most instances) or vertically into the bone through gaps, weak places, or fractures of the bony cartilaginous plate (25).

Clinical and experimental observations suggest that the disc may be one of the sources of idiopathic low back pain (26). In patients who develop definite disc herniation, one or more episodes of back pain frequently precedes the herniation. These episodes of pain may be similar to the pain experienced by patients who do not develop disc herniation. Hirsch (27) and Lindblom (28) increased the intradiscal pressure in patients with a history of back pain by injecting saline into the discs. They found that increased intradiscal pressure reproduced the patient's pain. If the disc was injected with a local anesthetic prior to the

increases in intradiscal pressure, pain did not develop. If diatrizoate meglumine and diatrizoate sodium (Hypaque) was injected into a disc and the dye extended into the anulus, severe pain was sometimes produced. If the dye remained in the nucleus, pain did not occur. Direct mechanical stimulation of the anulus and cartilage plate can also produce pain. These findings indicate that irritation or abnormalities of the disc can cause pain, but even if the disc is not the primary source of pain in some syndromes, alterations in the disc can produce symptoms by changing the loads on other structures, including facet joints, spinal ligaments, paraspinal muscles, and nerve roots.

Discal Back Pain and Sciatica

Patients present with back pain and sciatica, with back pain and no sciatica, and with sciatica and no back pain. The most overlooked diagnosis of disc protrusion in clinical practice probably involves the patient with back pain without sciatica. Early nuclear protrusion into the anular fibers often involves the patient with acute back pain and perhaps an antalgic lean to one side. It is well documented that the anulus fibrosus is well innervated by the sinuvertebral nerve, becoming more so from the central portion to the peripheral portion of the disc (19). Radiating cracks in the anulus fibrosus develop in the most centrally situated lamellae and extend outward toward the periphery (29). Turek (30) states that this cracking and fissuring begins as early as the 15th year and may take place silently over many years. The anulus, under the pressure of nuclear protrusion, becomes progressively weaker and thinner. As this pathologic state develops, the pain intensity and degree of antalgic lean of the patient increase.

As the anular fibers progressively thin and the protruding nuclear material makes mild contact with the nerve root, the manifestations of sciatica are first observed. If the anular fibers completely tear and the protruding material bursts forth, the intensity of the sciatica proportionately increases.

Pressure on protruding nuclear material is greater in the young person with a turgid nucleus, which contains up to 80% water, than in the older person in whom the nucleus pulposus has become dehydrated and converted into a hardened mass. Therefore, a patient may have a nuclear bulge creating low back pain resulting from aggravation of the anular fibers, back pain and sciatica may occur as the protruding disc material contacts the nerve root, or only sciatica is present if the disc protrudes through the anulus and contacts only the nerve root,

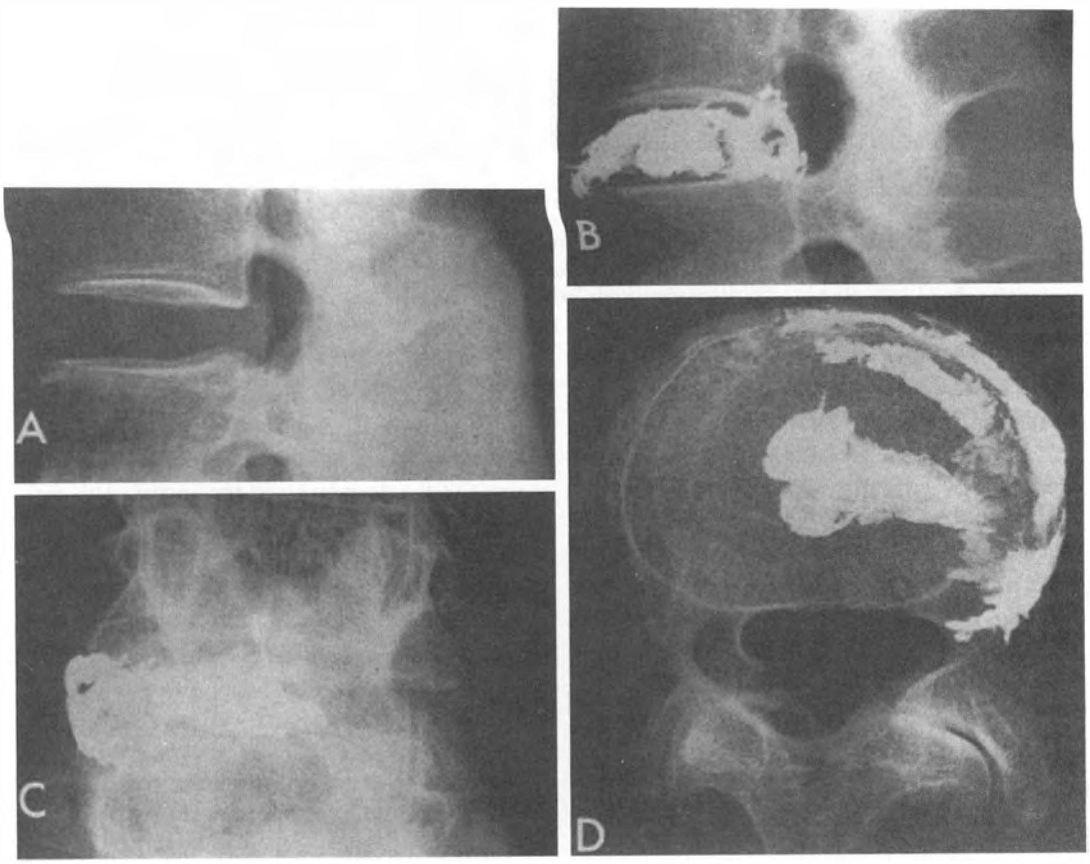


Figure 10.5. **A.** In the plain radiograph, the L3–L4 level looks normal. **B.** The lateral discogram shows severe degeneration. **C.** Posteroanterior view again shows marked asymmetry. **D.** The axial view gives a more exact picture of the nature of the disc lesion degeneration. (Reprinted with permission from Videman T, Malmivaara A, Mooney V. The value of the axial view in assessing discograms: an experimental study with cadavers. 1987;Spine 12(3):300.)

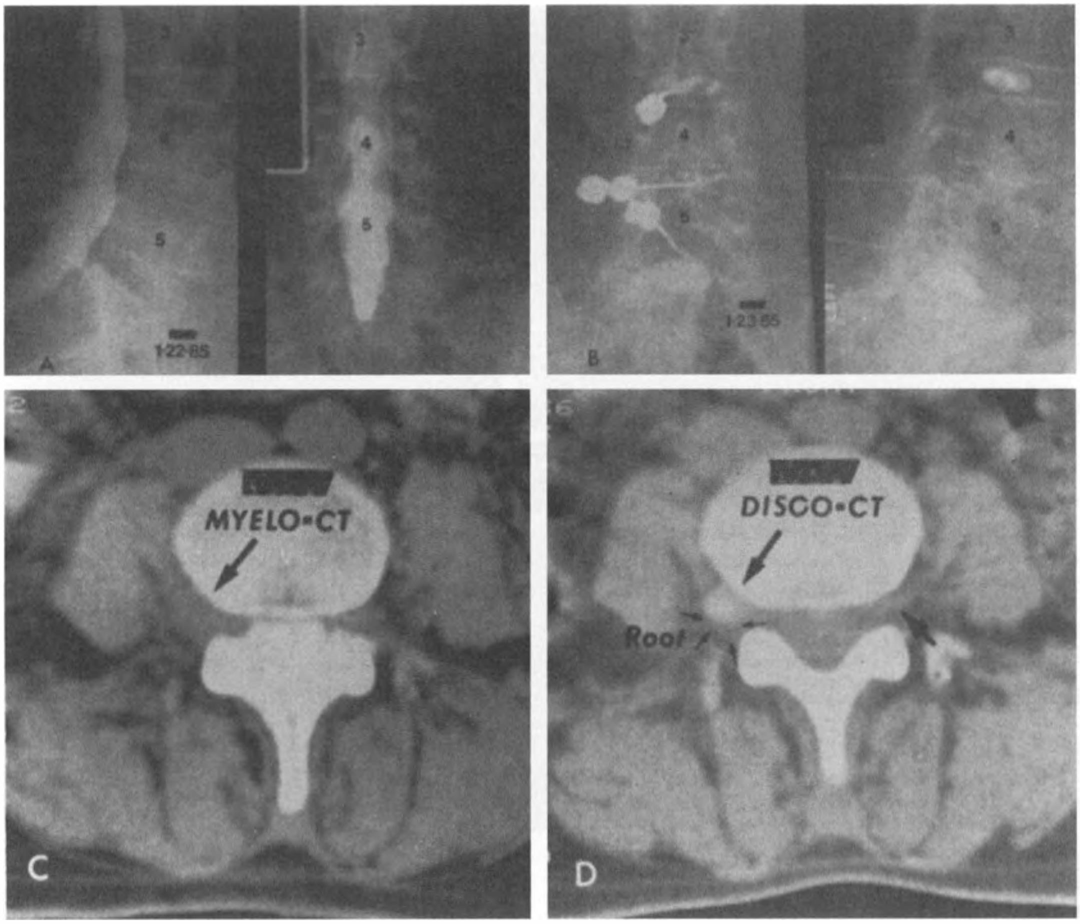


Figure 10.6. **A.** Negative myelogram with slight anular bulging at L4–L5 on lateral view. **B.** Discogram with only minimal degenerative changes. **C.** Myelographically enhanced computed tomography (CT) interpreted as negative, suggests asymmetry in soft tissues lateral to foramen on right (*arrow*). **D.** Positive discogram-CT with large extraforaminal disc fragment (*largest arrow*) and displaced right L4 nerve root (*small arrows*). (Reprinted with permission from Jackson RP, Glah JJ. Foraminal and extraforaminal lumbar disc herniation: diagnosis and treatment. Spine 1987;12(6):581.)

with no other structures innervated by the recurrent meningeal nerve being irritated.

Equally important is the fact that the nucleus that bulges through the anulus fibrosus and comes to lie free under the posterior longitudinal ligament may migrate cephalad and caudally along the posterior vertebral body. Nuclear material that breaks continuity with the remaining nucleus is called a "free fragment" or "a prolapsed disc."

White and Panjabi (31) prepared an update of Charnley's (32) hypothesis on low back pain.[a] Following are their classifications of back pain (31).

ACUTE BACK SPRAIN (TYPE I)

Acute back sprain (type I) characteristically occurs when a laborer attempts to sustain a sudden additional load. Immediate severe pain results that can last for several weeks. The pain, which is primarily in the low back, without sciatica, can be caused by several factors. Charnley suggested the possibility of rupture of some of the deep layers of the anulus. Although this rupture is possible, the inner fibers are not innervated, and relatively less loading and deformation occurs in the deeper fibers than in the periphery. Other possibilities exist, however. One is that peripheral anular fibers can be injured or ruptured along with any of the other posterior ligaments or musculotendinous structures; another is that some of these injuries may involve rupture of muscle fibers or be associated with nondisplaced or minimally displaced vertebral end plate fractures (Fig. 10.7). Whatever the cause, these conditions should respond to a period of rest, followed by a gradual resumption of normal activities.

ORGANIC OR IDIOPATHIC FLUID INGESTION (TYPE II)

An attack of low back pain and muscle spasm can be produced by the sudden passage of fluid into the nucleus pulposus for

[a] Charnley's article (32) is a classic exposition on the topic. His is a clear theoretic presentation of the mechanism, diagnosis, and treatment of the various combinations of back pain and sciatica. It is highly recommended for both the primary care physician and the specialist.

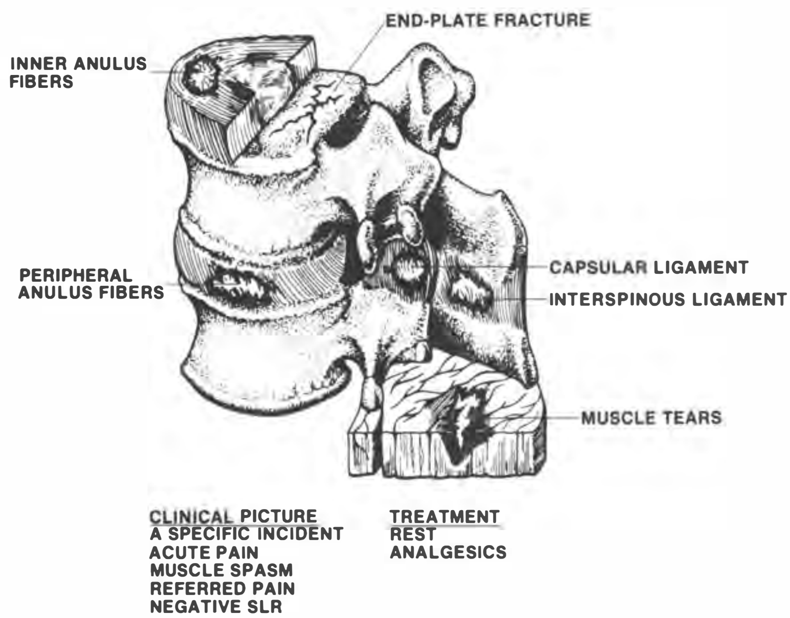


Figure 10.7. A clinical picture of an acute back sprain (type I), which can damage any number of ligamentous structures, the muscle, or even cause a vertebral end-plate fracture. *SLR,* straight leg raising test. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:286.)

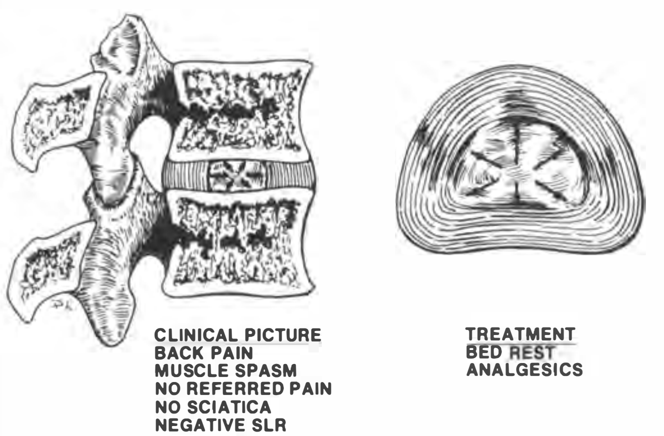


Figure 10.8. Organic or idiopathic fluid ingestion (type II). This mechanism may account for a large portion of back pain for which no distinct diagnosis nor cause has been determined. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:286.)

some unknown reason (32, 33) (Fig. 10.8). Charnley suggested that this passage of fluid irritated the peripheral anular fibers, causing the characteristic pain. Little has been found in the intervening 20 years to discredit this hypothesis. Naylor (33) suggests that increased fluid uptake in the nucleus is a precipitating factor in the biochemical chain of events that can lead to disc disease.[b] Indirect evidence, however, suggests that increases in fluid in the disc structure does not cause spine pain. This evidence is based on the observation that astronauts returning from outer space have heightened disc space but no back pain according to Kazarian (34). On the other hand, evidence, although inconsistent, suggests that fluid injection into the normal disc causes low back pain (35). This discrepancy may be partially explained by the differences in the rate of change in fluid pressure. The hypothesis of fluid ingestion is consistent with the clinical data because it is compatible with the characteristic clinical course of exacerbations and remissions, with or without progression to other clinical syndromes. In other words, movement of fluid in and out of the disc can explain the onset and resolution of the clinical symptoms. This may be the explanation for spontaneous idiopathic organic spine pain (cervical, thoracic, or lumbar) unrelated to trauma, which accounts for a significant number of the many cases of spine pain.

POSTEROLATERAL ANULUS DISRUPTION (TYPE III)

If failure or disruption of some of the anular fibers occurs, posterolateral irritation in this region can cause back pain with referral into the sacroiliac region, the buttock, or the back of the thigh (Fig. 10.9). This referred pain is caused by stimulation of the sensory innervation by mechanical, chemical, or inflammatory irritants. Thus, "referred sciatica," as Charnley called it, is distinguished from true sciatica by a negative straight leg raising (SLR) test and a lack of neuromuscular deficit. As suggested, this referred pain may be explained by the "gate" control theory. This referred sciatica may resolve itself through

[b] Naylor's article (33) provides a superb, comprehensive review of this hypothesis.

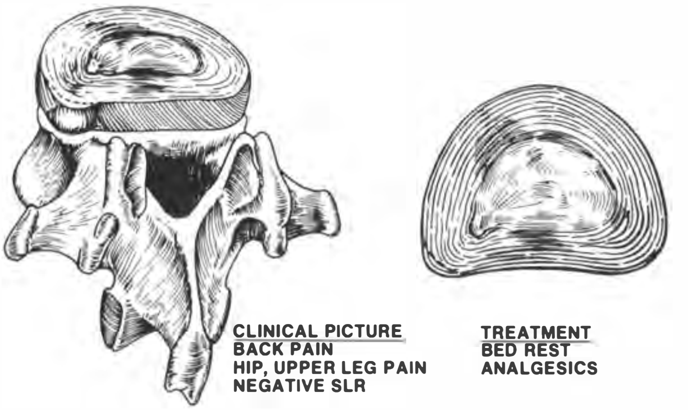


Figure 10.9. Posterolateral anulus disruption (type III). The *dotted line* represents the original normal contour of the disc. Hip and thigh pain are referred pain rather than true sciatica. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:287.)

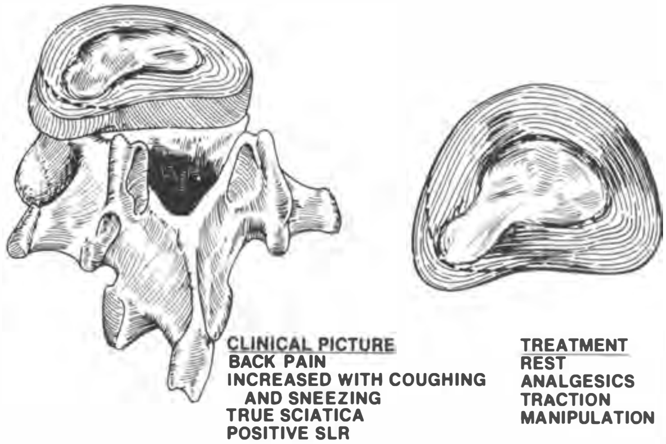


Figure 10.10. Bulging disc (type IV). In the patient with a bulging disc, the anulus is bulging to such an extent that nerve root irritation has caused sciatica. The *dotted line* shows the normal position of the anulus rim. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:287.)

reabsorption or neutralization of the irritants and/or phagocytosis and painless healing of the disrupted anular fibers.

BULGING DISC (TYPE IV)

Another proposed mechanism of low back pain and sciatica involves protrusion of the nucleus pulposus protrusion, which remains covered with some anular fibers and, possibly, the posterior longitudinal ligament (Fig. 10.10). "True acute sciatica" may be present with mechanical and, possibly, chemical or inflammatory irritation of the nerve roots. Pain may also be found in the back, buttock, thigh, lower leg, and even the foot, and it may be increased with coughing and sneezing; the SLR test is positive. In this situation, radiographs usually do not indicate narrowing. Traction or spinal manipulation may alter the mechanics and possibly be therapeutic. With rest, the irritation may subside and remain stable, or it may return spontaneously after mobilization.

A good example of type III and IV anular disruption and disc bulging, seen at our clinic, is presented in Figures 10.11–10.13.

Case 1

This 38-year-old woman was seen at the referral of another chiropractor for the chief complaint of low back, left buttock, and left upper thigh pain. The pain had started approximately 1 year prior to our first seeing the patient, following bending, lifting, and twisting at the waist while picking up a 30-pound dog. She felt a sharp pain at the time and could not stand upright. She saw a chiropractor the following day, who treated her and gave some relief. She continued to feel a nagging ache in her low back despite a home exercise program, and 9 months after the initial injury again sought chiropractic relief. At that time, a lesion on the left leg was diagnosed as malignant melanoma, and it was surgically removed.

In this case, we did not feel that surgery was necessary. Our treatment program included the following: (*a*) flexion-distraction manipulation applied at the three lower disc levels, with range of

Figure 10.11. Computed tomography scan at the L3–L4 level shows a 4.2-mm disc protrusion.

Figure 10.12. L4–L5 level shows a 6.1-mm central disc protrusion, and the vertebral canal, by computed tomography, measures 10.7 mm sagittal diameter.

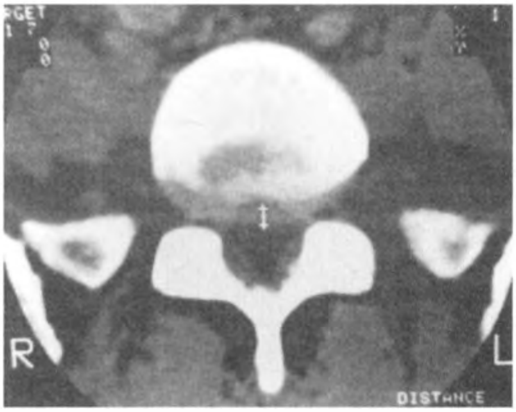


Figure 10.13. Computed tomography (CT) scan at the L5–S1 level shows a 3.8-mm L5–S1 disc bulge, and a sagittal vertebral canal CT measurement of 10.7 mm.

motion of the articular facets at each of these areas applied as the patient showed 50% improvement of the low back and upper left thigh pain; (*b*) low back wellness school to teach this patient the hazards of sitting and how to bend and lift in daily living with minimal stress to the lumbar spine; (*c*) a strong exercise program of seven Cox exercises to correct the weakness of the abdominal, low back, and gluteal muscles; (*d*) adjustment, in side posture, of an anterior innominate subluxation that accompanied the long left leg, followed by the wearing of a trochanter belt to support this left sacroiliac joint during healing; (*e*) appropriate instructions to apply hot and cold alternating packs to the low back, left buttock, and upper thigh at home; (*f*) Nautilus extension exercises, started on the third day of treatment.

During treatment, this patient's pain actually settled into the sacrum and sacrococcygeal articulation. Rectal adjustment was done of the coccyx to check its alignment with the sacrum.

This patient was treated in our clinic for 30 days and returned home with a letter of referral to her referring chiropractor. Her remaining complaint on dismissal was left L5–S1 pain.

SEQUESTERED FRAGMENT (WANDERING DISC MATERIAL) (TYPE V)

A sequestered nucleus pulposus and/or anulus fibrosus (Fig. 10.14) associated with the normal degenerative processes of the disc and other presently unknown pathologic changes may develop with time. This sequestrum can move about in a random fashion in response to the directions and magnitude of forces produced at the motion segment by an individual's activity. This movement may cause the sequestrum to irritate the anular fibers (by physical presence or chemical breakdown products) and to produce low back pain with or without sciatica. It can also produce a bulge in an area in which it can cause true sciatica. The sequestration can move about, so that it either is asymptomatic or it causes some combination of spine pain, referred pain, and true radiculopathy. Because of the movement of the sequestered fragment in response to forces at the motion segment, it may be possible, through axial traction or spinal manipulation of the motion segment, to move the sequestrum temporarily or permanently from a location in which it stimulates a nerve to one in which it causes no irritation. Subsequent motion of the disc fragment into areas of pain insensitivity or subsequent scarring may not cause recurrence. On the other hand, if no scarring exists, the random movement of the sequestered portion of the disc can include positions of subsequent nerve root irritation.

DISPLACED SEQUESTERED FRAGMENT (ANCHORED) (TYPE VI)

Another clinical and mechanical cause of low back pain and sciatica is displacement of a sequestrum of the anulus or nucleus into the spinal canal or intervertebral foramen (Fig. 10.15). The fragment is to some degree fixed in position. Nerve root

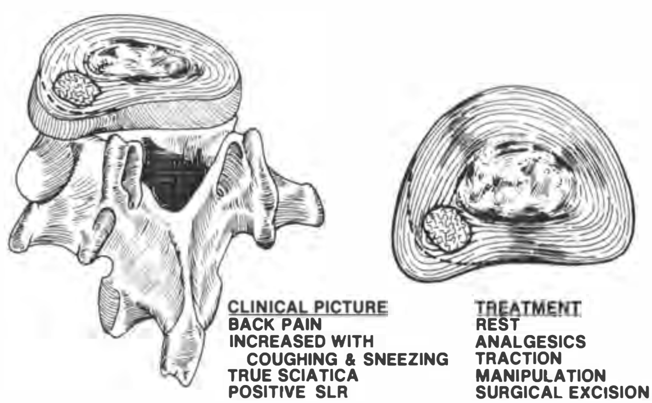


Figure 10.14. Sequestered fragment (the wandering disc) (type V). Surgical treatment results are better in the type V patient than in the types I to IV patient, but they are probably not as good in the type V patient as they are in the type VI and type VII patient. The wandering disc is a possible explanation for the clinical picture of exacerbations and remissions that is so frequently encountered. It may also be a partial explanation of why some patients show a good response to traction or manipulation. (Reprinted with permission of White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:288.)

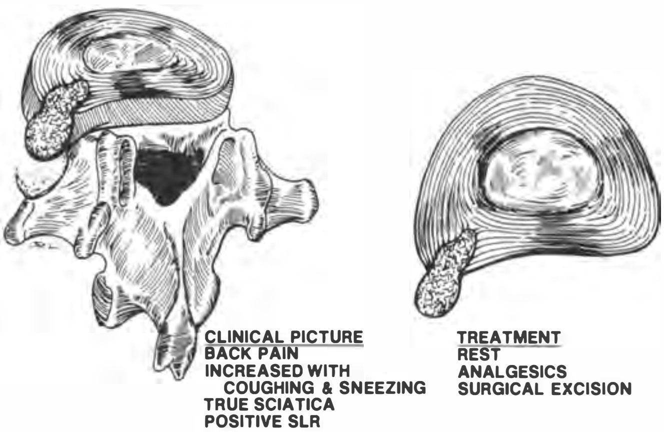


Figure 10.15. With type VI, there is sequestration and displacement, but there is some anchoring of the ligament so that the disc cannot move about. This is likely to be helped by traction or manipulation. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:289.)

irritation results from inflammation caused by mechanical pressure, chemical irritation, an autoimmune response, or some combination of the three. True sciatica exists with the positive SLR sign. In association with a displaced portion of the intervertebral disc (sequestration), narrowing of the interspace may occur at the involved level. Axial traction, manipulation, and random movement are unlikely to help. Chymopapain injected into the disc space may never reach or affect the sequestrum, especially if scarring or blockage has occurred in the hole in the disc structure. When this situation subsides spontaneously, hypothetically, it is the result of phagocytosis or some physiologic adjustment of the neural structures to the irritation. Patients with a displaced sequestered fragment show the best results when treated with surgery, as suggested by Charnley and subsequently confirmed by Sprangfort (32, 36).[c]

Examples of Charnley's type 5 and 6 disc lesions from our clinic are presented next.

Case 2

A 30-year-old woman developed low back pain following delivery approximately 7 to 8 weeks prior to seeing us. The start of her low back pain felt like a pinching and then eventually continued down the left lower extremity. The history revealed lower back pain 3 years previously, which was relieved with exercise.

Figure 10.16 reveals the marked sciatic scoliosis of this patient. Note the flexed left knee to relieve stretch on the sciatic nerve. This patient's low back and leg pain were aggravated by the Déjérine triad. Straight leg raising was positive sitting and recumbent at 10°, creating low back and leg pain. Marked reduction of her ranges of motion was noted. The deep reflexes and sensory examination were within normal limits. Circulation of the lower extremity was normal. The hamstring reflexes were +2 bilaterally. No atrophy was noted.

Figures 10.17–10.19 are neutral, right, and left lateral flexion studies of this patient. Note the strong right lateral flexion subluxation of L4 on L5 and the inability of this patient to laterally flex to the left. Figure 10.20 shows the posteroanterior (PA) film taken at the time of myelography. Note the large filling defect at the L4–L5 segment, which is also seen on Figure 10.21, in the lateral projection. Figure 10.22 is the oblique view, revealing the filling defect caused by the intervertebral disc protrusion compressing the dye-filled subarachnoid space. Figure 10.23 is the CT scan, revealing an extremely large left central IVD disc protrusion.

The IVD protrusion was surgically removed, and the patient had 100% relief of symptoms. Treatment was attempted with flexion distraction, but because of the extreme size of this disc lesion the patient could not tolerate any attempt at therapy or spinal manipulation. This is a good case of surgical necessity.

DEGENERATIVE DISC (TYPE VII)

Disc degeneration (Fig. 10.24) involves a disruption of the normal anular fibers of the disc to such an extent that the disc is no longer able to serve an adequate mechanical function. This disruption can be associated with degenerative arthritic processes of the vertebral bodies or the intervertebral joints. Pain may be chronic, intermittent, or absent.

Figure 10.16. Severe right sciatic scoliosis in a patient with left fifth lumbar nerve root paresthesia. Note that the left knee is held flexed to prevent stretch on the sciatic nerve (Neri's bow sign).

Figure 10.17. Right lateral list of the lumbar spine is seen in the patient in Figure 10.16. Note how the pelvis is posterior as evidenced by the loss of height of the pelvic ring and how high the symphysis pubis lies over the sacrum (*arrow*).

[c] Spangfort's article (36) is an excellent discussion of the significance of various physical findings in the evaluation and interpretation of low back pain and sciatica.

Figure 10.18. On right lateral flexion, minimal motion occurs, and the spinous processes (*arrows*) fail to rotate to the right concavity and instead rotate to the left convexity of the curve.

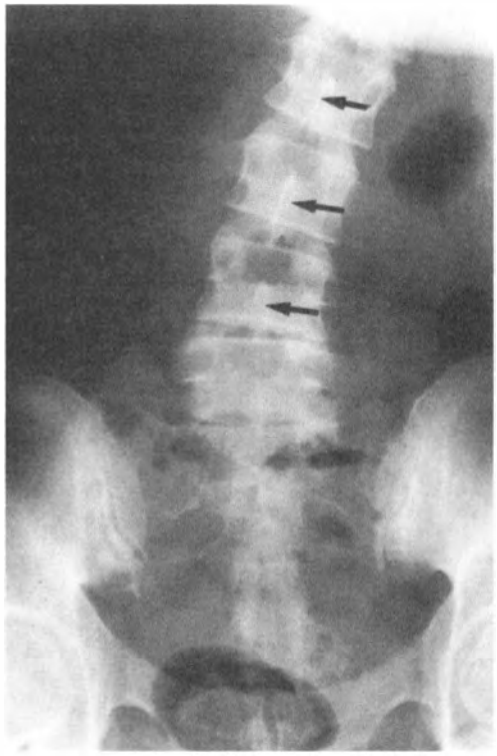


Figure 10.19. On attempted left lateral flexion, the spinous processes (*arrows*) are in the midline and actually represent the only motion seen in the lumbar segments in attempted left lateral flexion. No movement occurs into the left painful side of this lumbar spine and left lower extremity.

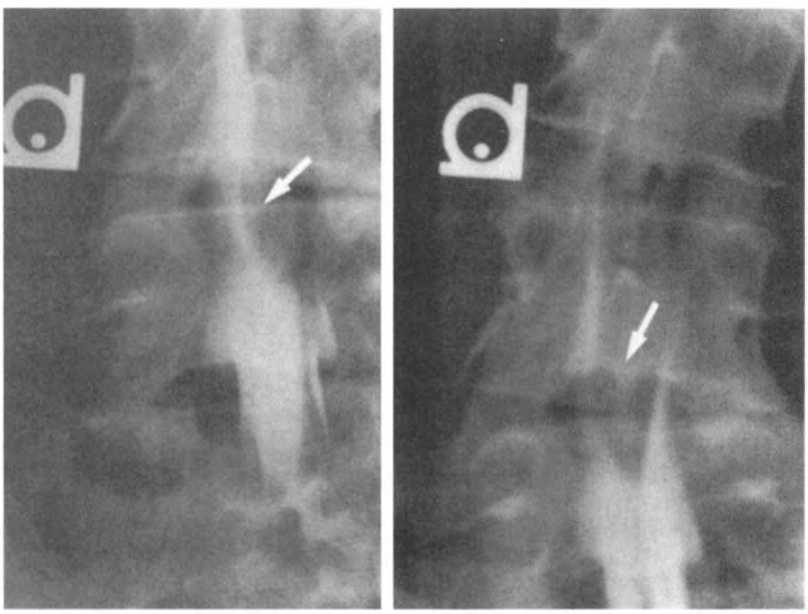


Figure 10.20. Myelography shows a large L4–L5 filling defect in the posteroanterior view (*arrow*).

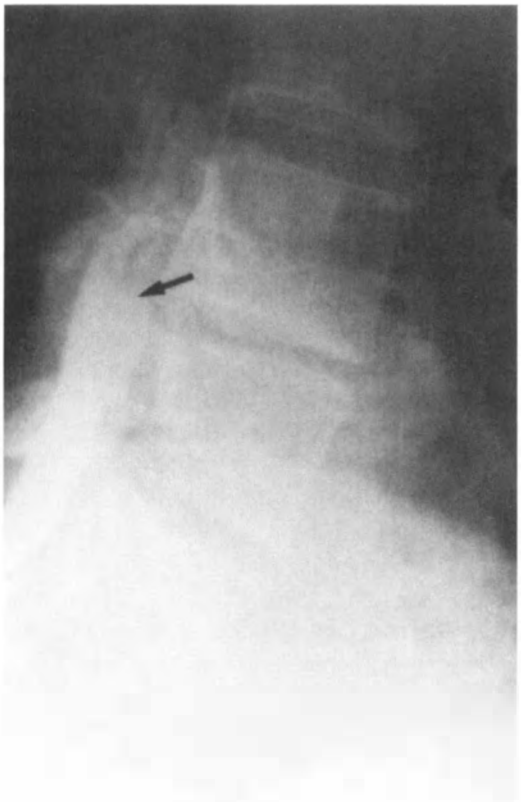


Figure 10.21. Lateral projection of Figure 10.20 shows the flexion of L4 on L5 and the bulging of the L4–L5 disc into the dye-filled subarachnoid space (*arrow*).

A good example of Charnley's type VII degenerative disc is shown in a case from our clinic:

Case 3

The patient in this case was a 52-year-old woman who had back surgery 1 year prior to her first visit to our clinic. She had low back and leg pain prior to the surgery; and 1 year following surgery, she was in greater low back pain and the pain was radiating into her right lower extremity fifth lumbar dermatome.

Figure 10.25 shows a radiograph taken prior to her back surgery. Pseudosacralization is seen at L5 on the right with marked loss of the L4–L5 disc space and discogenic spondyloarthrotic changes. The left L4 inferior articular facet is hyperplastic, creating a pseudoarticulation with the laminae of L5. L3 is in right lateral flexion subluxation, and tropism at this level is noted, the facet on the right being sagittal and the left coronal. Also note the arthrotic changes of the pseudosacralization between L5 and the sacrum and ilium on the right. Figure 10.26 shows a repeat x-ray study of her spine 1 year following her back surgery. Note the further degenerative change in the L4–L5 disc and also at the L3–L4 disc, where a marked loss of disc space and bone periosteal reaction is seen on contact with the vertebral bodies. This represents the concept of moving the ranges of motion up one segment cephalad following disc degeneration.

As we know in Bertolotti's syndrome, because of the transitional segment at L5 on the sacrum, the motion takes place at the L4–L5 level. As the L4–L5 disc degenerates and is surgically operated, as in this case, the motion shifts to the L3 level. As can be seen, this disc soon deteriorates when required to take up 95% of the flexion and extension motion of the lumbar spine. Normally, L5–S1 makes up 75% of the flexion and extension motion, and L4–L5 20%, with only 5% of the flexion and extension occurring in the upper lumbar segments. As each succeeding lumbar disc is required to assume more mobility as the one below degenerates, that disc is less capable of maintaining that motion and it undergoes degenerative change.

Figure 10.27 is a lateral view of the lumbar spine prior to surgery. Here is seen the L4–L5 disc degenerated and L3 posteriorly subluxated on L4, but still maintaining a good disc space.

Figure 10.28 taken 1 year following surgery shows that the

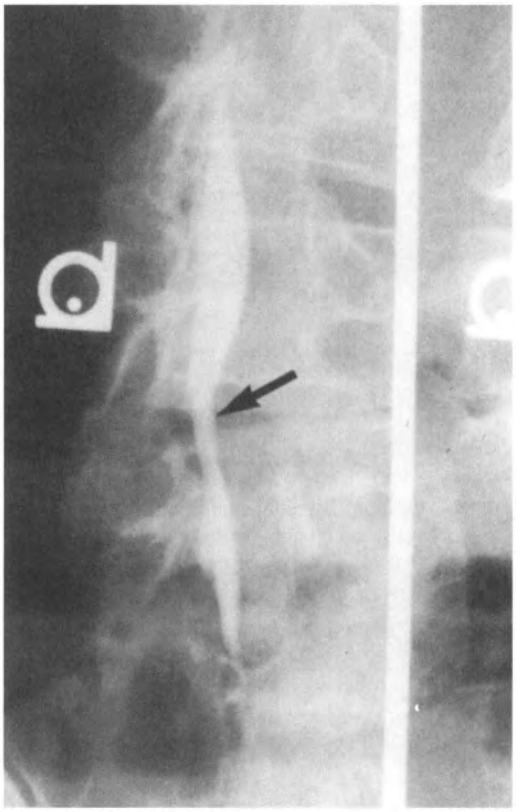


Figure 10.22. The oblique view shows the filling defect at L4–L5 due to the massive L4–L5 disc prolapse (*arrow*).

Figure 10.23. Computed tomography scan shows a large left central disc prolapse at the L4–L5 level (*arrow*).

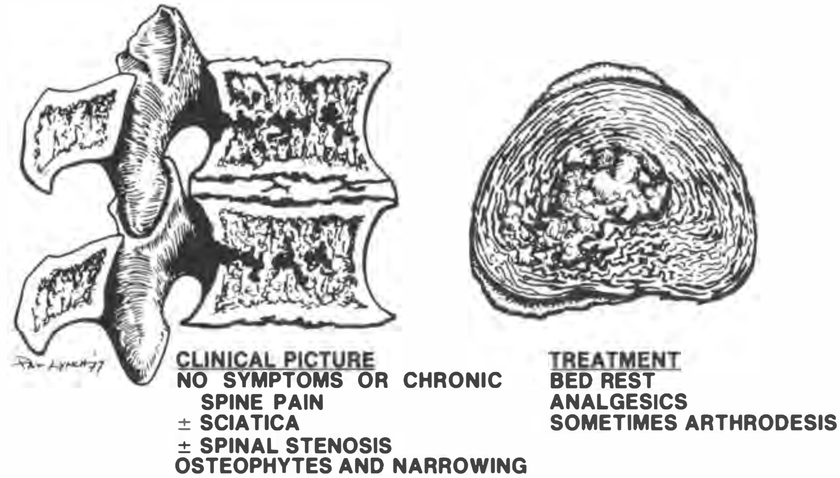


Figure 10.24. A degenerated disc (type VII) either may be the end process of the mechanical and biologic effects of normal functioning or may be associated with considerable pain and disability. Arthritis may also be in the intervertebral joints. It is important to emphasize that these various stages are a continuum. A given disc can move, decelerate, stop, or, in some instances, even reverse. (Reprinted with permission from White AA, Panjabi MM. Clinical Biomechanics of the Spine. Philadelphia: JB Lippincott, 1978:290.)

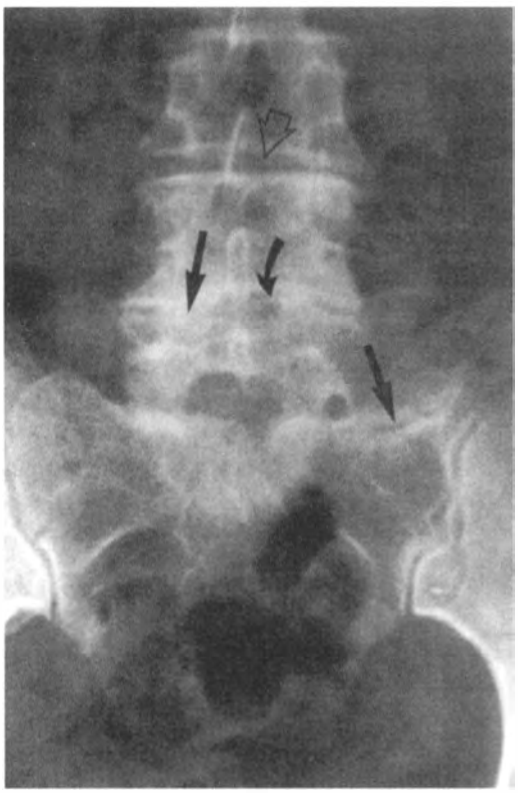


Figure 10.25. Pseudosacralization of the right L5 transverse process (*straight arrow*), with marked degenerative changes at the L4–L5 disc level (*curved arrow*) and to a lesser degree at the L3–L4 level (*open arrow*). L3 is in right lateral flexion on L4. The left L4 inferior facet is hyperplastic and creates a pseudoarticulation with the lamina of L5 (*long arrow*). Also note the degenerative change at the pseudoarticulation of the overdeveloped right L5 transverse process with the sacrum.

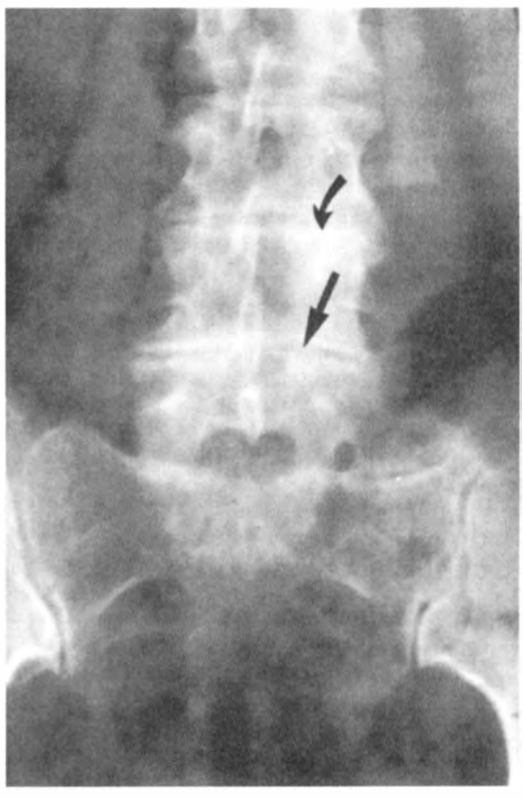


Figure 10.26. This is a posteroanterior view of the patient seen in Figure 10.25, 1 year after surgery for removal of an L4–L5 disc protrusion, and seen is the further disc degeneration at the L4–L5 (*straight arrow*) and L3–L4 (*curved arrow*) levels. The combination of a transitional segment and disc degenerative or protrusion changes above it is called Bertolotti's syndrome. The transitional segment has a rudimentary disc and places the movement that normally occurred at that level on the disc above. Thus, the increased stress causes the disc to become unstable and undergo degeneration. This is a good example of type VII disc degeneration by Charnley's classification.

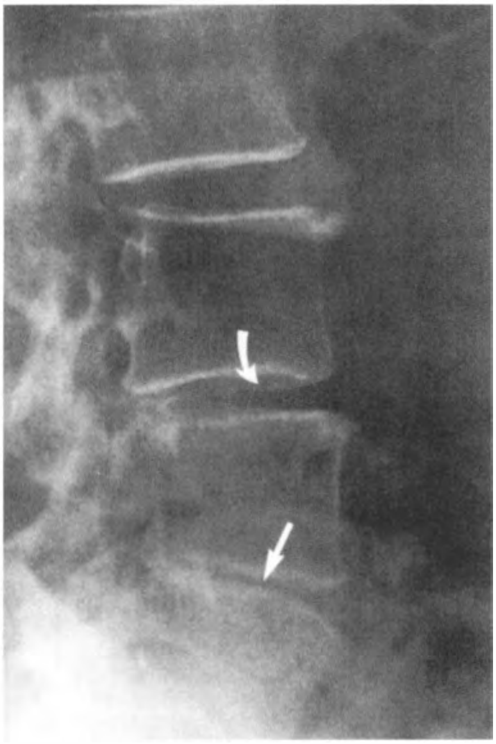


Figure 10.27. Lateral view of the patient shown in Figure 10.25, prior to surgery, shows advanced loss of joint space at the L4–L5 disc level (*straight arrow*), with vertebral plate sclerosis and anterolateral hypertrophic traction spurring. The L3–L4 disc (*curved arrow*) also shows, to a lesser extent, the same findings as L4–L5.

L4–L5 disc has increased its degenerative change; however, the L3–L4 disc is markedly degenerated, with marked anterolateral lipping and spurring and subchondrosclerosis of the opposing vertebral body plates.

This case is a good example of Bertolotti's syndrome at L5 with an L4–L5 disc protrusion. Following surgery at L4–L5, the movement shifted to the L3 segment, which then became the level of maximal mobility and also the level of maximal degenerative change—a good example of a "domino" effect of disc degeneration moving from caudal to cephalic disc levels.

ORGANIC IDIOPATHIC SPINE PAIN

Organic idiopathic spine pain is the type of pain present in patients who are diagnosed clinically as having organic spine pain without sciatica for which no known cause is evident. Pain can emanate from the disc, or it can result from increased fluid uptake by the disc (type II), any combination of the previously described causative factors, or some mechanism yet to be discovered.

DISCOGRAPHY: CONTRIBUTIONS TO DISC DISEASE DIAGNOSIS

Discography will be discussed because it clearly defines discal changes that result in altered spinal biomechanics and eventual pain. Controversy over this imaging modality exists, but few physicians treating low back pain and sciatica can resist the excitement of seeing the relationship of the disc nucleus pulposus, enhanced by contrast agent, to the anulus fibrosus. Nothing can match the definition of change from normal to degenerative as vividly as discographically enhanced computed tomography, or secondarily, discography plain film study.

Discography Is More Sensitive to Early Disc Disruption Than Is MRI

Magnetic resonance imaging can miss internal disc disruption that can be seen on discography (37, 38). Normal disc signal intensity on MRI does not rule out degeneration. Although a decrease in T2-weighted signal intensity on sagittal MRI is virtually always associated with anular degeneration, normal signal intensity does not exclude significant degeneration (39, 40). In patients with unrelenting low back pain of apparent discogenic origin, lumbar discography should be considered to investigate occult morphologic abnormalities of the intervertebral disc (37). MRI, which demonstrates disc degeneration, will never present normal morphology on discography (38).

In most cases, however, MRI is equal to discography in the diagnosis of degenerative or extruding disc disease (40). Figure 10.29 demonstrates the MRI findings of abnormal L4–L5 and L5–S1 disc degeneration with the discographic findings. Figure 10.30 similarly shows the contrasting findings of MRI and

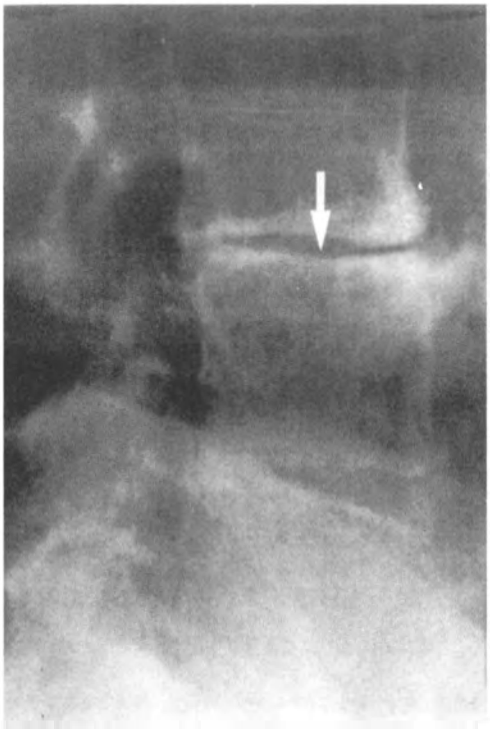


Figure 10.28. The patient shown in Figures 10.26 and Figure 10.27 1 year after surgery shows extreme L3–L4 discal degeneration (*straight arrow*), as evidenced by loss of joint space and subchondral sclerosis and anterolateral hypertrophic spurring. The vacuum phenomenon is seen in the anterior L3–L4 disc area. The L4–L5 disc shows the same degenerative changes as prior to surgery.

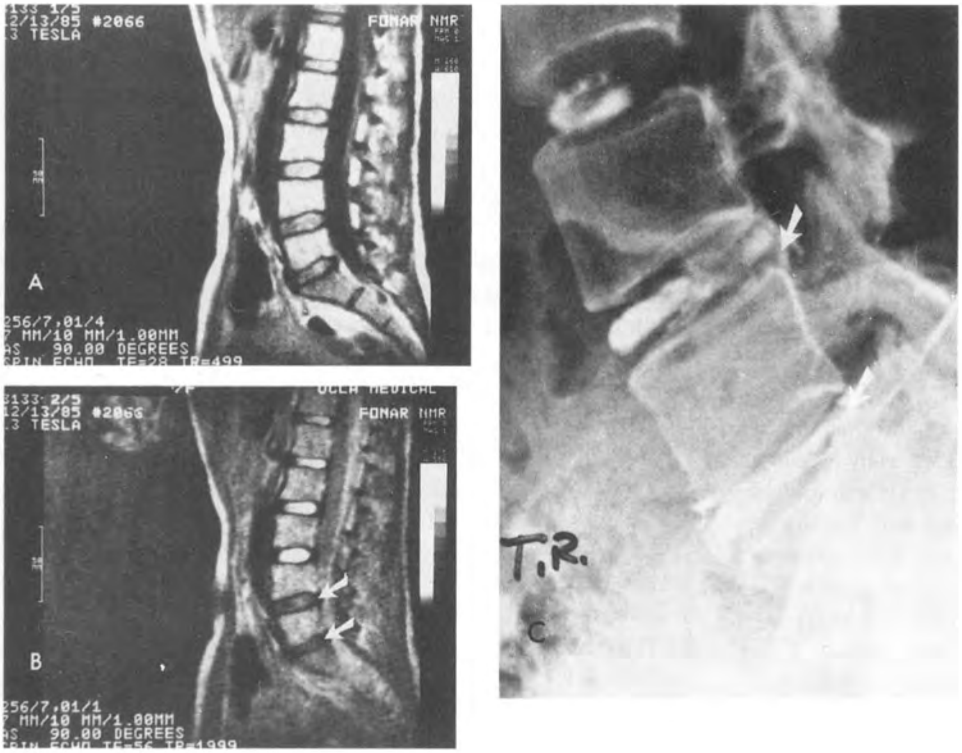


Figure 10.29. **A.** T-1 weighted sagittal magnetic resonance image (MRI) (SE 500/28) demonstrates no obvious abnormalities of the disc intensity. **B.** Sagittal MR image T2-weighted (SE 2000/56) demonstrates decreased signal intensity and focal disc bulges at the levels of L4–L5 and L5–S1 (*arrows*). **C.** Lateral radiographic discogram demonstrates degenerated herniated discs at the level of L4–L5 and degeneration at L5–S1 (*arrows*). L3–L4 is normal in appearance. (Reprinted with permission from Schneiderman G, Flannigan B, Kingston S, et al. Magnetic resonance imaging in the diagnosis of disc degeneration: correlation with discography. Spine 1987;12(3):276–282.)

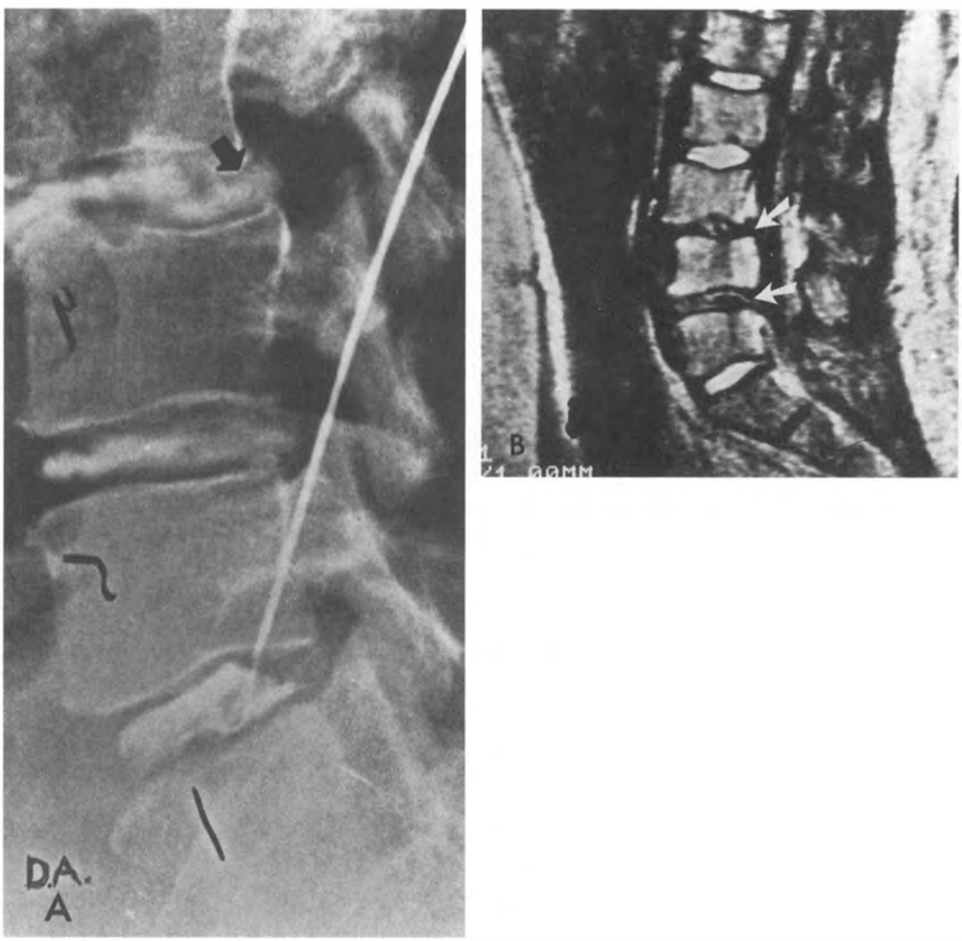


Figure 10.30. **A.** Lateral discogram examination demonstrates degenerated herniated discs at the level of L3–L4 and L4–L5. A normal disc is identified at L5–S1. Grade 1 spondylolisthesis is seen at L3 on L4 (*arrow*). **B.** Sagittal MR (SE 2000/56) demonstrates grade 1 spondylolisthesis L3 on L4 and marked loss of signal intensity at the levels of L3–L4 and L4–L5 (*arrows*). Note normal intensity at L2–L3 disc and L5–S1 disc. (Reprinted with permission from Schneiderman G, Flannigan B, Kingston S, et al. Magnetic resonance imaging in the diagnosis of disc degeneration: correlation with discography. Spine 1987;12(3):276–282.)

discography of L3–L4 and L4–L5 discs with degenerative changes and grade 1 true spondylolisthesis of L3 on L4.

Previously in this chapter the clinical value of discography was explained and shown in Figures 10.4–10.6, wherein only the axial view reveals the laterally escaping dye from the nucleus. Also, other modalities such as MRI, CT, and myelography might be falsely negative as the vertebral canal may not be invaded by the disc protrusion, but the tearing and leaking of nuclear material as a pain-producing entity can only be appreciated on the axial CT discogram.

Discography Twice as Accurate as MRI

Inferior and superior rim lesions of the anterior anulus frequently are (27 and 10%, respectively) found by histologic investigation. These tears are seen when greater amplitudes of rotation are observed. Discography does not demonstrate all peripheral anular lesions, but parasagittal MRI scanning was found to produce twice as many false-negative images as discography. A normal MRI signal may occur with a considerable reduction in the amount of nuclear material (41).

Extraforaminal Disc Fragmentation Diagnosis

Persistent radiculopathy, undiagnosed by conventional CT, MRI, or myelography, could be an extraforaminal disc herniation that has escaped detection. Discography is an imaging modality of excellent selectivity to uncover this difficult entity (42). CT scanning following lumbar discography (discographically enhanced CT scan) is an excellent modality for finding previously undiagnosed or negative evaluations (43).

Discography Reproduces Low Back Pain

Discography is rarely, if ever, painful in asymptomatic individuals, even in those with degenerative discs, but it is frequently painful in patients with low back pain. Internal disc disruption has been postulated as an important cause of low back pain. The key feature of discography is the patient's response to disc stimulation, not the appearance of the disc. In this regard, discography determines whether a degenerative disc has become symptomatic. *For discography to be positive, disc stimulation must reproduce the patient's pain, irrespective of the morphology of the disc.* With respect to clinical features, no conventional clinical test, or combination of tests, could differentiate reliably between patients with and without discogenic pain. For a proportion of patients, an alternative exists to proclaiming "there is nothing wrong with your back" (44).

Discography identifies the level of disc pain in patients being considered for spinal fusion (45). During discography the outer anulus appears to be the origin of pain reproduction (46). Painful discs have higher degeneration and disruption scores compared with painless discs (47) Pain provocation accompanying discography was not elicited with MRI, thus reducing MRI's ability to define the pathologic disc (48).

Discogenic-induced Lower Extremity Radiculopathy

Radiating Lower Extremity Pain from Within the Disc

Intradiscal injection of a local anesthetic, 1% lidocaine, after producing severe and persistent low back pain with unilateral or bilateral radiation to the lower extremities by injecting contrast agent into one disc, produced a 75 to 100% reduction of the low back pain in 13 patients, and a 75 to 100% reduction of radiating pain was experienced by 16 patients within 60 seconds. The conclusion was that the pain of some patients with low back pain and unilateral and bilateral radiation to the lower extremities arises from *within the disc.* In these cases the pain radiating to the lower limb seemed to be a referred type pain and was unrelated to direct nerve root compression or irritation by a disc fragment in the epidural space (49).

Figures 10.31 and 10.32 show two patients with discography reproducing the low back and lower extremity pain they had experienced. Figure 10.31 demonstrates an L5-S1 small posterior central disc hernia on CT scan without nerve root compression; however, discography reveals contrast medium leaking into the posterior disc space and vertebral canal, producing left lower extremity thigh and buttock pain. Figure 10.32 shows a small anterior tear of the inner anulus fibrosus that caused the left buttock and thigh pain complaint of a 23-year-old man. The benefit of discography is its ability to reproduce the patient's symptoms and signs, even when other imaging modalities show subtle or no signs of internal disc derangement or disc leaking.

How Is Radiculopathy Caused by Internal Disc Derangement?

Lindblom (50) introduced discography in 1948 and its validity is controversial today. Proponents state that the pain-sensitive structures responsible for the radiating pain to the lower extremity are located somewhere inside the disc, probably in the external part of the anulus fibrosus and in the longitudinal ligaments (49). Rat studies have documented sensory nerve fibers and endings in the disc and it is reasonable to infer their existence in the human. Radiation into the lower extremities because of anular disruption and disc rupture has been suggested (51–53).

Leaking of nuclear material through an anular tear establishes a chemical inflammatory reaction within the pain-sensitive anular peripheral fibers that produce the radiation into the lower extremities. This is poorly understood so far as the nervous system pathways involved in producing radiculopathy are concerned (54–59).

Pain produced following injection of contrast during discography has the following characteristics that support the concept that it is the sudden intradiscal pressure that stretches the nerve endings that causes the pain (49):

1. The postinjection pain is often violent and immediate.
2. In large posterior anular tears with intact posterior longitudinal ligaments, the pain occurs at the end of the injection when resistance to the injection is felt to start.

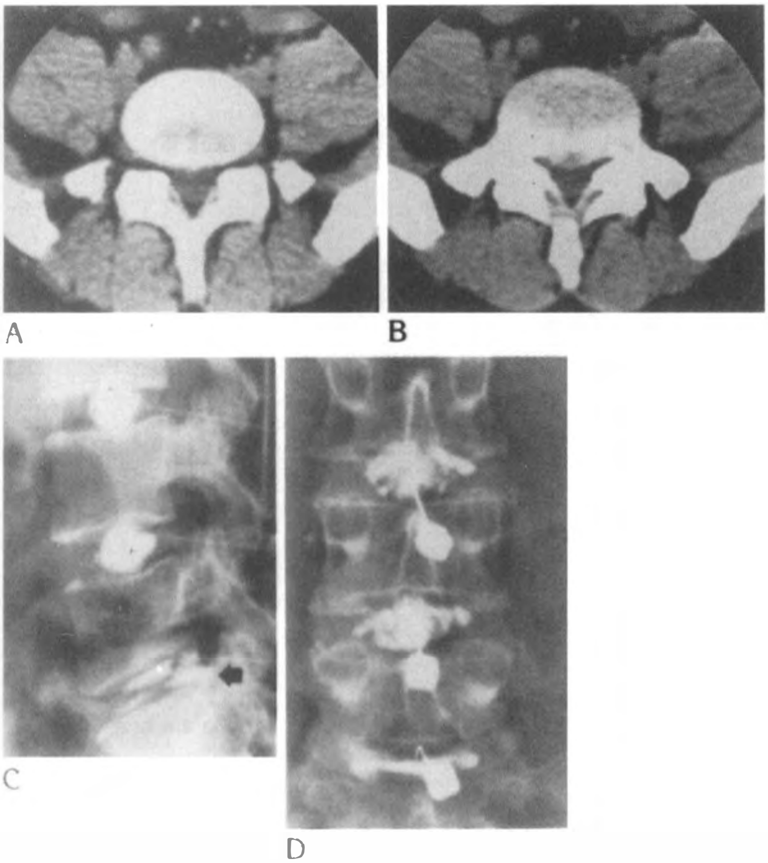


Figure 10.31. A 24-year-old man with low back pain radiating to both lower extremities. **A** and **B.** Consecutive 5-mm computed tomography sections through the L5–S1 disc space show small posterior central herniation without obvious nerve root compression. **C.** Three-level discogram, lateral projection, shows complete rupture of the L5–S1 posterior anulus with reflex of contrast medium beyond the posterior margin of the disc space (*arrow*). Injection reproduced the patient's typical low back pain with radiation down the left buttock and thigh. **D.** Anteroposterior view shows the central direction of the posterior anular tear. (Reprinted with permission from Milette PC, Fontaine S, Lepanto L, et al. Radiating pain to the lower extremities caused by lumbar disc rupture without spinal nerve root involvement. AJNR 1995;16:1605–1613.)

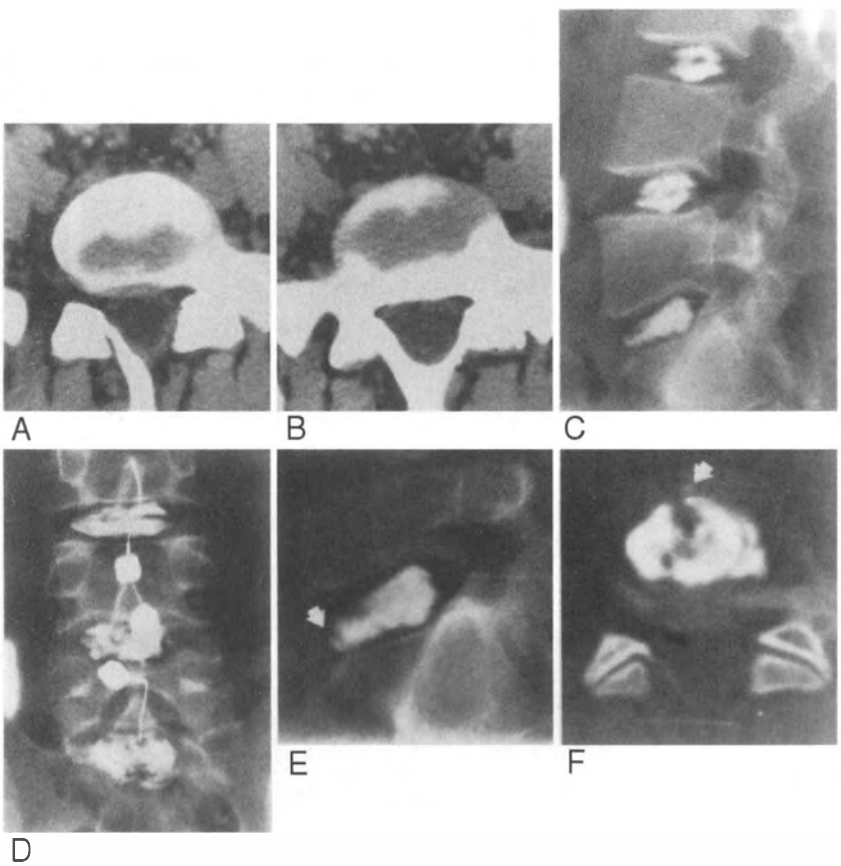


Figure 10.32. A 23-year-old man with low back pain radiating to the left buttock and left thigh. **A** and **B.** Consecutive 5-mm computed tomography (CT) sections through the L5–S1 disc space (suboptimal because inclination of the disc plane exceeded the maximal gantry tilt capacity). The disc appears normal. **C** and **D.** Three-level discogram shows abnormal extension of contrast medium into the central anterior part of the anulus of the L5–S1 disc. Injection into the disc reproduced the patient's typical symptoms, including radiation to the left buttock and thigh. Injection of L3–L4 and L4–L5 discs did not cause any pain; these discs show a normal appearance. **E.** Close-up lateral view of the L5–S1 disc shows incomplete rupture of the anterior anulus with extension of contrast to the approximate level of the inner concentric fibers of the outer part of the anulus (*arrow*). **F.** Postdiscogram CT section through the L5–S1 disc (not part of the usual protocol) confirms the limited extension of the anterior tear to the approximate junction of the internal and external parts of anulus (*arrow*). This study also failed to demonstrate the left direction of the tear or additional tears leading to the left side of the disc, which could explain this patient's radiating pain to the left lower extremity. (Reprinted with permission from Milette PC, Fontaine S, Lepanto L, et al. Radiating pain to the lower extremities caused by lumbar disc rupture without spinal nerve root involvement. AJNR 1995;16:1605–1613.)

3. Large posterior anular tears and posterior ligament tears produce little pain on injection. The contrast agent can be two or three times the amount normally injected because it can flow into the anterior vertebral canal. In a normal disc injection of contrast medium for discography, 1.0 mL to 2.0 mL (by hand injection using a 5 mL ordinary plastic syringe) was allowed within the nuclear space. A ruptured disc will allow more fluid without the high resistant pressure of a normal disc.
4. The contrast agent is not the irritating factor in discography (49).

Injection of local anesthetic into an intact anulus fibrosus with resultant relief of lower extremity pain supports the concept of discogenic pain and the conclusion that a simple disc anular tear, without direct nerve root compression by disc material, can account for low back pain with radiating pain to the leg. The fact that these discs are labeled "degenerated bulging discs" misleads the referring physician and the patient to think that the cause of the symptoms has not been identified (49).

Classification of Discographic Findings (60)

Figure 10.2 is the Dallas grading system for discogram changes:

Anulus Degeneration

0 = no change	No anular distortion
1 = local (10%)	Into inner anulus
2 = partial (< 50%)	Into outer anulus
3 = total (> 50%)	Beyond outer anulus

Bernard (61) classified disc appearance on CT-discography into seven types:

Type I:	Normal CT-discogram (Fig. 10.33)
Type II:	Anular tearing (Fig. 10.34)
Type III:	Anular tears leading to radial fissuring (Fig. 10.35)
Type IV:	Protruding disc herniation (Fig. 10.36)
Type V:	Extruded disc herniation (Fig. 10.37)
Type VI:	Sequestrated disc herniation (Fig. 10.38)
Type VII:	Internal disc disruption (Fig. 10.39)

Discography Demonstration—Normal and Abnormal

Figure 10.40 is a discogram of a normal nucleus pulposus within the anulus fibrosus.

Figure 10.41 is a frontal view of a discogram revealing right lateral escape of nuclear material into the anulus fibrosus of the disc.

Figure 10.42 shows both L4–L5 and L5–S1 escape of nuclear material into the anulus fibrosus of the discs. Note also the

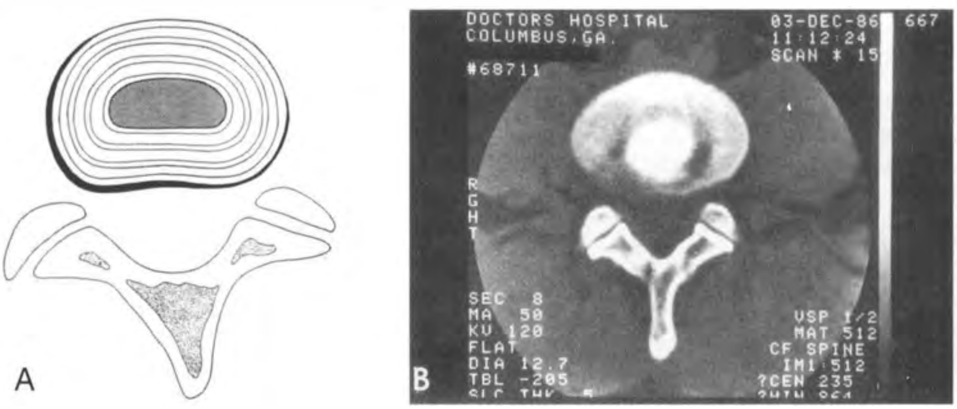


Figure 10.33. **A.** Schematic of a normal computed tomography (CT)-discogram, type 1. **B.** The internal disc morphology is more clearly seen on this normal CT-discogram using the bone window setting. (Reprinted with permission from Bernard T. Lumbar discography followed by computed tomography: Refining the diagnosis of low back pain. Spine 1990;15(7):690–707.)

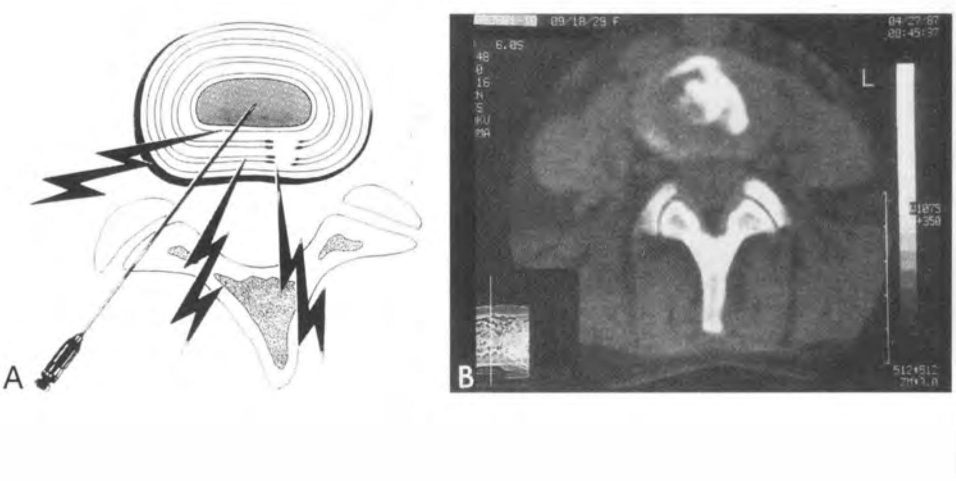


Figure 10.34. **A** and **B.** Pain may be the only abnormal finding in type II degenerated disc because insufficient anular tears exist for a radial fissure to be seen. (Reprinted with permission from Bernard T. Lumbar discography followed by computed tomography: refining the diagnosis of low back pain. Spine 1990; 15(7):690–707.)

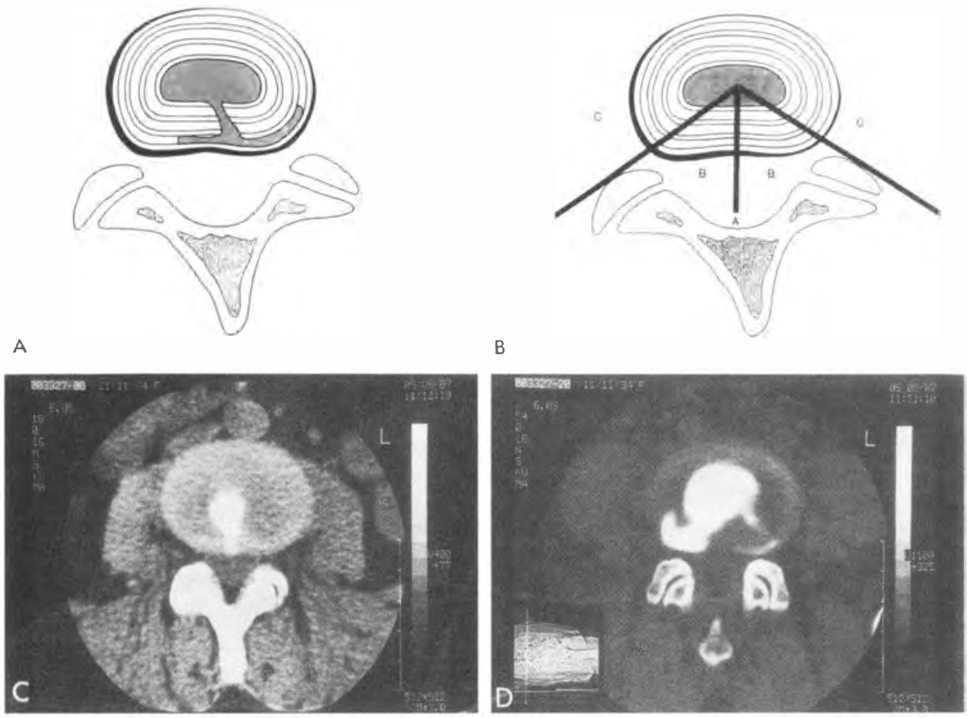


Figure 10.35. **A** and **B.** Confluence of anular tears leads to radial fissuring, which can occur posteriorly, posteriolaterally, or laterally. **C** and **D.** Examples of type III radial fissuring. (Reprinted with permission from Bernard T. Lumbar discography followed by computed tomography: Refining the diagnosis of low back pain. Spine 1990;15(7):690–707.)

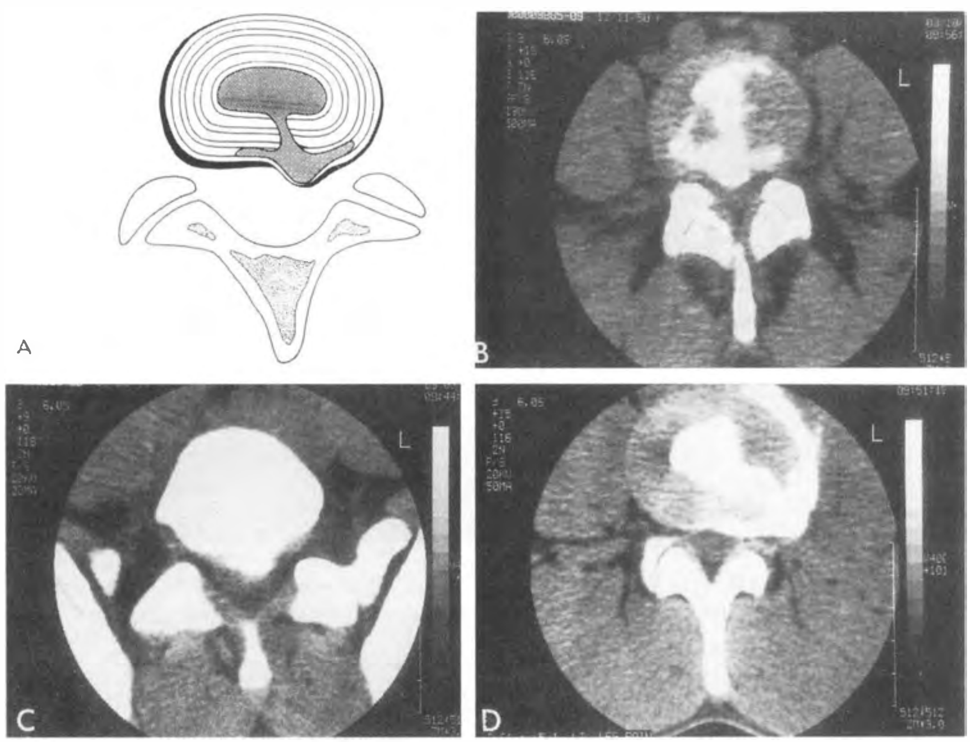


Figure 10.36. **A.** Type IV represents a protruding disc herniation. **B–D.** Types IVA, IVB, and IVC. (Reprinted with permission from Bernard T. Lumbar discography followed by computed tomography: Refining the diagnosis of low back pain. Spine 1990;15(7):690–707.)

Figure 10.37. Type V: extruded disc herniation. (Reprinted with permission from Bernard T. Lumbar discography followed by computed tomography: Refining the diagnosis of low back pain. Spine 1990;15(7): 690–707.)

Figure 10.38. Type VI: sequestered disc herniation. (Reprinted with permission from Bernard T. Lumbar discography followed by computed tomography: Refining the diagnosis of low back pain. Spine 1990; 15(7):690–707.)

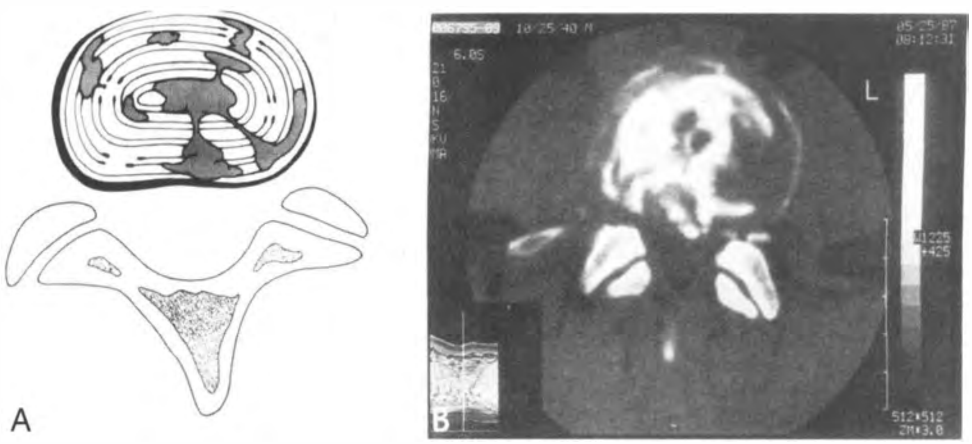


Figure 10.39. **A** and **B.** Type VII: internal disc disruption. (Reprinted with permission from Bernard T. Lumbar discography followed by computed tomography: Refining the diagnosis of low back pain. Spine 1990;15(7):690–707.)

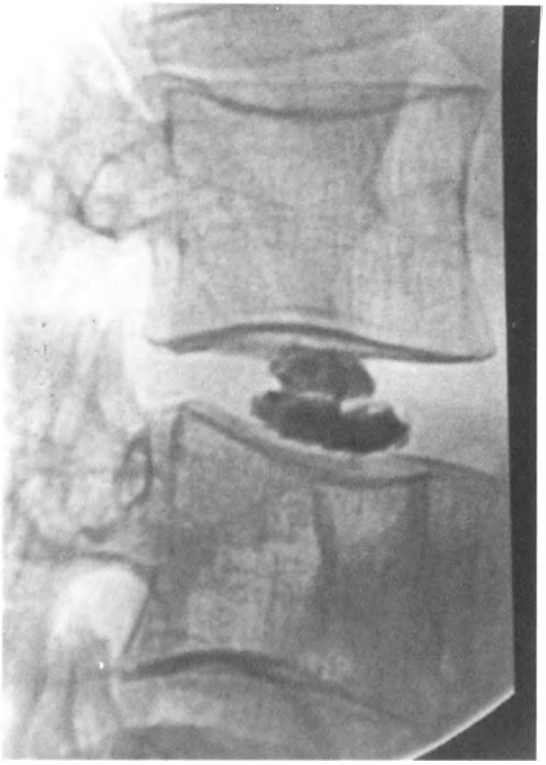


Figure 10.40. Normal discogram.

Figure 10.42. L5–S1 advanced disc degeneration showing nuclear leaking; not L4–L5 normal disc space shows marked internal disruption of the anulus with nuclear leaking.

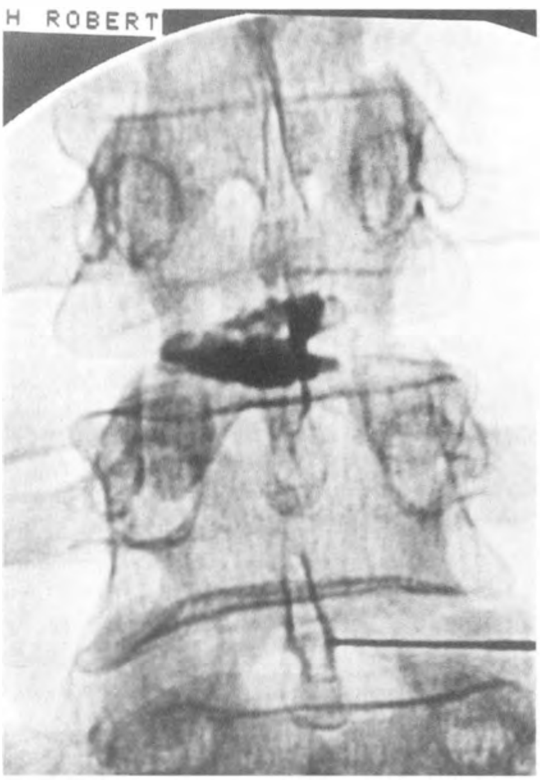


Figure 10.41. Right lateral nuclear leaking.

major thinning of the L5–S1 disc space and the contrasting relatively normal appearing space at the L4–L5 disc level where marked internal disruption of the anulus with escape of the dye into the outer zone of the anulus fails to exhibit narrowing of the disc space as might be expected with such a marked degenerative change. It is an example of the contrast between the disc appearance on plain x-ray film and actual visualization via discography.

Figure 10.43 further reveals the lateral migration of nuclear material into the anulus fibrosus on this frontal view of the disc seen in Figure 10.42 at the L5–S1 level.

Figure 10.44 shows escape of the contrast medium anterior to the vertebral body and flowing inferiorly. This type of study is enlightening as it shows the degree to which nuclear material can escape and track superiorly or inferiorly along the vertebral bodies, either subligamentous or extraligamentous. Also, the formation of traction spurs and osteophytes at the sites of anular fiber tearing and nuclear escape can be appreciated from this study.

Figure 10.45 exhibits marked escape of dye into the outer zone of the anulus fibrosus, indicating progressive radial fissuring of the disc to allow such internal disruption. The dye has leaked through the lateral anulus (*arrow*).

Figure 10.46 shows unilateral escape of dye with a strange deformation of the flow at the outer anular margin (*arrow*).

Figure 10.47 demonstrates the cloacal flow of dye posteriorly through a radial tear in the anulus fibrosus (*arrow*).

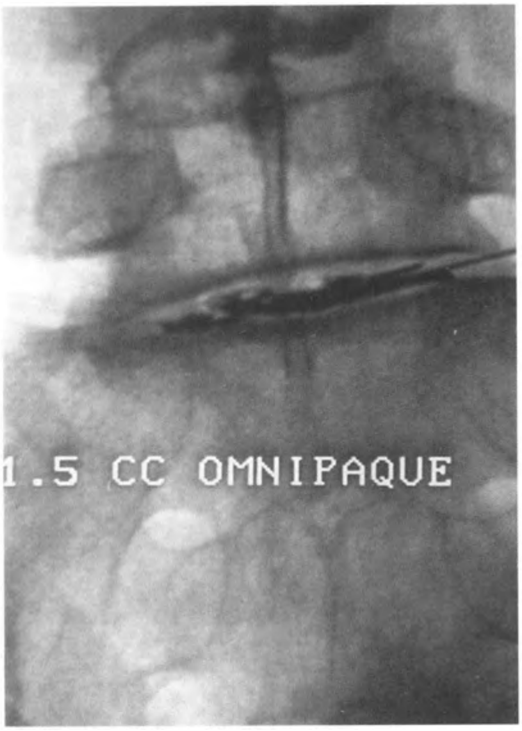


Figure 10.43. Marked lateral nuclear migration into the anulus.

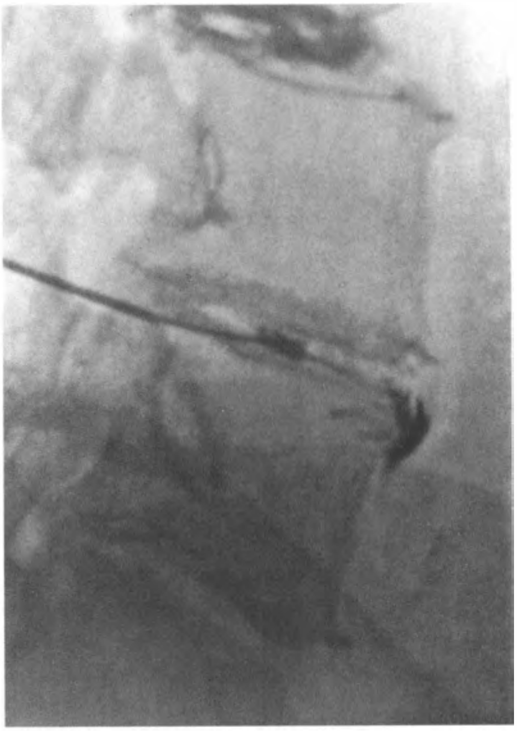


Figure 10.44. Anterior nuclear leaking.

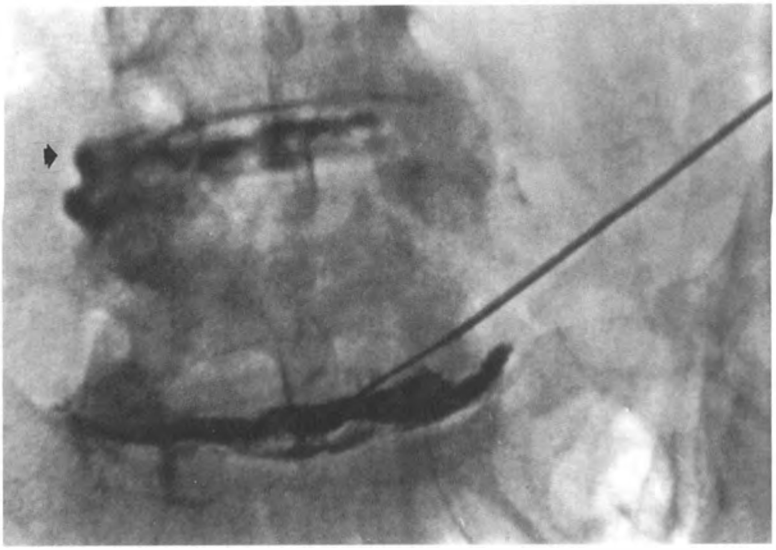


Figure 10.45. Outer anular leaking of contrast material.

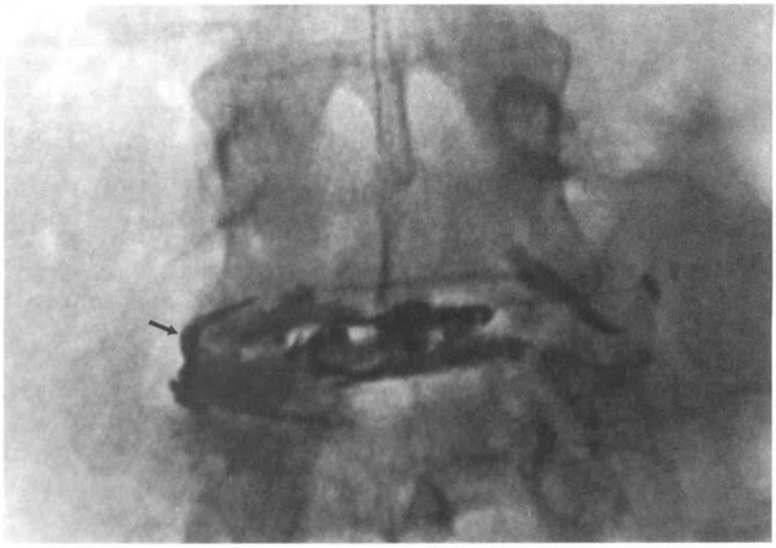


Figure 10.46. Unilateral contrast media creating strange deterioration of outer anular margin.

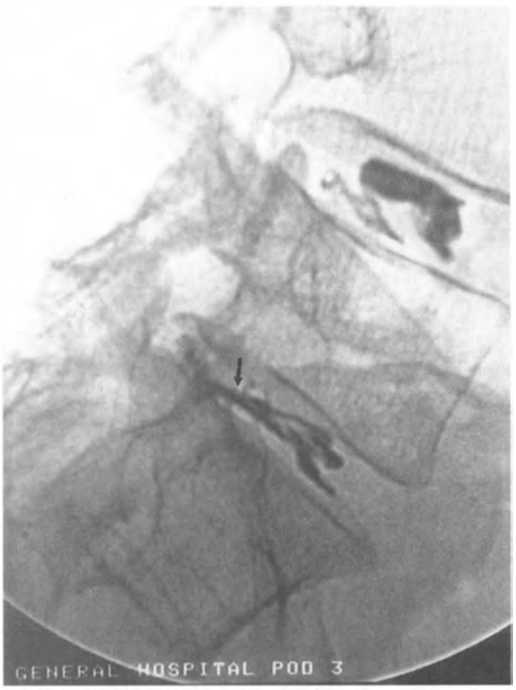


Figure 10.47. Posterior radial tear of anulus.

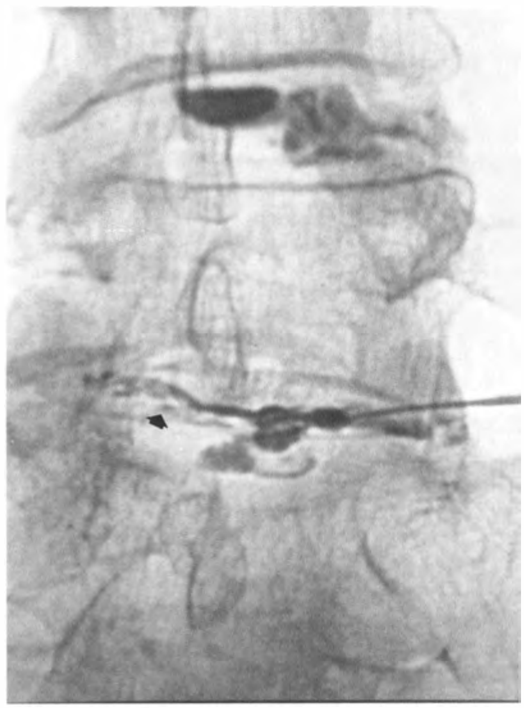


Figure 10.48. Frontal view of Figure 10.47 showing lateral escape of contrast.

Note how the flow is directed toward the vertebral body plate of the sacrum, which is common for radial fissures to do.

Figure 10.48 is a frontal view of Figure 10.47 showing the dye also escapes laterally (*arrowhead*), which is not appreciated on the lateral projection in Figure 10.47.

Figures 10.49 and 10.50 are the lateral and frontal views of the L3-L4 disc space showing both the posterior and lateral escape of the dye through radial anular tears and internal disruption of the disc.

Seeing the tearing and internal disruption of the discs on these discograms enhances appreciation for the nerve innervation within the anular fibers and the potential source of pain they represent. Further, in discs that appear normal on plain x-ray film, or even MRI, the realization that such advanced internal change can take place and escape detection without discography is disturbing to the clinician evaluating low back pain patients.

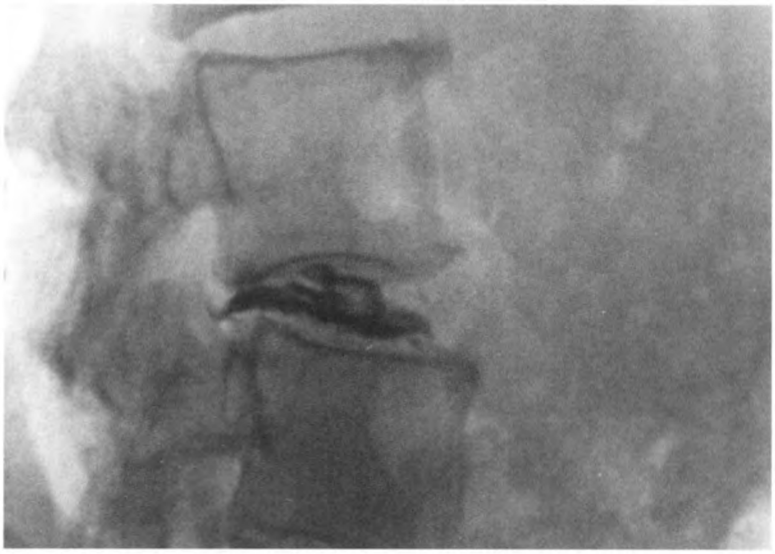


Figure 10.49. Lateral view of L3–L4 disc space showing posterolateral contrast leak.

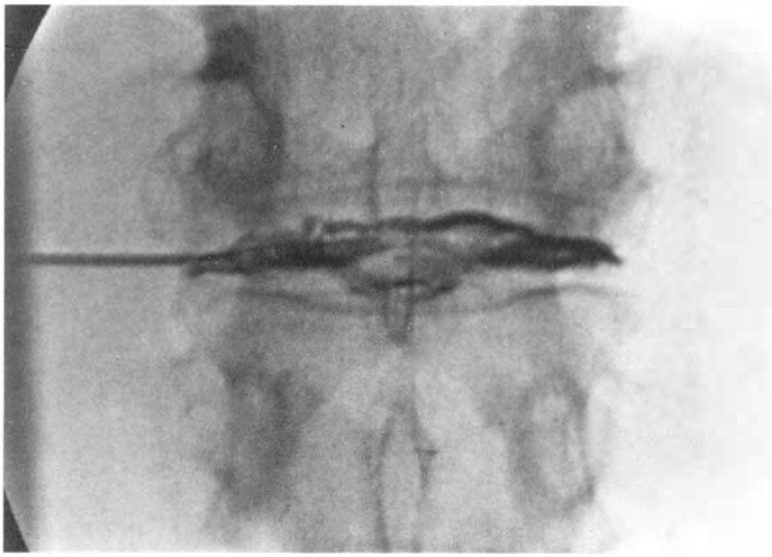


Figure 10.50. Frontal view of L3–L4 disc space showing posterolateral contrast leak.

Case 4

A 33-year-old woman complained of left low back pain and a "pins and needles" feeling down both anterior and posterior thighs, legs, and feet. The left foot swells and is cold to touch. The middle toes are more numb than the others. The pain started following an air compressor falling on her. Chiropractic care after the injury did not help her and she lost 30 pounds. Moving furniture caused increased numbness and tingling of the left lower extremity, and an MRI of the lumbar spine showed degenerative L5–S1 disc disease. Naprosyn did not help. Persistent, unrelenting pain to all forms of care led to a discogram being ordered.

Figures 10.51 and 10.52 are anteroposterior and lateral lumbar spine studies which reveal minor anterolateral vertebral body end plate hypertrophy. Surgical clips are from a nephrectomy because of an infection.

Figures 10.53 and 10.54 are lateral and frontal discogram views showing leaking of nuclear material at the L2–L3 and L3–L4 levels anteriorly, laterally, and posteriorly into the anulus fibrosus.

Figure 10.55 reveals L3–L4 nuclear material to leak anteriorly (*long arrow*), under the anterior longitudinal ligament as a subligamentous leak, as well as posterolaterally (*short arrow*).

Figure 10.56 at the L5–S1 level shows right posterolateral nuclear leak (*arrow*), which does not cause a focal herniation of anular material into the vertebral canal. Therefore, all of these discography studies fail to show any disc herniations into the vertebral or osseoligamentous canals to cause cauda equina or nerve root compression.

Figures 10.57 and 10.58 are sagittal T2 and axial cuts, which again fail to show any posterolateral disc protrusion on sagittal section, although evidence is seen of lower disc space hypointensity, especially at L5–S1. Figure 10.58 specifically is shown to reveal the absence of the right renal shadow and the presence of the left kidney (*arrow*).

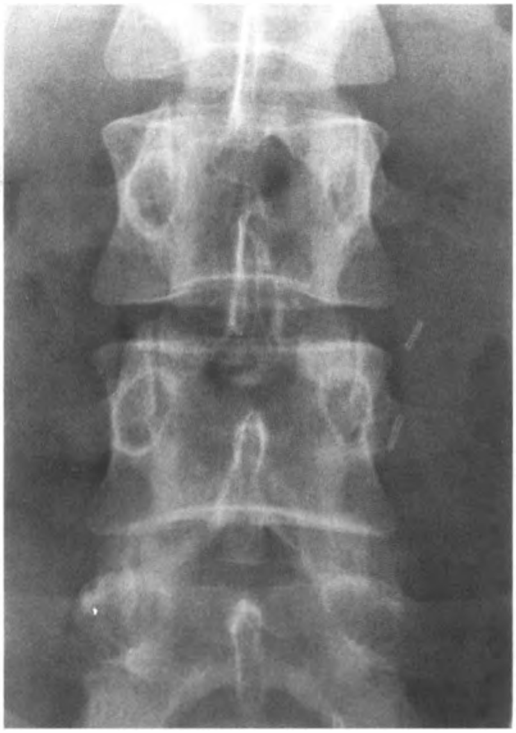


Figure 10.51. Anteroposterior lumbar spine radiograph.

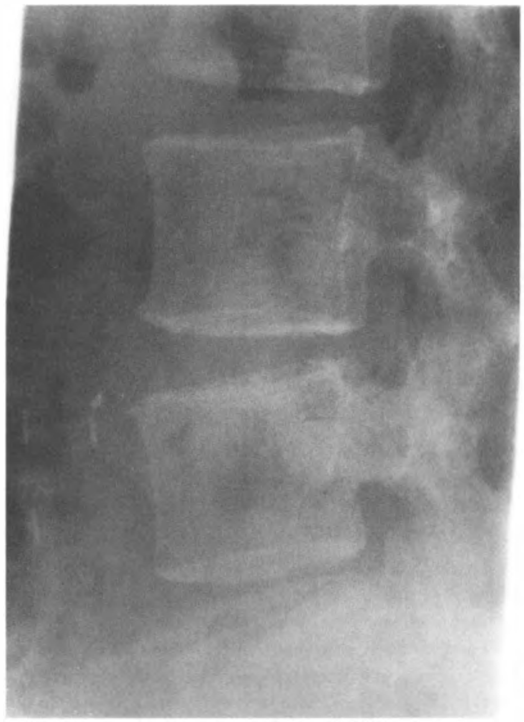


Figure 10.52. Lateral lumbar spine radiograph.

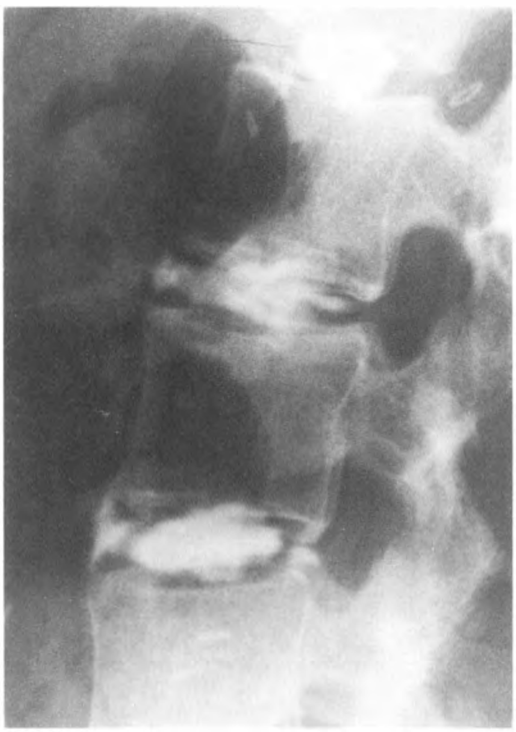


Figure 10.53. L2–L3 and L3–L4 nuclear leaking—lateral view.

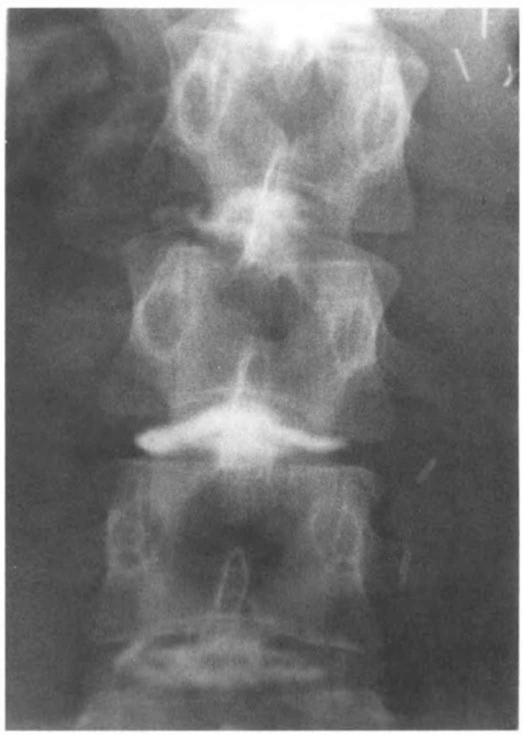


Figure 10.54. L2–L3 and L3–L4 nuclear leaking.

Figure 10.55. L3–L4 anterior nuclear leaking and posterolateral leaking.

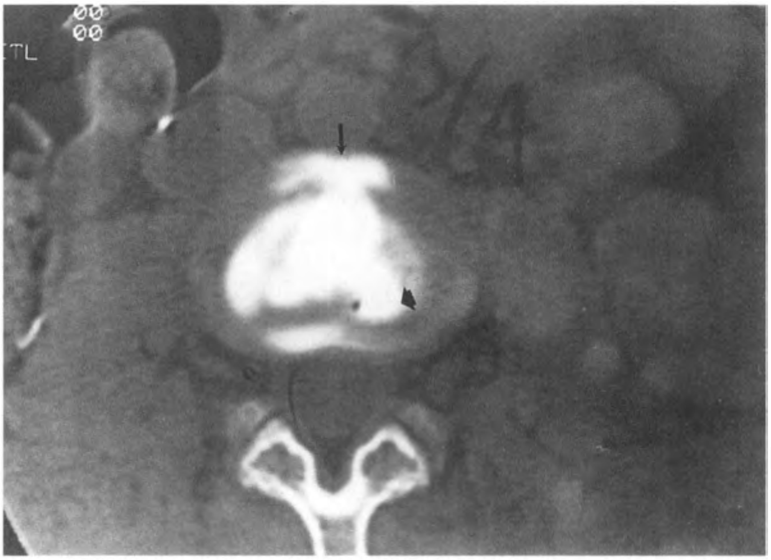


Figure 10.56. L5–S1 right posterolateral nuclear leaking without herniation.

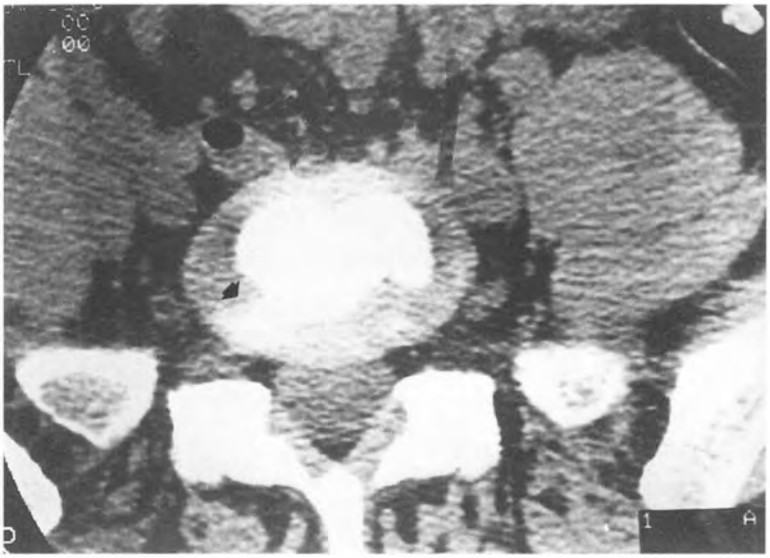


Figure 10.57. Sagittal magnetic resonance imaging shows hypointensity of disc.

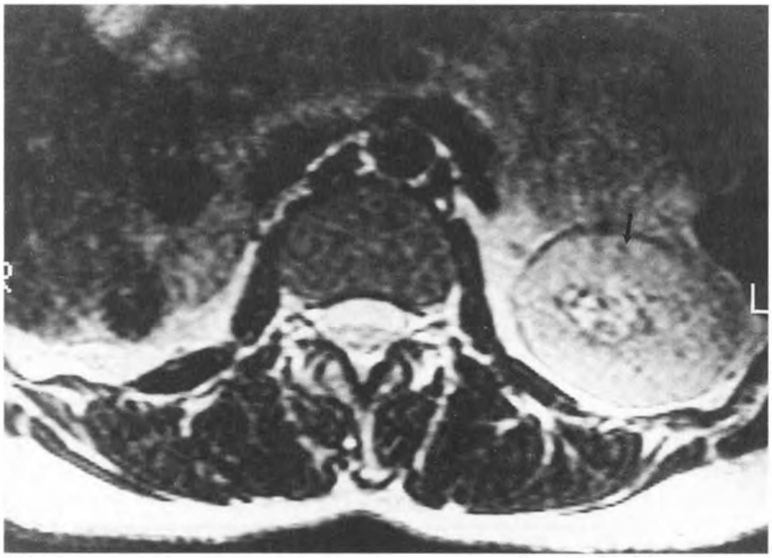


Figure 10.58. Axial magnetic resonance imaging shows right renal shadow absence.

From this discographic study the impression was that all lumbar discs except L1–L2 were internally deranged with escape of nuclear material anteriorly, laterally, and posteriorly throughout the lumbar spine. The L2–L3 level showed anterior and left posterolateral disc nuclear escape, the L3–L4 level revealed anterior and left posterolateral nuclear escape (Fig. 10.55), the L4–L5 level revealed lateral escape of nuclear material, whereas the L5–S1 level (Fig. 10.56) revealed right posterolateral nuclear escape that did not cause discal herniation.

As discussed throughout this textbook, it is to be remembered that anular fiber irritation is a cause of low back, flank, groin, and thigh pain. In light of the fact that this patient had no evidence of disc herniation to warrant surgery, the clinical assumption was made that her pain was caused by anular disc disruption and nuclear escape into the anular rents.

Treatment was distraction adjustments and range of motion restoration of the lumbar spine. No progressive neurologic deficits were seen in this case. The patient attended low back wellness school, did the Cox low back pain exercises for strength and flexibility, took glycosaminoglycan and glucosamine sulfate, and returned home for care to a local chiropractor following 3 weeks of care in our clinic. The result was a slow relief of the low back and left lower extremity pain. No other follow-up is known.

Criteria for Discography Use

The Executive Committee of the North American Spine Society states: "Discography is indicated in the evaluation of patients (*a*) with unremitting spinal pain, with or without extremity pain, of greater than four months' duration; (*b*) when the pain has been unresponsive to all appropriate methods of conservative therapy; (*c*) patients should have undergone investigation with other modalities . . . [CT, MRI, myelography]; (*d*) when a decision has been made that the clinical problem will require surgical management" (62).

Position Statement From the North American Spine Society Diagnostic and Therapeutic Committee

"Particular applications include patients with persistent pain in whom disc abnormality is suspect, but noninvasive tests have not provided sufficient diagnostic information or the images need to be correlated with clinical symptoms. In patients in whom fusion is being considered, discography's role in such cases is to determine if discs within the proposed fusion segment are symptomatic and if the adjacent discs are normal. Discography appears to be helpful in patients who have previously undergone surgery but continue to experience significant pain. In such cases, it can be used to differentiate between postoperative scar and recurrent disc herniation and to investigate the condition of a disc within, or adjacent to, a fused spinal segment to better delineate the source of symptoms. Discography can be used to confirm a contained disc herniation, which is generally an indication for such surgical procedures. Frequently, discography is followed by axial computed tomography scanning to obtain more information about the condition of the disc" (63).

Discography Differentiates Herniated from Degenerated Discs

A herniated disc is painful on discography, but degenerative disc disease is not painful on discography (1477 IVDs in 523 patients). Pain provocation showed little relation to intradiscal deterioration, whereas a strong relation was found between pain and herniated nucleus pulposus (64). The end plate may be a possible pain source during clinical discography (65). Discographically painful discs may be surgically arthrodesed for relief (66).

Complications Reported with Discography

Diagnostic discography was complicated by discitis in 7 of 4400 injections (16%). Spinal cord compression is a rare complication of cervical discography. The inherent danger associated with discography mandates blinded, controlled clinical trials to establish the true efficacy of the procedure in evaluating degenerative disc disease (67).

Syndesmophytes Revealed by Discography to be Anterior Disc Protrusion

Anterior protrusion of a lumbar disc is a recognized lesion since Cloward first reported it more than 40 years ago by discography, and it is one of the causes of syndesmophyte formation. Perhaps an anterior syndesmophyte could serve as an indication for performing discography (68).

Torsion Shifts Axial Motion Posterolateral Where Discography Shows Herniation

Lumbar flexion-extension and rotation movements under axial loads are thought to be important factors implicated in lumbar disc herniation, with all these movements combining to form complex loads that induce disc herniation. Flexion-extension movement under axial loading accelerated the formation of posterior anular fissures at the weak points of the disc. Torsion shifts the center of spinal movement to a posterolateral direction in the disc. Interestingly, these directions are identical to the herniation routes of the intraforaminal and extraforaminal disc herniations shown by computerized lumbar discography as oblique routes to the sagittal plane in the disc. Lumbar computed tomographic discography allows the herniation routes to be observed in detail, and these findings provide useful clinical information (69).

WHEN TO ORDER DIAGNOSTIC IMAGING STUDIES

The appropriate time to order radiologic or other studies such as CT and MRI depends on patient status and response to treatment (70).

MRI Used Only After 4 to 6 Weeks of Conservative Care

For the patient with acute low back pain, ordinarily no initial imaging studies are needed. If fracture, tumor, or infection is suspected, however, plain x-ray studies may be helpful in ruling them out. If radiculopathy, signs of neural compression, or back pain unresponsive to conservative therapy persists after 4 to 6 weeks, MRI may help to provide a definitive diagnosis. CT is appropriate if stenosis or spondylolysis is suspected yet inadequately depicted by MRI. If multiple levels of pathology are suspected, selective nerve root blocks or discography can help determine at which level the back pain is originating. Concentric anular bulging is a normal MRI finding in the aging spine, appearing in 80% of asymptomatic patients aged more than 60 years (71).

MRI Combined with Positive Neurologic Signs Confirms the Diagnosis of Herniated Disc

If the patient has radiating leg pain below the knee, paraesthesia of the dermatome, positive straight leg raise, and neurologic deficits, an abnormal MRI will confirm the appropriate diagnosis (72).

The following outline lists indications for ordering diagnostic imaging for patients with suspected radiculopathy caused by intervertebral disc herniation (70).

If the patient has . . .	Order imaging studies . . .
Loss of bladder or bowel function or rapid deterioration in neurologic function	Immediately, on an emergency basis
Slow, progressive neurologic loss of motor or sensory or reflex function	As soon as possible, to avoid future permanent neurologic deficits
No neurologic deficits but severe pain	After a 4 to 6 week delay while conservative treatment is attempted to resolve pain; sooner if patient is severely incapacitated and bedridden
Mobility with some leg pain	After a 6 to 10 week delay unresponsive to conservative treatment and depending on results of clinical examination and treatment
More leg pain than back pain	Earlier rather than later
Back pain only	Perhaps never, because radiographic results are unlikely to change treatment protocol significantly

MRI Evidence of Disc Bulge or Herniation May Be Frequently Coincidental

More than 50% of asymptomatic persons show bulging or herniated discs on MRI or other imaging modalities, and they should be regarded as normal findings unless clinical findings confirm their importance. Back pain affects nearly half of all adults during a given year, and about two thirds of adults have back pain at some time in their lives. Up to 85% of patients with low back pain cannot be given a definitive diagnosis (73).

One expert neuroradiologist was 30% more likely to interpret a study as showing a disc protrusion than a second expert neuroradiologist reading the same films. More precise terminology in the interpretation of imaging studies along with the recognition of the wide range of normal findings will allow better use of modern imaging procedures (73).

MRI Lacks Specificity and Sensitivity

Sensitivity is the ability of a test to accurately identify a disease by being positive. Specificity is the ability of a test to identify patients without disease, or to find a negative outcome for the presence of a disease. MRI can identify a lesion, but is unable to detail the relationship of the finding with the patient's symptoms. Figure 10.59 is a chart showing the CT and MRI positive findings of normal persons showing disc herniation, spinal stenosis, facet abnormality, or other pathology. Note that 79% of persons over age 60 show bulging discs (74).

Computed tomography and magnetic resonance imaging results on normal subjects

CT results: "normal subjects" (N = 52)

	Age	
	Under 40	Over 40
Herniated disc	20%	27%
Spinal stenosis	0%	3%
Facet abnormality	0%	10%
Any abnormality	20%	50%

From Wiesel et al. (1984).

MRI results: "normal" subjects (N = 67)

	Age	
	Under 60	Over 60
Herniated disc	22%	36%
Spinal stenosis	1%	21%
Bulging disc	54%	79%
Degenerated disc	46%	93%

From Boden et al. (1990).

Figure 10.59. (Reprinted with permission from Deyo R. Understanding the accuracy of diagnostic tests. In: Weinstein JN, Rydevik ABL, Sonntag VKH, eds. Essentials of the Spine. New York: Raven Press, 1995:65.)

MRI Confirmation of Disc Herniation in Asymptomatic Persons

Magnetic resonance imaging examination of 41 women without symptoms showed that 54% had a disc bulge or herniation at one or more disc spaces at the L3–L4, L4–L5, and L5–L1 levels. Anular "tears" can be painful, possibly because of the contents of the nucleus pulposus leaking into the epidural space with related nerve irritation. The reported prevalence of posterior radial tears at autopsy in asymptomatic people is 40% for those between ages of 50 and 60 years and 75% for those between 60 and 70. Anular tears may lead to disc degeneration (75).

No Correlation Between Pain and Disability and Disc Size or Type

Twenty-five patients underwent physical examinations at 6 weeks and 6 months. Initial symptoms and clinical course were correlated with type, size, location, and enhancement of disc herniations. Agreement between clinical and MRI findings for level and side of herniated nucleus pulposus and radicular symptoms was excellent. *There was no correlation of pain and disability with disc size, behavior, or type* (76).

Matsubara et al. (77) reported on 32 conservatively treated sciatica patients with MRI-proven lumbar disc herniations. MRI was performed at the acute onset, and at 6 and 12 months after relief. The spinal canal occupied by the disc herniation in the acute stage was 32%, on average, 29% at 6 months, and 25% at 1 year. The size of the disc herniation decreased 20% in size in 34% of the patients, 10 to 20% in 28% of the patients, and was unchanged in 38% of the patients. *Symptoms and signs do not correlate with the degree of herniated nucleus pulposus reduction.*

The mere presence of neural compromise in a disc herniation does not signal that it is symptomatic or requires treatment (78).

Surgical Need Is a Clinical, Not An Imaging, Decision

Most imaging shows abnormal discs. Both doctor and patient must understand that these are most commonly caused by incidental degenerative conditions and that the imaged abnormality does not necessarily explain the symptoms (79).

The indications for disc surgery are clinical, few, and clearly defined:

1. Sciatic pain, not relieved after an adequate first trial of rest (generally, about 3 weeks).
2. Return of sciatic pain within 1 year.
3. Progressive or profound weakness of foot movement.
4. Urinary bladder retention occurs rarely.

The indications for surgery for prolapsed intervertebral disc or spinal stenosis are clinical. No radiologic indications are found for surgery. Imaging serves to confirm the clinical diagnosis and, at times, to locate the condition more precisely (79).

Asymptomatic Patients Show Disc Herniations on MRI

Magnetic resonance imaging studies of the lumbar spine compared 46 asymptomatic and 46 patients with low back and sciatic pain and found 76% of the asymptomatic patients showed disc herniations on MRI versus 96% of the symptomatic patients (80).

Early Imaging Discouraged Without Presence of Neurologic Complications (81)

"Let's get a magnetic resonance imaging scan to see if there is anything wrong with the spine" is the beginning of a dangerous thought process. This danger arises from the high prevalence of abnormal findings on images of asymptomatic individuals. Excessive reliance on diagnostic studies without precise clinical correlation can lead to erroneous or unnecessary treatment of degenerative disorders of the lumbar spine (82).

When and What to Image

Acute low back pain. Imaging is not necessary during the first 6 weeks if the patient does not have neurologic findings, constitutional symptoms, a history of traumatic onset of the symptoms or of a malignant tumor, or an age of more than 50 or less than 18 years. After 6 weeks, if no clinical improvement

has occurred, plain anteroposterior and lateral radiographs may be ordered.

Chronic low back pain. MRI is the best imaging modality for the assessment of intervertebral disc degeneration.

Herniated disc. MRI.

Leg pain without neural compression. Chemical radiculitis can result from leakage of irritants from the nucleus pulposus through an anular tear. In such a situation, discography may demonstrate extravasation of contrast medium at the correct level and side of the affected nerve root.

Failed back surgical syndrome. It has been estimated that 300,000 first-time laminectomies are performed in the United States annually, and as many as 15% of these patients may have continued or recurrent pain and disability.

Nonunion of the site of a spinal arthrodesis is difficult to diagnose with use of noninvasive imaging. Stereophotogrammetry can help document small degrees of motion. Plain tomography can be useful in observing the trabecular bone pattern and the continuity of the fusion mass, and CT (axial or three-dimensional reconstructions) can increase visualization of a lumbar fusion mass (82).

Pain in a lower limb after a previous operation needs imaging to distinguish scar tissue from treatable entities, such as a residual or recurrent herniation of an intervertebral disc or spinal stenosis. With unenhanced CT, scar tissue can be distinguished from disc material in 43 to 60% of these patients. CT with intravenous injection of a contrast agent increases the likelihood of a correct diagnosis to 70 to 83%. The diagnostic accuracy of contrast medium-enhanced MRI approaches 96 to 100%.

Arachnoiditis can be seen on MRI scans postoperatively as one of three distinct patterns: central clumping of the nerve roots, the appearance of an empty sac because of peripheral clumping of the nerve roots, and a soft tissue mass in the subarachnoid space. Arachnoiditis occurs in less than 5% of patients who have persistent symptoms postoperatively (82).

Physician Opinion Determines Testing

Patient symptoms and findings do not dictate testing, rather physician opinion does. Less than 20% of family physicians or orthopaedic surgeons would order imaging studies for acute uncomplicated back; however, more than 50% of neurosurgeons or neurologists would order such studies. A need for additional clinical guidelines as well as better adherence to existing guidelines is needed. When indirect costs associated with disability compensation and lost productivity are included, the total annual costs associated with back pain in the United States may be as high as $100 billion. Early imaging has been discouraged and should generally be reserved for patients with neurologic abnormalities suggesting nerve root compression whose pain has not been relieved after several weeks of conservative therapy (81).

Imaging Limited For Low Back Pain and Sciatica Patients

The American Society of Neuroradiology made the following observation and strong statement:

"Which MR pulse sequences are best for imaging the lumbar spine?", although still useful, is no longer the most important question; rather it is, "Is imaging necessary for the workup of low back pain?"

It is well known in spine imaging (cervical and lumbar) that approximately 30 to 35% of asymptomatic patients will show disc abnormalities on either CT or MRI studies.

In one study of 120 patients with herniated disc disease, 82% had only conservative treatment; of those, 71% had a "good outcome." Of those patients treated conservatively and rescanned by CT, 63% showed a decrease in the herniated nucleus pulposus, 29% showed no change, and 8% had an increase in the herniated nucleus pulposus (83).

What explains the decrease in the herniated nucleus pulposus on the CT scan in the face of conservative treatment? The herniated disc material actually may be resorbed. Portions of it may regress back into the native disc space over time. Another possibility is that on the CT scan, what is called a "herniated disc" may, in fact, be inflammatory tissue plus disc, making the initial assessment of the disc to appear larger than it really is.

Why didn't CT findings discriminate between outcomes, that is, between patients requiring surgery and those who responded to conservative treatment? As a result of this observation, one legitimately can ask what the purpose of an imaging study is if it does not have an impact on treatment or outcome.

For patients with sciatica, no imaging studies appear warranted because they do not change treatment. It is not yet clear whether imaging is even indicated in those patients who fail the initial course of conservative treatment. For low back pain that is not or is atypical of sciatica, imaging is likely indicated. It is, therefore, important for the radiologist, in conjunction with other subspecialists, to develop guidelines for primary care physicians to when an imaging study is appropriate in the workup of back pain (83).

Conservative Care Urged Before MRI or Other Imaging Ordered

Magnetic resonance imaging has not improved surgical or nonsurgical management strategy. Without neurologic deficit, evidence of fracture, infection, or neoplasia, nonsurgical therapy should be administered for adult patients with low back pain syndrome of less than 7 weeks duration before further diagnostic imaging is ordered (84).

COMPARISON OF IMAGING MODALITIES IN DISC HERNIATION DIAGNOSIS

MRI Found Superior to Other Imaging

The sensitivity, specificity, and accuracy of CT myelography (CTM), MRI, and myelography in making the diagnosis of herniated nucleus pulposus (HNP) and spinal stenosis were compared in a retrospective study involving 59 surgical procedures in 57 patients who had all three tests performed preoperatively.

It seems that CTM is the most sensitive and accurate test in diagnosing HNP and spinal stenosis, whereas myelography is the most specific, although no statistical significance was noted in this study. However, because MRI did compare favorably with CTM in most instances, particularly in revision surgery, it may be the procedure of choice because of its noninvasiveness and relative lack of side effects (85).

Magnetic resonance imaging accurately predicted the oper-

ative findings in 98 of 102 disc levels (96%). Significantly less accurate were myelography (81%) and postmyelogram CT scan (57%). When myelography and CT scan were used jointly, the accuracy was 84%. MRI is a clinically superior diagnostic test in the evaluation of patients with suspected lumbar disc herniation, and it should be the diagnostic study of choice when available. Its noninvasive nature, multiplanar capabilities, and the lack of ionizing radiation are particularly desirable for patient and physician (86).

MRI Advantages in Diagnosis

To begin the discussion of the advantages of MRI, I will share one of the finest explanations of CT and MRI physics I have read. It is written and reprinted with permission by Richard J. Herzog (87).

Magnetic Resonance Imaging/Computed Tomography

It will be assumed that one has already acquired plain films prior to ordering these additional costly studies and that they are obtained only to answer a specific diagnostic or therapeutic question.

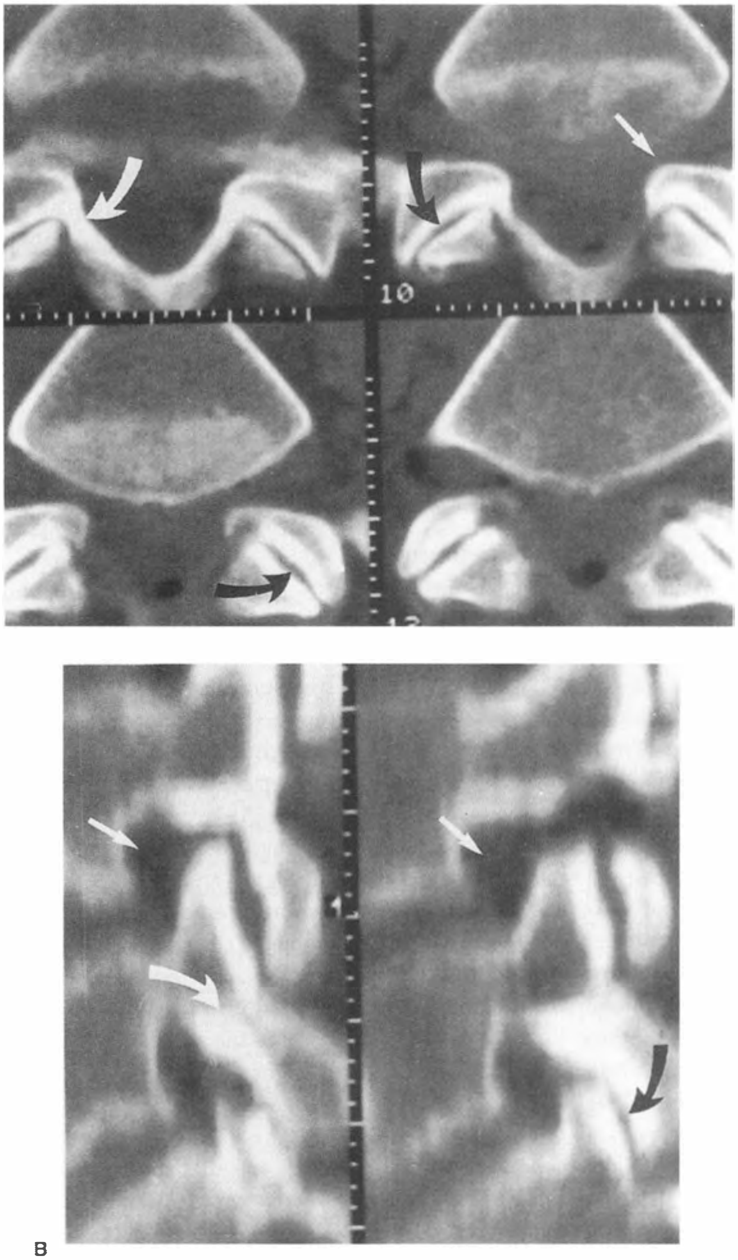
With computed tomography, an x-ray source is used to generate cross-sectional images. CT images are representations of differential x-ray attenuation by tissue. This attenuation is determined by the tissue's electron density. Spatial and contrast resolution is dependent on the energy of the x-ray source, slice thickness, field of view, and scanning matrix. A variety of pre-and postprocessing software programs are available to optimize the evaluation of soft tissue or osseous structures. To obtain a high-resolution multiplanar CT study, it is necessary to utilize thin (1.5 mm), contiguous sections in the cervical spine and overlapping (5 mm thick with a 2-mm overlap) or contiguous (3 mm thick) sections in the lumbar spine to create optimal computer-generated sagittal and coronal reconstructed images. The diagnostic quality of CT with multiplanar reformations (CT/MPR) is highly dependent on patient immobility to prevent misregistration artifacts. An entire CT study can currently be performed extremely quickly, particularly with the new spiral CT scanners, and, therefore, it is usually not difficult for a patient to maintain a single position. With current rapid scanning techniques, patient x-ray exposure has been significantly reduced, but still the risk of radiation exposure must be considered when ordering an examination. If a CT study is needed, a multiplanar exam should be performed, including sagittal and coronal reformations. Multiplanar CT can be obtained if the initial axial sections are contiguous or overlapping. The strength of CT is its excellent resolution of bone (Fig. 10.60) and, therefore, it is frequently ordered in cases of trauma to detect fractures and fracture fragment displacement. Computed tomography is also frequently obtained preoperatively in the evaluation of patients with stenosis or tumors that have invaded the osseous structures.

With the implementation of high-quality MRI and CT, it is now rare that myelography or CT/myelography is needed. It still may be indicated if a diagnosis of arachnoiditis, meningeal metastasis, dural tears, pseudomeningoceles, or epidural abscess is being considered. In some centers CT-myelography is still obtained in the preoperative evaluation of patients with spinal stenosis. Radionuclide studies are usually limited to situations where screening of the entire body is required (e.g., infection or metastatic disease). Tomographic radionuclide studies may be of benefit in the detection of stress reactions or fractures of the pars interarticularis.

Whereas an image created with an x-ray source is determined by the electron density of the tissue, MR images are a construct of totally different physical properties of tissue. If a nucleus of an atom contains either unpaired protons or neutrons, it will have a net spin and angular momentum. Each spinning nucleus is surrounded by a magnetic field and can be thought of as a small bar magnet or dipole, with a north and south pole. If the body is placed in a static external magnetic field (i.e., the MR magnet), the normal random position of the nuclear dipoles in the body will be altered, and they will align themselves along the vector of the externally applied magnetic field. A magnetization vector of the tissue, which is the sum of the dipoles oriented in the same direction as the applied static magnetic field, will be created. When the spinning nuclei are aligned in the external magnetic field, they also precess (wobble) around the axis of the applied magnetic field. At present, virtually all clinical MR imaging is performed by imaging hydrogen nuclei (proton imaging). Hydrogen is an ideal atom for imaging, being the most abundant resonant nucleus in soft tissues and providing a strong MR signal.

To create an MR image, radio waves of a specific RF are pulsed into the body, which induces the transition of a fraction of the spinning protons from their equilibrium state into a higher energy state. With the termination of the RF pulse, the excited nuclei release energy and return to their lower energy state. This characteristic absorption and release of energy is called *nuclear magnetic resonance.* The transition between energy states is necessary for the construction of an MR image. The process of returning from the excited to the equilibrium state is called *relaxation* and is characterized by two independent time constants, T1 and T2. The T1 (longitudinal relaxation time) reflects the time required for excited protons to return to their equilibrium state. When the hydrogen nucleus is excited by the application of an RF pulse, in addition to changing to a higher energy state the initially random precession of the nuclei prior to excitation will become coherent (in phase) after excitation. This results in a magnetization vector perpendicular (transverse) to the external magnetic field, which can be directly measured by a receiver coil. With the termination of the RF pulse, there is rapid loss of coherence of the precessing nuclei, and the T2 (transverse relaxation time) is the time reflecting the loss of the transverse magnetization.

T1 and T2 relaxation are intrinsic physical properties of tissue. The MR signal intensity is mainly dependent on the T1, T2, and proton density (number of mobile hydrogen



A

B

Figure 10.60. Normal lumbar spine anatomy—computed tomography scan with multiplanar reformation bone window. **A.** On the axial and (**B**) reformatted sagittal computed tomography images, there is excellent delineation of the facet joints (*curved black arrows*), neural foramina (*straight white arrows*), and pars interarticularis (*curved white arrow*). (Reprinted with permission from Herzog R. Radiologic imaging of the spine. In: Weinstein JN, Rydevik BL, Sonntag VKH, eds. Essentials of the Spine. New York: Raven Press, 1995;7.)

ions) of the tissue being evaluated. To obtain an anatomic image, spatial encoding of the energy released by the excited protons must be performed in three anatomic planes. This is accomplished by creating small gradient magnetic fields within the larger static applied field. The methods for obtaining MR data are designated *pulse sequences*. Spin echo (SE) and gradient echo (GE) are currently the pulse sequences most often employed. The spin-echo pulse sequence is probably the most commonly used MRI sequence, and the images created are dependent upon several scanning parameters. The repetition time (TR—the time between RF pulses) and the echo time (TE—the time between the application of the RF pulse and the recording the MR signal) are determined before acquiring the image. By varying the scanning contribution of the T1, T2, and proton density of the tissue will determine image contrast. A *T1-weighted image*, which emphasizes the T1

properties of a tissue, is produced with a short TR (400 to 600 ms) and a short TE (15 to 30 ms). T1-weighted images are ideal for evaluating structures containing fat, subacute or chronic hemorrhage, or proteinaceous fluid because these materials have a short T1 and yield a high signal on T1-weighted sequences. T1-weighted images, frequently thought of as fat images, are excellent in the delineation of anatomic structures. An MR image produced with a long TR (1500 to 2000 ms) and a short TE (15 to 30 ms) is referred to as a *proton-density* or *spin-density weighted image,* and the signal intensity reflects the absolute number of mobile hydrogen ions in the tissue. A *T2-weighted sequence,* which emphasizes the T2 properties of tissue, requires a long TR (1500 to 3000 ms) and a long TE (60 to 120 ms). The signal intensity on T2-weighted images is related to the state of hydration of the tissue. Any tissue rich in free or extracellular water (e.g., cerebrospinal fluid, cysts, necrotic tissue, fluid collections, intervertebral discs, and neoplasms) will demonstrate increased signal intensity on T2-weighted sequences. Mineral-rich tissue (e.g., bone) contains few mobile protons and consequently demonstrates very low signal intensity on all pulse sequences. Gas, containing no mobile hydrogen ions, generates no MR signal.

In addition to signal intensity, tissue and organ configuration must be evaluated to detect pathologic changes. Spatial resolution, the ability to delineate fine detail, is determined by slice thickness, field of view (FOV), and the size of the acquisition and display matrices. Ideally, when imaging small structures, thin sections with a large matrix (256 × 256 or 512 × 512) should be utilized, but MRI, like CT, is affected by signal-to-noise constraints, and image degradation may result from low signal-to-noise ratios. Improved spatial resolution on MRI evaluations can be achieved by using surface coils, with their higher signal-to-noise ratio, but at the cost of a smaller field of view.

As in all imaging procedures, artifacts are a source of image degradation in MRI studies, resulting in significant loss of diagnostic information. Motion artifacts are the most common cause of image degradation. In CT studies patient motion results in the degradation of a single image, but movement during MRI scanning will cause degradation of all images in a sequence. To decrease scan time, new fast-scanning methods have been developed (e.g., fast spin-echo imaging and gradient echo imaging). With gradient echo imaging, gradient reversal is used to restore the transverse magnetization vector in order to generate an MR signal, instead of using an additional radio frequency (RF) pulse, which is utilized with spin-echo imaging. There is a wide range of potential image contrast using gradient echo imaging by manipulating its reception time (TR), echo time (TE), and flip angle. The contrast obtained with gradient echo imaging is referred to as T2* (T2 star) and is different from the standard T2 contrast obtained in spin-echo sequences. Potential degradation of gradient echo imaging due to magnetic field inhomogeneities is greater than with spin-echo imaging. The information obtained from gradient echo sequences is different from standard T1- and T2-weighted sequences, and it cannot be considered a simple replacement for a standard spin-echo sequence.

Standard MRI studies of the spine include sagittal spin-echo T1- and T2-weighted sequences along with a spin-echo T1-weighted axial sequence (Fig. 10.61). Gradient echo T2* axial and sagittal sequences are also frequently obtained. In the cervical spine thinner sections are needed compared to lumbar spine because of the smaller size of anatomic structures. The strength of MRI resides in its excellent soft tissue contrast, direct multiplanar imaging, and absence of ionizing radiation. The major contraindication to an MRI study is the presence of any electrical device in the body (e.g., a cardiac pacemaker or medication pump), brain aneurysm clips, some cochlear and ocular implants, some vascular filters, and metallic fragments in the eye or spinal canal. Patients with claustrophobia may have difficulty with the performance of the exam, but they usually can complete the study if they receive information about the study before undergoing the exam. Medications can also be provided to relieve anxiety, if needed. For evaluation of the traumatized patient, MRI-compatible spine stabilizers and support equipment are now available.

The most common clinical conditions involving the spinal column that require diagnostic evaluation are degenerative spinal disease (which includes disc and facet degeneration along with spinal stenosis), postoperative disorders, spinal trauma, metastatic disease, spondyloarthropathies, and spinal infection.

Degenerative Disc Disease

When trying to understand the dynamic changes that are identified in the degenerating spine, it is helpful to think of each disc level in the spine as a motion segment or a functional unit comprising the discovertebral joint and the two facet joints. It is important to evaluate all components of this functional unit with imaging studies and not merely to focus on isolated pathologic changes (e.g., disc herniation).

For the evaluation of disc degeneration, plain films are of limited value. Decreased disc height, bony sclerosis, gas or calcification within the disc space, and end plate hyperostosis are associated with degenerative changes of the disc, but these findings are of little predictive value in determining the cause of spinal or radicular pain. For the evaluation of disc disease, both MRI and CT provide excellent delineation of disc herniation. The major difference between the imaging techniques is that MRI can detect pathoanatomic and chemical changes within the disc prior to changes in disc contour. On an MRI spin-echo T2-weighted sequence, the signal intensity of the disc is related to the state of hydration of the nucleus pulposus and the inner anular fibers.

With aging and degeneration comes a gradual desiccation of the mucoid nuclear material and transformation of the disc into a more solid fibrocartilaginous structure. With desiccation and degeneration of the disc comes a loss of the high signal intensity in the disc on the T2-weighted imaging. The development of radial anular tear is probably the necessary step in the devel-

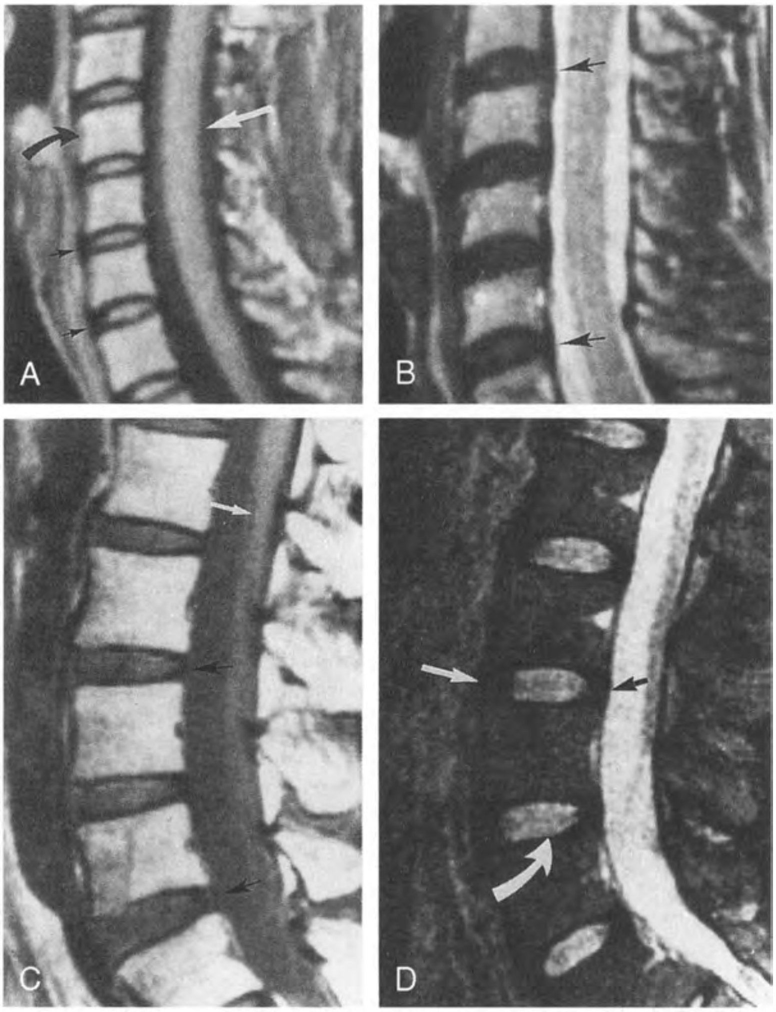


Figure 10.61. Normal spine anatomy—magnetic resonance imaging. **A.** On the sagittal T1-weighted image of the cervical spine, a demonstration of the cervical spinal cord (*straight white arrow*), the vertebral bodies (*curved black arrow*), and the discovertebral joint (*short black arrows*). **B.** On the sagittal T2-weighted image, high signal intensity is seen within the cerebrospinal fluid surrounding the spinal cord, which results in excellent delineation of its margins and optimal evaluation of the posterior margin of the discovertebral joints (*black arrows*). **C.** On the sagittal T1-weighted image of the lumbar spine, excellent delineation of the conus medullaris (*white arrow*). The intervertebral disc space is well delineated, but the posterior margin of the disc is not well defined because of the similar signal intensity of the posterior outer anular fibers and the adjacent cerebrospinal fluid (*black arrows*). **D.** On the sagittal T2-weighted image, there is increased signal intensity within the cerebrospinal fluid and excellent delineation of the posterior margin of the disc (*black arrow*). An increased signal intensity is seen within the central portion of the disc (*curved white arrow*), which represents a combination of the nucleus pulposus and the inner anular fibers. The anterior anular fibers (*straight white arrow*) are also delineated. (Reprinted with permission from Herzog R. Radiologic imaging of the spine. In: Weinstein JN, Rydevik BL, Sonntag VKH, eds. Essentials of the Spine. New York: Raven Press, 1995;7.)

opment of a disc herniation. With MRI, it is possible to delineate these tears before the displacement of nuclear material (i.e., disc herniation).

Displacement of nuclear material into the region of the outer anular fibers will cause a focal contour abnormality of the disc (i.e., a *disc protrusion*). As long as the disc material is contained by the outer anulus or the posterior longitudinal ligament, it is considered a contained herniation. If it penetrates the outer anular-posterior longitudinal ligament complex, it is called a "disc extrusion" (Fig. 10.62). If the disc material separates from its disc or origin, it is called a "sequestered fragment." This fragment can migrate cranial or caudal to disc space.

Both CT and MRI are excellent techniques to detect and characterize disc herniations. After a disc has herniated, the disc material within the disc space will continue to degenerate, and on an MRI study the degenerated disc will demonstrate low signal intensity on spin-echo T2-weighted images. Within the

degenerated disc may be found fluid-filled fissures and granulation tissue, which are detected as foci of high signal intensity on T2-weighted images. This should not be confused with an inflammatory process.

End plate degeneration is frequently associated with disc degeneration. These changes can be detected on the MRI study as areas of abnormal signal intensity in the subchondral bone; these areas reflect the presence of fibrovascular tissue or fatty infiltration. Bony sclerosis or proliferation can be detected with CT or plain films.

With the increased use of MRI, it has become clear that degenerative disc disease is a process that often begins in the second or third decade and progresses as an individual ages. Evidence of a disc herniation or a disc degeneration is frequently identified on imaging studies, in both symptomatic and asymptomatic individuals. The significance of these findings can be determined only by precise correlation to the clinical findings.

Accompanying disc degeneration is alteration of the biomechanical status of the functional unit, which may precipitate degenerative changes in the facet joints. Degenerative changes of the facet joints include cartilage erosion, subchondral cysts, bony sclerosis, and osteophyte formation. Both CT and MRI can detect these degenerative changes, but only MRI can delineate the articular cartilage changes and demonstrate joint effusions. Plain films are much less sensitive in detecting early degenerative changes of the facet joints.

Unrecognized Radiographic Changes Seen on MRI

Plain x-ray study shows bilateral pseudosacralization of the fifth lumbar transverse processes in Figure 10.63 (*arrows*). Figure 10.64 is the lateral view showing the rudimentary disc at L5-S1 (bottom *arrowhead*) with hemispheric spondylosclerosis of the anterior superior plate and body of L4 (top *arrowhead*). Note the L1 inferior vertebral body plate appears to show a minimal vertebral plate defect (*arrow*). Figure 10.65 is a T1-weighted sagittal image showing a large nuclear disc invagination into the inferior anterior plate of L1 (*arrow*), which is not appreciated on the plain lateral view in Figure 10.64. Also note the inferior plate and cancellous bone type I degenerative changes of L3 that are not appreciated on plain x-ray film (*arrowhead*).

Source of Pain As Determined by MRI

White bulged and white flat discs have a 90% chance of having no provocative pain with discography and a 95% chance of negative discography, and therefore a strong negative correlation with discogenic pain is suggested (88).

Hyperintense Discs on T1-weighted MRI

Potential causes of T1-weighted increased signal intensity are hemorrhage, abscess, or rare disease (ochronosis, homocystinuria, and so on). Degenerative disc changes should be considered the cause of abnormally increased signal intensity in intervertebral discs on T1-weighted MRIs. In most patients, no clinical significance should be attributed to this finding (89). Hyperintense discs are suggestive of degenerative disc disease (90).

Osteophytes are associated with disc bulging in the middle part of the spine and with end plate irregularities in the lower part of the lumbar spine (91).

GADOLINIUM-ENHANCED MRI ADVANTAGES IN DIAGNOSIS

Gadolinium (Gd-DTPA) enhancement allows differentiation of scar tissue from recurrent disc herniation. Epidural scar tissue is vascular and is enhanced by the administration of intravenous contrast Gd-DTPA. Enhancement of epidural fibrotic tissue occurs on early images taken 6 to 10 minutes after contrast administration. Intervertebral disc material does not enhance on early images, thus allowing differentiation of fibrotic vascular scar tissue from recurrent herniated disc material (92, 93).

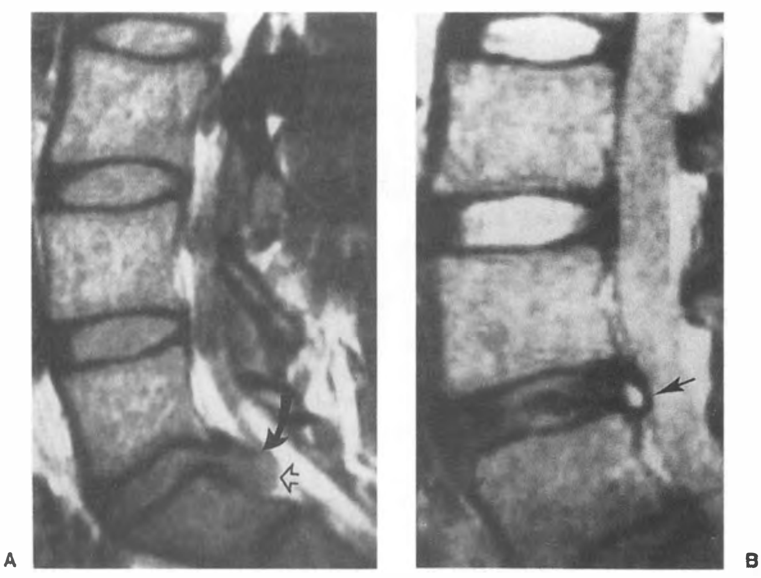


Figure 10.62. Lumbar spine disc extrusion and protrusion. **A.** At the L5–S1 disc level, on the sagittal proton-density weighted image is seen a posterior disc extrusion (*open black arrow*) that has penetrated through the posterior outer anular-posterior longitudinal ligament complex (*curved black arrow*). **B.** In another patient, the sagittal T2-weighted image shows a posterior disc protrusion (*arrow*) contained by the posterior outer anular-posterior longitudinal ligament complex. (Reprinted with permission from Herzog R. Radiologic imaging of the Spine. In: Weinstein JN, Rydevik BL, Sonntag VKH, eds. Essentials of the Spine. New York: Raven Press, 1995;7.)

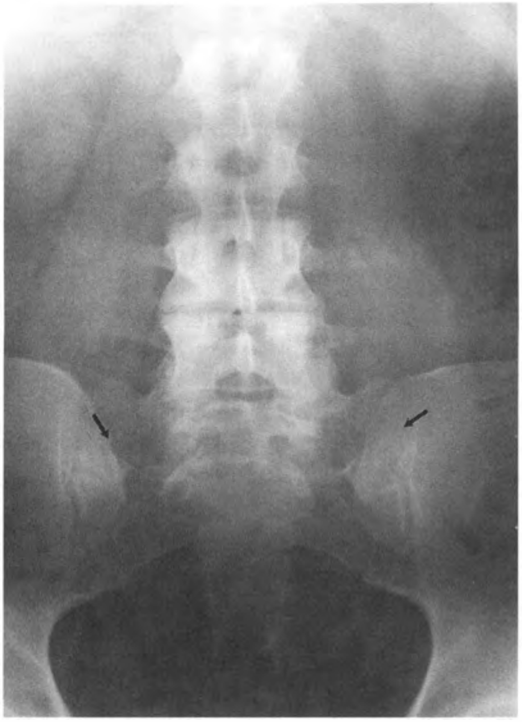


Figure 10.63. Bilateral pseudosacralization of L5 on the sacrum (*arrows*).

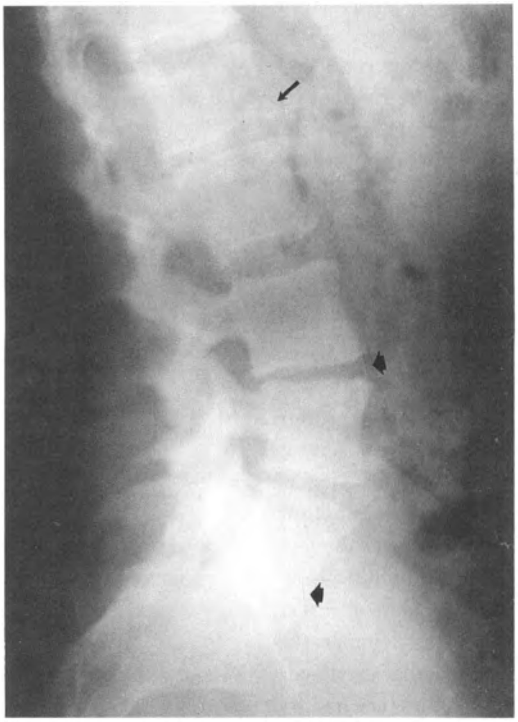


Figure 10.64. The rudimentary L5–S1 disc is seen (bottom *arrowhead*) with L4 superior plate and body hemispherical spondylosclerosis (top *arrowhead*). An inferior L1 plate defect is suggested (*arrow*).

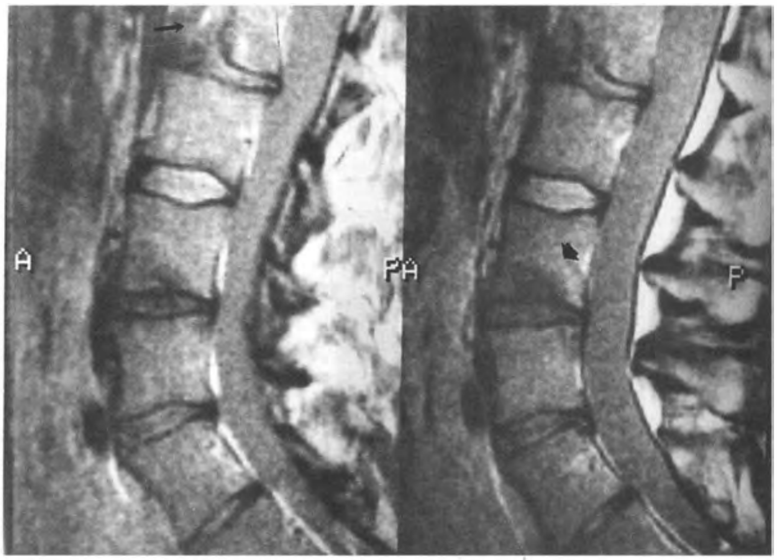


Figure 10.65. T1-weighted sagittal magnetic resonance image shows type I L3 inferior plate degenerative changes (*arrowhead*) and an inferior L1 anterior inferior plate nuclear invagination Schmorl node that is not appreciated on plain x-ray film (*arrow*). The L3 body plate change is not appreciated on plain x-ray film.

Contrast-enhanced MRI used in patients within the first 6 to 8 weeks after surgery can produce misleading images. Hematoma and postsurgical changes surrounding the thecal sac during this postoperative period can mimic signal changes of a true disc herniation (94).

An example of MRI-enhanced scar tissue and disc herniation differentiation is shown in the following case in which a patient with three prior back surgeries had total relief with distraction adjustment.

Case 5

A 43-year-old airline pilot was seen complaining of low back pain and left lower extremity pain after falling down the stairs. Three prior lumbar disc surgeries, all at the L5–S1 levels, had been performed. Surgery was again recommended to him but he sought chiropractic care first. The MRI study (Fig. 10.66) shows a large free fragment lying within the spinal canal and extending posteriorly behind the sacrum. Figure 10.67 is the precontrast axial T1 image showing the large right central and paracentral mass that compresses the thecal sac and first sacral nerve root (*arrow*). Figure 10.68 is the postcontrast MRI showing enhancement of the right free fragment mass indicating scar tissue fibrosis (*arrow*) whereas a free fragment of disc material is seen lying to the left of the midline (*arrowhead*), which probably is responsible for the left leg dermatome pain.

One week of daily distraction adjustments at the L5–S1 level resulted in complete relief of the low back and left lower extremity pain and the patient returned to work as an airline pilot 3 weeks later.

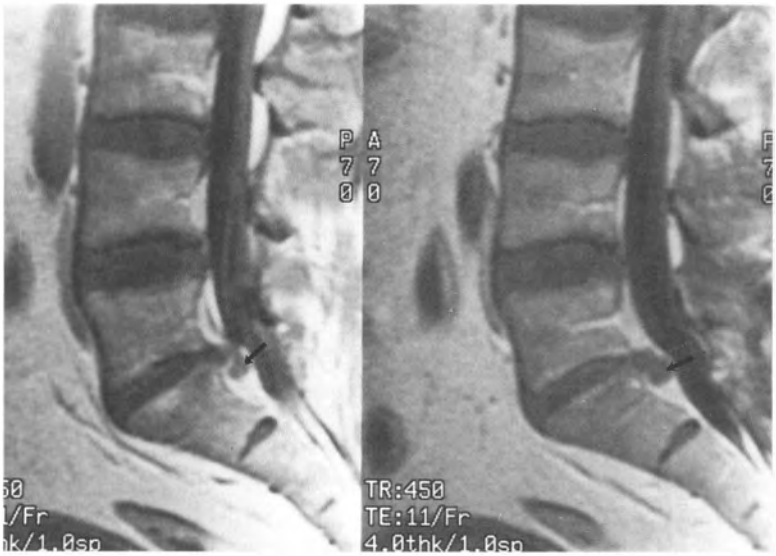


Figure 10.66. Sagittal T1-weighted magnetic resonance image shows the large free fragment lying posterior to the L5–S1 disc space and sacrum (*arrow*).

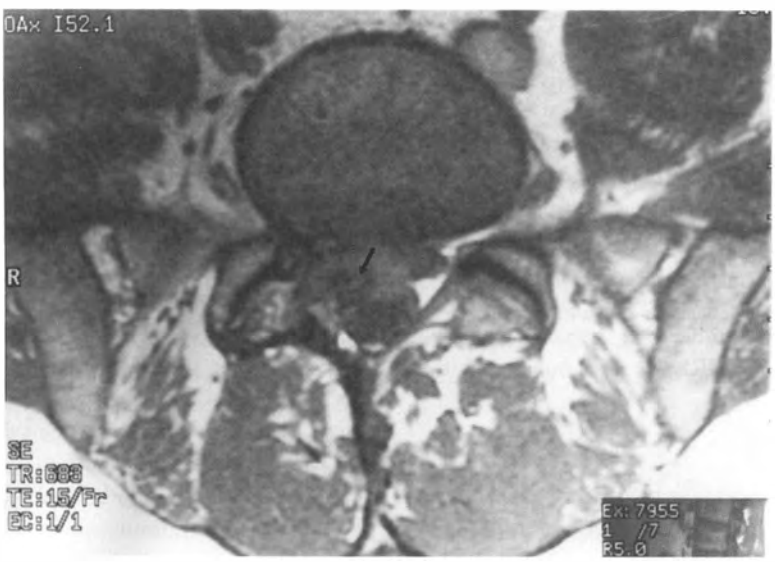


Figure 10.67. Axial precontrast T1-weighted magnetic resonance image shows a large mass lying within the right central and posterolateral vertebral and osseoligamentous canal that could be scar or recurrent disc herniation material (*arrow*).

Figure 10.68. Postcontrast axial magnetic resonance image at the L5–S1 level shows enhancement of the free fragment within the right central and posterolateral vertebral canal (*arrow*) indicating scar tissue, whereas an area of disc density (*arrowhead*) lies within the left posterior midline and displaces the thecal sac and contacts the left S1 nerve root.

Gadolinium-Enhanced MRI Shows Anular Tears

Anular tears have been noted to be enhanced on T1-weighted images after the administration of gadolinium. This is presumably secondary to the ingrowth of scar tissue into the tear, a consequence of the body's attempt at healing. The failed back surgery syndrome has been reported to occur in 10 to 40% of patients, and the causative factors include new or recurrent disc herniation, stenosis, arachnoiditis, and epidural scar (95).

Symptomatic Nerve Root Identified with Gadolinium MRI

Magnetic resonance imaging with gadolinium shows enhancement of the symptomatic nerve root in patients with a lumbar disc herniation, and the degree of enhancement reflects the severity of the sciatica. Contrast-enhanced MRI may become a diagnostic tool for detecting affected nerve roots, and it may provide new insights into the pathogenesis of sciatica (96, 97).

CT Benefits Over MRI

Foraminal or extraforaminal lumbar disc herniation are not well seen on myelography and MRI, whereas high-resolution CT demonstrates them best (98).

Contrast CT Recommended

Noncontrast CT does not visualize the subarachnoid space, and therefore cannot diagnose cauda equina tumors and other intradural lesions that can mimic lumbar disc herniations. A "negative" noncontrast CT does not eliminate the need for MRI or myelogram-CT in the patient with unexplained acute low-back pain with or without neurologic symptoms and signs (99).

CONTAINED (PROTRUDED) AND NONCONTAINED (PROLAPSED) DISCS

Definitions and Principles

The change within the nucleus pulposus when it escaped the confines of the anulus is classified as either a protrusion or prolapse. Protrusion of nuclear material occurs when the protruding nucleus is contiguous with the remaining nucleus and the anulus fibrosus is stretched, thinned, and under pressure. The protrusion can cause only back pain if the outer nerve-innervated anulus is irritated, or it can cause both back and leg pain if the anulus bulge contacts the dural lined nerve root within the lateral recess of the vertebral column. The pressure within the nucleus is 30 psi (100), and this pressure was found to be 30% less in the standing position than in the sitting position, with 50% less pressure in the reclining position than in the sitting position (101). The cerebrospinal fluid pressure is 100 mm of water in the recumbent posture and 400 mm in the sitting posture (102), which is important in treating the disc lesion, as sitting is to be avoided. An epidemiologic study (103) demonstrated that suburban dwellers who drive to work have twice the incidence of severe back pain than do those who do not drive, and that those workers who drive during most of their working day (e.g., truck drivers) have three times the incidence.

Fahrni (104) surveyed a jungle people in India who squat rather than sit and found that they had a zero incidence of back pain and a greatly diminished incidence of disc degeneration on x-ray film.

Gresham and Miller (105) carried out postmortem discograms on 63 fresh autopsies; these patients who came to autopsy were between 14 and 80 years of age and had had rela-

tively asymptomatic backs. All of the specimens that came from patients between 46 and 59 years of age revealed evidence of disc degeneration at L5–S1.

Prolapse exists when the extruded nucleus loses continuity with the remaining nuclear material and forms a free fragment, or what in Europe is termed a "sequestered disc fragment," within the spinal canal. Arns et al. (106) state that the first stage of a disc lesion is nuclear bulge, which causes lumbago and symptoms of Déjérine's triad. The second stage is the onset of sciatica as the nuclear bulge contacts the nerve root, and the third and final stage is prolapse.

Opinions to the efficacy of myelography, electromyography, and discography in the diagnosis of disc protrusions are varied. Semmes (107) states that because nearly one third of myelograms are not definitive or are misleading, the history and clinical findings prove more reliable. He states that myelography is used too frequently for diagnosis and as an indication for surgery, and that he has used it in less than 3% of his last 350 surgeries. In the Scandinavian countries, oil-based media has been banned for use in myelography because of the risk of arachnoiditis (103). Herlin states that myelography is not a sufficiently reliable method of investigation in the diagnosis of sciatica (108), and has used myelography in only 10% of his cases (108).

Extruded Disc Shows Leg Pain Alone or As Major Complaint

To determine whether the presence of an extruded lumbar disc prolapse could be predicted from clinical symptoms, the relative proportions of back and leg pain in 100 prospective discectomy patients was observed. Of 27 patients who presented with leg pain, only 26 (96%) were found subsequently to have an extruded disc fragment. *Patients with leg pain only and those with a marked predominance of leg pain over back pain have a high probability of harboring an extruded disc fragment* (109).

Pelvic Disease and Disc Compression of Nerve Roots

A connection between lumbar disc degeneration and pelvic disease has been documented by Herlin. Such a connection had been suspected for years, and painful and chronic infectious conditions of the urogenital organs have been associated with compression of one or several of the lower sacral nerve roots. He further states that endometriosis sometimes is combined with sciatica, and that it seems justifiable to investigate the relationship between sacral nerve root compression and the development of endometriosis. He believes that, in males, lower sacral nerve root compression leads to the development of endometriosis; and he feels that it ought to be considered as a cause of chronic prostatovesiculitis (108). He has documented that, in one patient, two miscarriages were caused by sacral nerve root compression, which subsequently caused most of the patient's sciatica (108). In another patient, he thought there was a probable connection between chronic urogenital infection caused by disc compression of sacral nerve roots and rheumatoid arthritis (108). Herlin has also documented a connection between sacral nerve root compression and chronic prostatitis. He also believes that, although no definite proof exists, the possibility of sacral nerve root compression as a cause of sterility must be considered. He presented a case of pain originating bilaterally in the medial region of the gluteal muscles and radiating into the minor pudendal labiae and clitoris, with a decrease in the duration of orgasm intensity. Following surgery for the removal of a medial fifth lumbar disc lesion, the patient's sexual function normalized within 2 months (108).

Some (110, 111) believe that disc disease should be ruled out in young and middle-aged patients who develop problems of urinary retention, vesicle irritability, or incontinence. Amelar and Dubin (112), however, link lumbar disc disorders with sexual impotence and bladder function disturbances through organic parasympathetic involvement rather than psychological causes.

Pudendal Plexus

Understanding the neurovisceral connection between a disc lesion and disease of the pelvic organs requires understanding the pudendal plexus. The pudendal plexus is formed from the second, third, and fourth sacral nerves and is the innervation of certain pelvic organs (113). Parasympathetic fibers innervate the urinary bladder, prostate gland, and seminal vesicles. The uterus and external genitalia also are innervated by nerve fibers from this plexus, and the alimentary tract is controlled by the pudendal plexus as well. The pudendal nerve, a branch of the pudendal plexus, gives rise to the inferior hemorrhoidal nerve, the perineal nerve to the transversus perinei profundus, the sphincter urethrae membranacea, bulbocavernosus, ischiocavernosus, transversus perinei superficialis, the corpus cavernosum urethrae, the urethra, the mucous membrane of the urethra, the urogenital diaphragm, and a scrotal branch to the scrotum and labiae. Another branch of the pudendal nerve, the dorsal nerve of the penis, innervates the urogenital diaphragm, the corpus cavernosum penis, and dorsum of the penis ending in the glans. The clitoris is innervated similarly.

Neuroanatomically, pressure on the sacral nerve roots by a disc lesion can create an aberrant nerve supply to the organs described and resultant disease. On this neurologic basis is seen the reason many authorities feel that a disc lesion should be considered in the cause of any condition of the urogenital or reproductive system.

Occurrence and Onset of Back and Leg Pain

The onset of sciatica or back pain represents a starting point for diagnosis. It is possible for a disc to protrude and contact a nerve root, resulting in the sudden onset of sciatica without accompanying back pain. This protrusion can result in isolated pain in an area of specific nerve innervation such as the heel,

calf, great toe, or posterior thigh. Back pain preceding sciatica indicates irritation of the anulus fibrosus, ligaments, and dura mater innervated by the recurrent meningeal nerve prior to contact with the involved nerve root. The sudden onset of leg pain without back pain indicates disc extrusion (prolapse) (19).

A differential diagnosis between protrusion and prolapse may include the findings shown in Table 10.3.

The cauda equina symptoms caused by large midline disc protrusions contacting several roots of the cauda equina present a particular problem in diagnosis. Difficulty with urination, incontinence, rectal difficulties, difficulty in walking, or symptoms of abdominal viscera are indicative of the diagnosis of a large midline disc protrusion. These represent true surgical emergencies and must be handled as such.

Delay in the onset of pain in disc injuries can be the key to diagnosis. The spinal cartilage has a poor blood supply and reacts slowly to injuries. Therefore, it may be 2 or 3 days after injury before the oozing of the nuclear material and the slow swelling of the disc result in the pain that follows an injury and protrusion of a disc (114). Table 10.4 contains specific diagnostic criteria of disc lesions.

Table 10.3

Clinical Differentiation Findings in Protrusion and Prolapse

Differential Diagnosis	Protrusion	Prolapse
Pain on compression and distraction	Yes, usually	Not as frequently
Flexion and extension	Yes	Only on flexion
Cough, sneeze, and strain	Yes	Not always
Onset of pain	Gradual	Sudden, intense

Table 10.4

Specific Diagnostic Criteria of Disc Lesions[a]

L3–L4 Disc Protrusion (L4 Nerve Root Compression Findings

Weakness of the quadriceps muscle (Fig. 10.69); diminished or absent patellar reflex (Fig. 10.70)

The test for the straight leg raising sign may be negative in lesions of the L3–L4 disc; pinwheel examination may reveal hyperesthesia or hypoesthesia of the L4 dermatome

L4–L5 Disc Protrusion (L5 Nerve Root Compression) Findings

Weakness of tibialis anterior muscle, extensor digitorum, and hallucis longus muscles (Fig. 10.71)

Weakness of the extensor hallucis muscle (Fig. 10.72)

Weakness of the peroneus longus and brevis muscles; weakness in these muscles also occurs when an L5–S1 disc protrusion compresses the S1 nerve root (Fig. 10.73)

Dysesthesia of the L5 dermatome is determined by simultaneous testing of the sensation of the extremities (Fig. 10.74)

Foot and great toe dorsiflexion (ankle eversion) strengths depend on the nerve supply of the peroneal nerve to the anterior tibialis and extensor muscles; the SLR will be positive in proportion to nerve compression by the disc

L5–S1 Disc Protrusion (S1 Nerve Root Compression) Findings

Several muscles are tested for L5–S1 compression of the first and second sacral nerve roots

Weakness of the biceps femoris, semimembranosus, or semitendinosus muscles (Fig. 10.75)

Weakness of the gluteus maximus is found by comparison of contralateral sides; the opposite pelvis should be stabilized while the thigh on the side to be tested is compressed (Fig. 10.76); the gluteus maximus muscle is innervated by the inferior gluteal nerve whose origin is in the roots of L5–S1–S2

The gluteal skyline sign was present in 60% of patients with disc lesions of the lower lumbar spine; this sign is second only to the straight leg raising sign in frequency and was the only finding except for pain in 13% of the patients with disc protrusion (115); the patient is asked to contract his buttocks; flaccidity is found on the side of the disc protrusion (Fig. 10.77)

Diminished or absent ankle jerks (Achilles reflexes) may be noted (Fig. 10.78)

Weakness of the calf muscles (Fig. 10.79)

Weakness of the flexor muscle of the great toe (Fig 10.80)

Dysesthesia of the S1 dermatome by comparing the sensation of each extremity simultaneously (Fig. 10.81)

The SLR will be positive, the severity depending on the pressure of the disc bulge or protrusion on the compressed nerve root

[a]It must be stated that these tests are strong indicators for disc level involvement, but there is some overlap of innervation to those muscles supplied by all three nerve roots—L4, L5, and S1.

Figure 10.69. Quadriceps muscle testing.

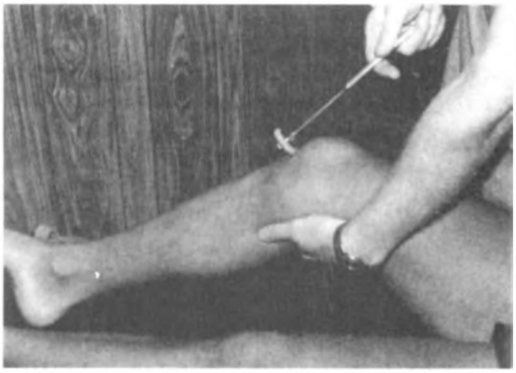


Figure 10.70. Patellar reflex testing.

Figure 10.71. Dorsiflexion (ankle eversion) of the foot.

Figure 10.72. Dorsiflexion of the great toe.

Figure 10.73. Eversion of the foot.

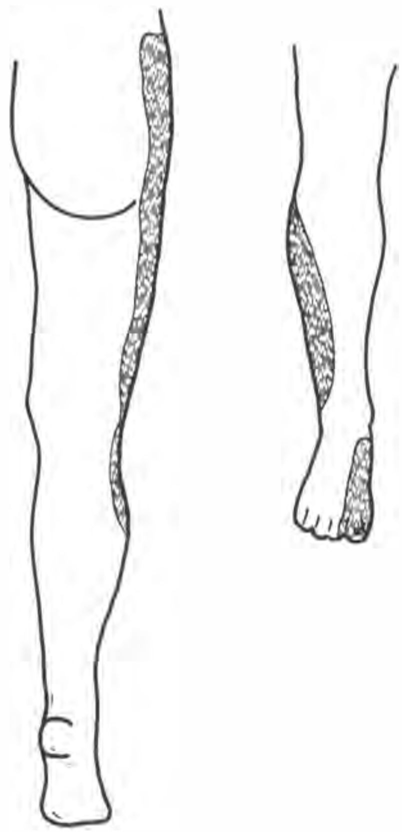


Figure 10.74. Dysesthesia and pain distribution of the L5 dermatome.

Figure 10.75. Hamstring muscle strength testing.

Figure 10.76. Gluteus maximus muscle testing.

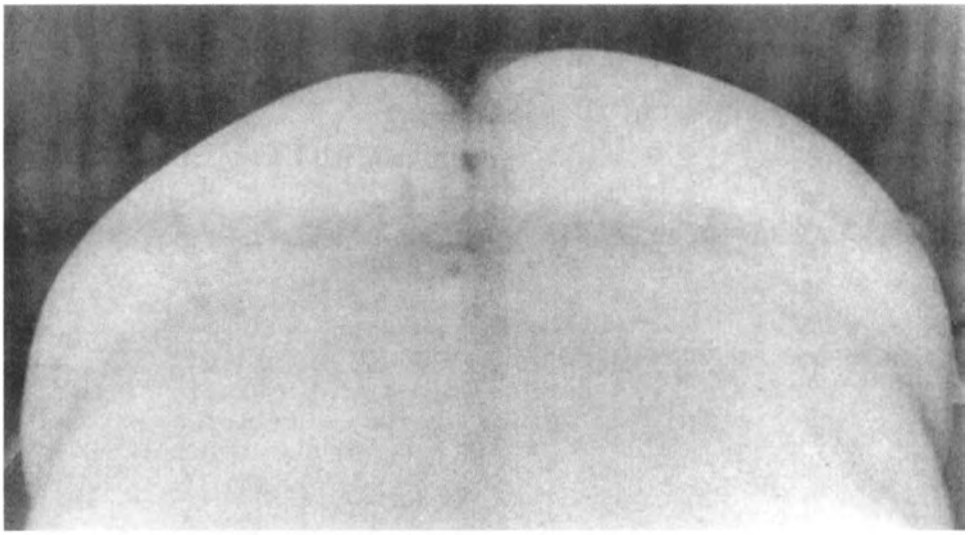


Figure 10.77. Gluteal skyline sign in a 36-year-old man with a history of 3.5 months of right low back and first sacral nerve root sciatica. The ankle jerk reflex is absent, and a marked loss of the gluteus maximus muscle tone is seen, as noted by the flattened contour of the right gluteus maximus muscle. The computed tomography scan and myelogram were positive for a prolapse of the L5–S1 disc on the right. Surgery was necessary to remove the fragment.

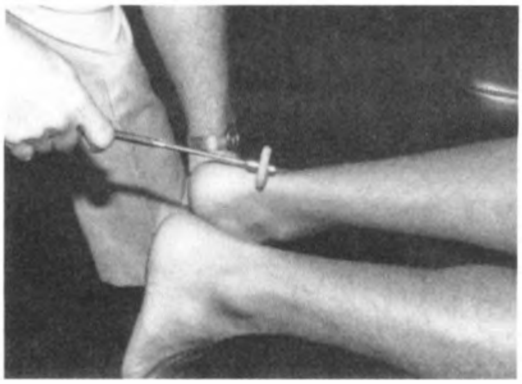


Figure 10.78. Ankle jerk testing.

Figure 10.79. Plantar flexion of foot.

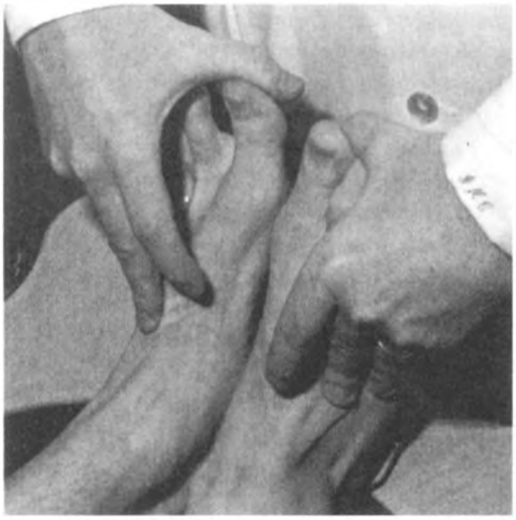


Figure 10.80. Plantar flexion of the great toe.

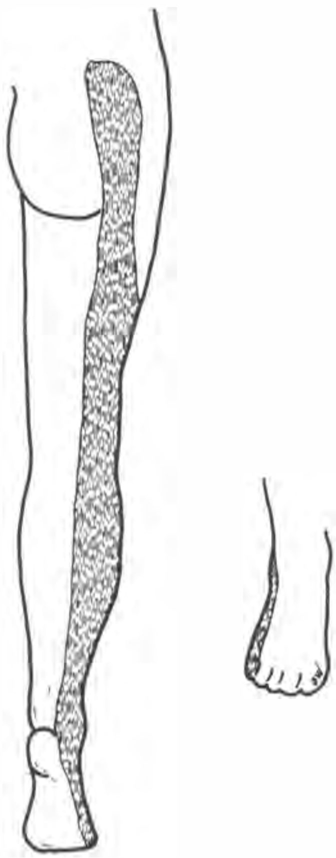


Figure 10.81. Dysesthesia and pain distribution of the S1 dermatome.

Motor Changes in Discal Lesions

Motor changes in these radicular compressions requires some attention, because they represent perhaps the most serious side effects of disc protrusion. Disc lesions can cripple, and motor changes are the most serious side effects for the patient and the most serious potential medicolegal problems for the physician, whether the approach be conservative or surgical.

Occasionally, muscle weakness caused by neurapraxia or degeneration can be present with little or no pain. Of course, muscle weakness usually follows sensory changes of the lower extremity. Nevertheless, regardless of whether a patient complains of low back pain, leg pain, or an inability to walk on the toes or heels the clinician must always do kinesiologic muscle testing.

Depending on the muscle involved, patients may complain of falling, having equilibrium problems (which really means they tend to limp because of weak muscles), or having the knee "give out" under them. The patient may present with gait changes, such as limping because of calf muscle weakness and inability to lift the heel, or "stubbing" the great toe on carpet or steps because of weak anterior tibialis muscles or peroneal muscles. The patients may walk with the knee flexed to prevent "stretching" of the swollen or inflamed sciatic nerve—a "walking" Néri bowing sign.

Validity of Determination of L4, L5, S1 Dermatome Innervation

L5 and S1 Dermatome Mapping

In 75% of 31 L5 nerve root blocks, the region of superimposition extended from the midline of the trunk posteriorly, across the buttock, through the lateral side of the thigh, the lateral side of the leg, and the medial side of the dorsum of the foot to the first digit.

In S1 blocks, the region of at least 75% of 20 patients showed superimposition extended from the midline of the trunk posteriorly, across the buttock, through the posterior, lateral aspect of the thigh and leg, to the fifth digit of the foot (116). Dermatomes are good diagnostic tools to diagnose the level of disc herniation (117).

Pain drawing is a simple yet powerful diagnostic tool to identify the level of disc herniation. Sixty-eight percent of L4–L5 disc herniation patients exhibit anterolateral leg pain compared with only 23% of L5–S1 disc patients. Seventy nine percent of the patients with complete L4–L5 hernias showed marked anterior leg pain. Seventy five percent of L5–S1 disc herniation patients and 85% of the patients with complete L5–S1 hernias showed marked posterior foot pain. Bilateral back pain suggested protruding hernia, and pain radiating to the foot suggested sequestrated hernia (118).

Root Stimulation Better Than Electromyography (EMG) to Localize Root Involvement

Needle electrical stimulation of the lumbosacral roots at the laminar level of the T12–L1 or L1–L2 intervertebral spaces was compared with conventional needle EMG. Lumbar electrical stimulation showed root abnormalities objectively in 80% of patients whereas the diagnostic value of needle EMG was 65%. Therefore, electrical root stimulation is superior to routine EMG for localizing lumbar root involvement (119). Ninety percent of patients with disc protrusion or degeneration will

have L5 or S1 nerve root involvement. Five percent of people have congenital failure of segmentation of these nerve roots, which can present problems in localization of the involved nerve root. Electronic stimulation of 50 patients' L5 and S1 nerve roots proved that segmental innervation is essentially reliable in identifying the dominant nerve root. Sixteen percent of these patients exhibited significant departure from the usual dermatome innervation (120).

First sacral nerve projection pain is most common. Clinical test results in 403 patients were compared with myelographic and operative findings to determine the accuracy of the clinical examination in diagnosing the involved disc and nerve root (121). L5 dermatome involvement had the same accuracy for localizing an L4–L5 disc lesion as myelography, 80% of the time finding an L5 dermatome distribution caused by an L4–L5 disc lesion. S1 nerve root involvement was caused by an L4–L5 disc 34% of the time and an L5–S1 disc 63% of the time. Associated L5 and S1 dermatome pain was found in L4–L5 disc involvement 75% of the time.

Kortelainen et al. (121) concluded that, in single nerve root involvement with motor, reflex, and sensory disturbances, the accuracy of clinical investigation was equal to that of myelography. With two nerve roots involved, clinical diagnosis is not completely reliable in lower lumbar herniated discs, necessitating CT or EMG prior to surgery to avoid unnecessary explorations.

Anomalous Nerve Root Anastomosis

Some patients suffering from lumbar disc herniations do not manifest the typical clinical symptoms expected for anomalous nerve anastomosis because 30% of 60 fresh cadaver studies showed intradural and extradural anastomosis and divisions of nerve roots between L4 to sacral nerve roots (127). This could account for the dual dermatome sensation in these patients.

Initial Treatment Based on Clinical Investigative Impression

In our diagnostic approach to the intervertebral disc lesion, we rely on the clinical workup for an impression of the disc level and type. If the patient is not at least 50% improved within 3 to 4 weeks of manipulative care based on this impression, more invasive testing (e.g., MRI, CT, EMG, or perhaps myelography) is ordered. Of course, if serious findings (e.g., cauda equina signs), increased motor weakness, or unbearable pain sets in, we move quickly to the more definitive diagnostic imaging modalities. However, based on our clinical impression, we successfully relieve well over 90% of our low back pain patients and avoid the more invasive and costly imaging modalities.

Schoedinger (123) states that a detailed history and physical examination, in combination with a positive diagnostic imaging tool such as CT or myelography, are sufficient to establish the diagnosis of disc rupture.

Semmes (107) states that the clinical findings and history are more reliable than myelograms, because nearly one third of myelograms are uncertain or misleading. He even stated that myelography was wholly unnecessary in the diagnosis of the average patient requiring surgery for a ruptured disc. I would assume that his concept would be altered by modern imaging modalities such as CT and MRI.

Gainer and Nugent (124) stated that lumbar disc herniation is one of the most common causes of back pain and leg pain, and it is usually easily diagnosed by a history and physical examination.

Accuracy in Diagnosing Disc Lesion from Clinical Findings

A 63% correlation was found between clinical neurologic signs and operatively proved pathology (125). Furthermore, a 55% correlation was reportedly found between neurologic signs and a herniated lumbar disc; a positive straight leg raising sign and positive neurologic signs produced the correct diagnosis in 86% of patients (125). The same positive neurologic examination and positive SLR, coupled with a positive myelogram, increased the accuracy to 95%. I would suggest that the 86% accuracy, in the absence of cauda equina syndrome signs or worsening motor deficit, is strong enough clinical indication to justify 3 weeks of conservative care before using the more invasive and institutionally necessitated CT or MRI.

S2 and S3 Nerve Root Compression Signs

White and Leslie (126) reported that the posterior two thirds of the scrotum is supplied by the second and third sacral nerves. They stress the value of examination of the lumbar spine in cases of unexplained scrotal pain.

Summary of Diagnosis of Disc Lesions

Steps in diagnosis of disc lesions are as follows:

1. Note the specific distribution of pain into the lower extremity and whether it involves the L4, L5, or S1 nerve root.
2. Note whether there is any lean of the lumbar spine.
3. Do x-ray studies reveal any right or left lateral flexion of the vertebrae at the level of disc involvement ascertained from dermatome evaluation? That is, if an L5 dermatome sensitivity is found, does the L4 vertebra have a right or left lateral flexion subluxation? If it is the S1 dermatome, does the L5 vertebra have a left or right lateral flexion?
4. Correlate the findings from above to differentiate protrusion from prolapse. Statistically, prolapses are much more difficult to treat than are protrusions.
5. Correlate the SLR sign with a medial or lateral disc. That is, is it positive on the side of sciatica, indicating lateral disc or medial disc, or is it positive on the well leg raising sign, indicating a medial disc on the side of sciatica?
6. Investigate the site of original pain (e.g., back or leg) to rule out tumor, infection, or organic disease as a probable cause. Refer to Table 10.5 for information in making the differential diagnosis between a tumor and a disc lesion.

7. If a disc involvement truly seems probable, after the site has been determined to be either medial or lateral, explain to the patient that manipulative therapy may not be adequate and that surgical intervention may be necessary.

EXAMINATION

Gleis and Johnson (122) recommend a preprinted pro forma examination form for recording the findings of a physical examination for lumbar pain. They recommend that the examination be done in a logical sequence that helps the examiner reach a working diagnosis and treatment plan. We present such an approach.

Table 10.5

Differential Diagnostic Findings of Discal Versus Tumor Etiology

Differential Diagnosis	Neoplasm	Protrusion
Sitting and standing	No change	Aggravates
Bilateral	Often	Seldom
Night pain	Yes	Less
Character of pain	Unrelenting	Intermittent
Cauda equina symptoms	More	Less
Onset first leg or back pain	Back usually	Either

History

Table 10.6 is the patient pain drawing that is created at the first visit and at subsequent 2-week intervals. Note that the patient's complaints are listed in order of decreasing importance—most prominent symptoms first—and each symptom or complaint is given a visual analogue score. The patient maps out the area of pain by the designated symbols of abnormal sensation. On the 2-week re-examination, the patient will fill out the pain drawing and give new values to the subjective symptoms.

In addition to the visual analogue scale, Oswestry, Roland Morris, and Quebec disability scales are recorded.

Pain Drawings and Nonorganic Signs

A correlation between pain drawings and Waddell's nonorganic physical signs demonstrated that a large proportion of patients with high Waddell scores had nonorganic pain drawings (128). An initial impression diagnosis of psychogenic, benign,

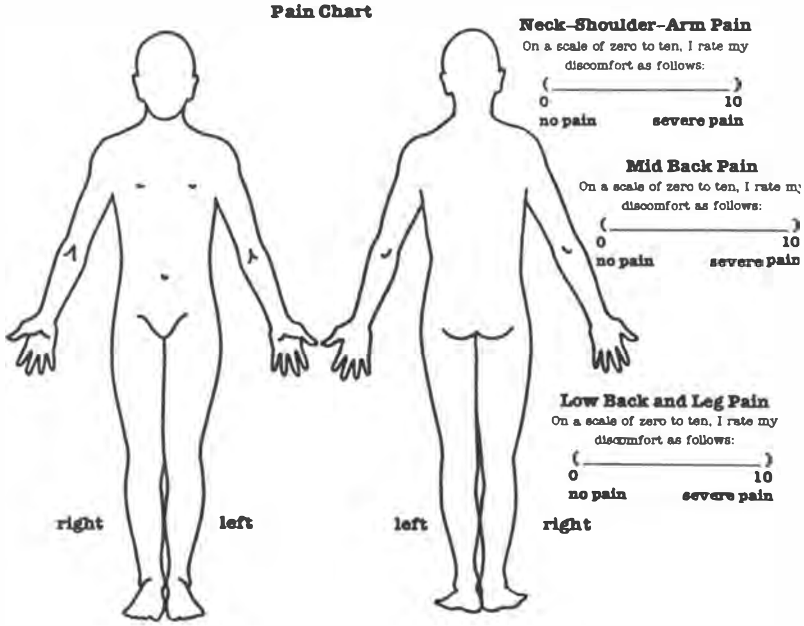
Table 10.6

SHOW AREA(S) OF PAIN OR UNUSUAL FEELING

Mark the areas on this body where you feel the described sensations.
Use the appropriate symbols.
Mark areas of radiation.
Include all affected areas.

Numbness	Pins & Needles	Burning	Aching	Stabbing
- - - - -	00000	xxxxx	*****	/////
- - - - -	00000	xxxxx	*****	/////
- - - - -	00000	xxxxx	*****	/////

Pain Chart



Date: ____________

____
Signature

and herniated disc cases is most consistently done by pain drawings, whereas spinal stenosis and serious underlying disorder drawings produced the most variance in results (129).

The nonorganic physical signs of malingering are pain drawings that show pain over the entire spine and extremities with no definite nerve root distribution of pain. Often the pain markings are off the body parts with written descriptions of the abnormal feelings. (Waddell's nonorganic physical signs are given later in this chapter.) I feel counselors are best called in when dealing with exaggerated symptoms because I lack the expertise to deal with such cases. Please refer to Chapter 16 in this text on the psychology of low back pain for further discussion of pain drawing signs of malingering.

Table 10.7 shows the low back pain examination form that we use. A history of the patient usually is compiled by an assistant. The patient's chief complaint should be recorded exactly as possible (e.g., pain in the low back radiating into the calf of the right leg, or pain in the side of the leg with numbness of the great toe). The history of the complaint should include specific details on how the pain began (i.e., whether the pain in the back started with or without leg pain, or whether the leg pain started sometime after the pain in the back). *A chronologic sequence of back or leg pain from its first incidence in life to the present should be recorded by month and year, including the present symptoms.* Note that on this form the date of pain onset and the date of first examination are requested. These dates allow the doctor to notice the time lapse between the onset of symptoms and the consultation. If this lapse has been long, the patient may have sought other care, and a careful screening of past procedures and diagnosis is necessary.

The history of the patient should include any surgical interventions. Be particularly alert to any disease that could metastasize to the spine and mimic a disc lesion. Any symptoms of gastrointestinal, genitourinary, and menstrual problems should be listed. These allow for documentation of any pudendal plexus symptoms that should be evaluated following the mechanical relief of back pain. Thus, it is possible to evaluate the effects of chiropractic treatment not only on biomechanical faults but also on organic disease.

Family incidence of back pain also is recorded. This record should include whether the father, mother, or siblings have had low back pain, leg pain, or surgery; whether the back pain or leg pain started first or both began simultaneously; whether the pain is aggravated by coughing, sneezing, straining at the stool, bending and lifting, or sitting; and how far down the lower extremity the pain radiates.

Physical Examination

As you proceed through the examination, mark the proper answer on the examination form and keep in mind the findings indicative of intervertebral disc protrusion (contained disc) and prolapse (noncontained disc) as shown in Table 10.8.

Patient Sitting

Minor's sign (Fig. 10.82). Minor's sign is manifest when the patient, in rising from sitting, lifts the body weight with the arms and places the body weight on the unaffected leg. The patient may place the hand on the low back; thus, the painful lower extremity is spared weightbearing.

Bechterew's sign (Fig. 10.83). The test for Bechterew's sign is performed by having the patient extend the knee while in a sitting position. This sitting straight leg raise again stretches the sciatica nerve root and creates either back or leg pain or both if a disc lesion exists.

The SLR sign is a more positive sign of disc lesion in younger people (i.e., under age 40) than it is in older people. This is because, as the intradiscal pressure decreases with age, nucleus turgor lessens, and the nucleus is less likely to compress severely against the nerve root during such maneuvers as SLR, Valsalva, or Bechterew's.

The sitting straight leg raising sign often is positive, whereas the supine recumbent SLR is negative. The reason for this difference is that the higher intradiscal pressure with the patient sitting adds to the nerve root compression; this, when coupled with the stretching of the nerve root during leg raising, creates a much more positive sign of nerve root compression. Fisk (130) states that the hip joint acts as a pulley and tractions the sciatic nerve going from 15° to 30° of leg raising. Between L4 and L5, the L5 nerve root normally moves 2.5 cm during the full range of SLR.

Always perform the straight leg raising test slowly, whether the patient is sitting or recumbent, as it can create much pain in the low back or lower extremity for the patient and negatively affect the results of other testing.

Valsalva maneuver and Lindner's sign (Fig. 10.84). For the Valsalva maneuver, the patient attempts to expel air against a closed glottis. This movement can be described to the patient as straining to move the bowel. During this maneuver, the intradiscal pressure increases, and the increased force against the anterior dura lining of the nerve root accentuates the patient's back or leg pain. Note also that the patient is asked to flex the head on the chest, which increases the traction of the nerve root against the disc bulge (Lindner's sign).

Raney (131) has stated that with a contained disc (i.e., the posterior anulus is not ruptured) flexion or maintenance of the flexed position obliterates the disc bulge and, assuming that motion of an irritated nerve root over a bulging disc is often the source of the patient's back and leg pain, thus could be the explanation for relief of pain with flexion treatment. He demonstrated that both the Valsalva maneuver and abdominal compression obliterate the myelographic defect. Again, if it is assumed that motion of an irritated nerve root over a disc bulge is one of the causes of pain, the findings here could explain how abdominal compression or Valsalva maneuver done abruptly increases the patient's pain as the defect appears and disappears, and thereby moves the nerve root over the disc.

Bechterew's test, Lindner's sign, and Valsalva maneuver (Fig. 10.85). If Bechterew's test is added to the Valsalva maneuver, further stretching the nerve roots behind the intervertebral disc space, this increased stretching accentuates the patient's pain in nuclear matter escape. The combined positive reaction of the Valsalva maneuver, Bechterew's test, and

Table 10.7

Low Back Pain Examination Form

Low Back Examination Form

Name ____________________ Date __________ Pain Onset __________
Occupation: ____________________ Age: _____ Sex: M. F. S.M.D.W.
Chief Complaint: ____________________

History:
Mechanism
Narration
Onset
Palliative/Provocative
Quality
Radiation
Severity
Time
U had this before

PMHx.:

Dr's:	C/V dz.:
Sx:	DM:
GU	GI:
CA:	Drugs:
Injuries	Other Treatments:

Family History:

CA:	DM:
C/V dz:	L.B.P./Sciatica:
Other dz.:	

Social History:

Nicotine:	ETOH:

Activities:

Physical Examination:

		Location of Pain.	Aggravated By.
B/P: __________	Lung Sounds:	Rt. Leg _____	Coughing _____
Pulse: __________	Heart Sounds:	Lt. Leg _____	Sneezing _____
Respirations: __________	Lymph nodes:	Both _____	Straining _____
Temp: __________	Skin:	Alternating _____	Bending and Lifting _____
Ht.: __________	Abdomen:	Leg Pain Onset:	Sitting _____
Wt.: __________	Prostate:	Before B.P. _____	
		After B.P. _____	
		With B.P. _____	

continued

Physical Examination Sitting:

Test/Sign	Minor's Sign	Bechterew's Sign	Valsalva maneuver	Valsalva maneuver w/Bechterew's sign
Negative				
Pos. LBP				
Pos. LP				

Examination Standing:

Examination	Kemp's Sign	Toe Walk	Heel Walk	Spinal Tilt	Neri's Bow	Lewin's Sign	Lordosis	Gait
Normal								
Abn. R							Increased	Rt. Limp
Abn. L							Decreased	Lt. Limp

Examination: **Supine:**

Pain upon Palp.		Percussion	Range of Motion			Sensory	
Negative		Neg.		Range	Pain	Norm.	
L	R		Flexion	_____	_____		
L1	L1	L1				Right	
L2	L2	L2	Extension	_____	_____	Left	
L3	L3	L3	Lateral			Hypes.	Hyperes.
L4	L4	L4	Flexion			L1	
L5	L5	L5	Rt.	_____	_____	L2	
S1	S1	S1	Lt.	_____	_____	L3	
TFL.	TFL.		Rotation			L4	
G. Max.	G. Max.		Rt.	_____	_____	L5	
G. Med.	G. Med.		Lt.	_____	_____	S1	
Pirif.	Pirif.					S2	
Addct.	Addct.						

Examination Supine:

Examination	SLR._____° R. L.	Braggard's sign	Medial Hip Rot.	WLR._____° R. L.	Lindner's sign	Patrick's sign
Negative						
Pos. LBP						R. L.
Pos. LP						
Pos. Both						

Muscle Strengths: **Reflexes:**

(0–5)	Dorsi-flexion	Plantar Flexion	Hallux Flexion	Hallux Extension	Foot Eversion		Right	Left
Normal						**Patellar**	0, 1, **2**, 3, 4, 5	0, 1, **2**, 3, 4, 5
Weak R						**Ankle**	0, 1, **2**, 3, 4, 5	0, 1, **2**, 3, 4, 5
Weak L						**Babinski**	Neg. Pos.	Neg. Pos.

Examinaton Supine: **Circulation:** **Measurement:**

Examination	Cox's Sign	Amoss' Sign	Moses' Sign	Milgrams' Sign			Circumference
					Fem. Art.	Norm.⇓	R. Thigh_____
					Pop. Art.	Norm.⇓	L. Thigh_____
Negative					Post.Tib.Art.	Norm.⇓	R. Calf_____
Positive			R. L.	R. L.	Dors. Pedis	Norm.⇓	L. Calf_____

continued

Examination Prone:

Examination	Yeoman's Sign	Ely's Sign	Nachlas' Sign	Popliteal Fossa Pain	Prone Lumbar Flexion
Negative					
Pos. Rt.					
Pos. Lt.					

Nonorganic Physical Signs:

Examination	Libman's Sign	Tenderness to Skin Pinch	Mannkopf's Sign	Burn's Bench	Flip Test	Plantar Flexion	Flexed Hip Test	Axial Load	Rot. Of Shoulders & Pelvis
Neg.		Specific							
Pos.		Nonanatomic							

X-Rays Standing or Recumbent:

Spinal Mechanics						
Spinal Tilt	Scoliosis	Sacral Angle	Lumbar Lordosis Angle	Facet Asymmetry	Facet Syndrome	Van Akkerveeken Stability
None L. R. L1 L2 L3 L4 L5	None L. R. L1 L2 L3 L4 L5 Mild Moderate Severe	______ °	________ °	Sagittal Coronal L. R. L1–L2 L2–L3 L3–L4 L4–L5	Present Absent L4–L5 L5–S1	Stable Unstable

Congenital Abnormalities:

Spina Bifida	**Spondy-lolysis**	**Spondy-lolisthesis**	**Transitional Vertebrae**	**Stenosis Sagittal Diameter Spinal Canal Vertebral Body**		**Intercrestal Line Cuts**
None L1 L2 L3 L4 L5 S1 S2 S3	None L1 L2 L3 L4 L5 S1	None L1 L2 L3 L4 L5 Percent_____	Sacralization L. R. Lumbarization L. R. True False	L1–2 L2–3 L3–4 L4–5 L5–S1	L1–2 L2–3 L3–4 L4–5 L5–S1	L4 Body L5 Body

Acquired Anomalies:

Schmorl's Nodes	Narrowed Disc Space	Spondylosis	Articular Facet Arthrosis	Retrolisthesis	Other
None L1 L2 L3 L4 L5	None L1–L2 L2–L3 L3–L4 L4–L5 L5–S1	None Slight L1–L2 L2–L3 L3–L4 L4–L5 L5–S1	None Slight L. R. L1–L2 L2–L3 L3–L4 L4–L5 L5–S1	None L1 L2 L3 L4 L5	

continued

FLOW CHART FOR CORRELATIVE DIAGNOSIS

LOW BACK AND/OR LEG PAIN (BELOW KNEE DIAGNOSIS)

DISC

DERMATOME
- L4 — ___L3-L4 DISC
- L5 — ___L4-L5 DISC
- S1 — ___L5-S1 DISC

LEAN
- NO LEAN — ___SUBRHIZAL
- INTO SIDE PAIN — ___MEDIAL
- AWAY SIDE PAIN — ___LATERAL = ___ ___ ___

LEVEL RT MEDIAL
LT LATERAL
SUBRHIZAL

DEJERINE TRIAD
- POSITIVE — PROTRUSION
- NEGATIVE — PROLAPSE

LEG PAIN WORSE THAN LOW BACK
- NO — PROTRUSION
- YES — PROLAPSE

___ PROTRUSION (722 1) - CATEGORY III ___ PROLAPSE(722 1) - CATEGORY IV

BERTOLOTTI'S SYNDROME (L4 DISC WITH L5S1 TRANSITIONAL SEGMENT) YES NO

LOW BACK PAIN (NO LEG PAIN BELOW KNEE) DIAGNOSIS

CATEGORY I	CATEGORY II	CATEGORY V	CATEGORY VI		CATEGORY VII		CATEGORY VIII
Annular Tear (722 1)	Nuclear Bulge (722.1)	Discogenic Spondyloarthrosis	Facet Syndrome		Spondylolisthesis		Stenosis
- low back pain	- low backpain - buttock pain into thigh to knee		Stable (< 3 MM)	Unstable (> 3 MM)	True (Pars Defect)	False (Degenerative)	(Eisenstein < 12 mm or Body Canal > 4:1)
Clinical Judgment No hard objective findings—rotation and flexion injury		___ L1-L2	___ L1-L2	___ L1-L2	L1 ___		L1 ___
		___ L2-L3	___ L2-L3	___ L2-L3	L2 ___		L2 ___
		___ L3-L4	___ L3-L4	___ L3-L4	L3 ___		L3 ___
		___ L4-L5	___ L4-L5	___ L4-L5	L4 ___		L4 ___
		___ L5-SI	___ L5-SI	___ L5-SI	L5 ___		L5 ___
					% ___		Pedicogenic (< 12 mm) Acquired (Degenerative Facets)

CATEGORY IX	CATEGORY XI	CATEGORY XII			CATEGORY XIII	CATEGORY XIV	CATEGORY XV
F.B.S.S (Failed Back Surgical Syndrome)	Sprain ___	SUBLUXATION			Tropism	Transitional Segement	Scoliosis and other Pathologies
	Strain ___					Yes ___ No ___	
Level		Sacroiliac	L1	Retrolisthesis	___ L1-L2	RT ___ L5 ___ True ___	Levo Dextro
Fusion ___	___ L1-L2	RT ___	L2	Anterolisthesis	___ L2-L3	LT ___ L6 ___ False ___	
Microsurgery ___	___ L2-L3	LT ___	L3	Right Lateral Flexion	___ L3-L4	Sacralization ___	
Laminectomy ___	___ L3-L4	Post ___	L4	Left Lateral Flexion	___ L4-L5	Lumbarization ___	Thoracic L1 L2 L3 L4 L5 Sacrum
Chemonucleolysis ___	___ L4-L5	Ant ___	L5	Right Rotation	___ L5-SI		
Epidural Steroid ___	___ L5-SI	Short Legs		Left Rotation			
Rhizotomy ___		RT ___		Hyperflexion			
		LT ___		Hyperextension			

CORRELATIVE DIAGNOSIS OF LOW BACK PAIN AND LEG PAIN

RT	L3-L4	Lateral	Discal Protrusion (722.1)	WITH
LT	L4-L5	Medial	Discal Prolapse (722.1)	
	L5-S1	Subrhizal		
		Central		

CORRELATIVE DIAGNOSIS OF LOW BACK PAIN		L1-L2	L2-L3	L3-L4	L4-L5	L5-SI
CATEGORY V	Discogenic Spondyloarthrosis (722.52)					
CATEGORY VI	Stable or Unstable Facet Syndrome (724.8)					
CATEGORY VII	Spondylolisthesis (True or False) (756.16)					
CATEGORY VIII	Stenosis (Pedicogenic or Degenerative)(724.02)					
CATEGORY IX	Post Surgical Care (722.83)					
CATEGORY XI	Sprain or Strain (847 2)					
CATEGORY XII	Subluxation (739.3)					
CATEGORY XIII	Tropism (756.10)					
CATEGORY XIV	Transitional Segement (756.19)					
CATEGORY XV	Scoliosis (737.0)					
	or Other Pathology					

Table 10.8

Criteria for Diagnosis of Sciatica Due to a Herniated Intervertebral Disc

1. Leg pain is the dominant symptom when compared with back pain. It affects one leg only and follows a typical sciatic (or femoral) nerve distribution.
2. Paresthesiae are localized to a dermatomal distribution.
3. Straight leg raising is reduced by 50% of normal, and/or pain crosses over to the symptomatic leg when the unaffected leg is elevated, and/or pain radiates proximally or distally with digital pressure on the tibial nerve in the popliteal fossa.
4. Two of four neurologic signs (wasting, motor weakness, diminished sensory appreciation, and diminution of reflex activity) are present.
5. A contrast study is positive and corresponds to the clinical level.

Based on McCullough JA. Chemonucleolysis. J Bone Joint Surg 1977;159B:45–52.

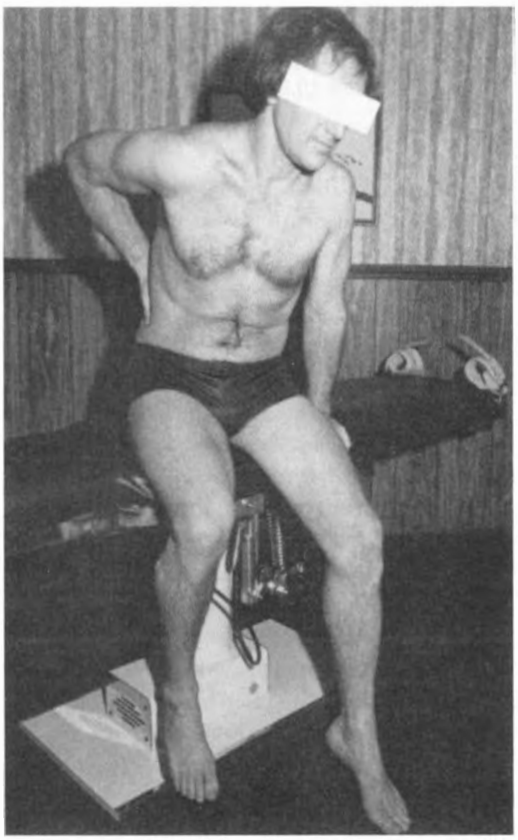


Figure 10.82. Minor's sign.

Figure 10.83. Bechterew's sign.

Figure 10.84. Valsalva maneuver and Lindner's sign.

Figure 10.85. Bechterew's test, Lindner's sign, and the Valsalva maneuver.

Lindner's sign indicates the presence of a disc lesion. One test alone might not be positive.

Patient Standing

Néri's bowing sign (Fig. 10.86). With Néri's sign, as the patient bows forward, the affected leg flexes, as in a curtsey, as the sciatic nerve is irritated. Knee flexion removes the tractive irritation from the inflamed sciatic nerve.

Lewin's standing sign (Fig. 10.87). Lewin's standing sign is manifested with the patient's knees placed in extension. Increased pain in the low back or leg can cause the knee to snap back into flexion. If this is observed, a disc, gluteal, or sacroiliac disturbance is indicated.

Gait (Fig. 10.88). Note whether the patient limps while walking and, if so, which extremity is affected.

Patient lean (Fig. 10.89). Note whether the patient leans to the right or the left. Later, correlation of this antalgia with the side of the pain will aid in determining whether the nuclear bulge is medial, lateral, or subrhizal.

In detailing the meaning of the sciatic scoliotic antalgic lean of a patient, that is, whether the patient leans away from the side of pain for a lateral disc lesion or into the side of pain for a medial one, remember two important findings. First, Lindblom (132) enhanced our thinking on the importance of the lateral bending significance of disc protrusion by his finding that, in rat tails tied into "U" shapes, degeneration and rupture occurred on the concave side of the spine while the convex side remained normal. Second, Porter and Miller (133) stated that 20 patients they studied did not indicate the side of the list to be related to the side of the sciatica or to the topographic position of the disc in relation to the nerve root. It is, therefore, up to the clinician to carefully integrate lateral flexion lists with other findings to arrive at the correct clinical impression.

Figure 10.87. Lewin's sign.

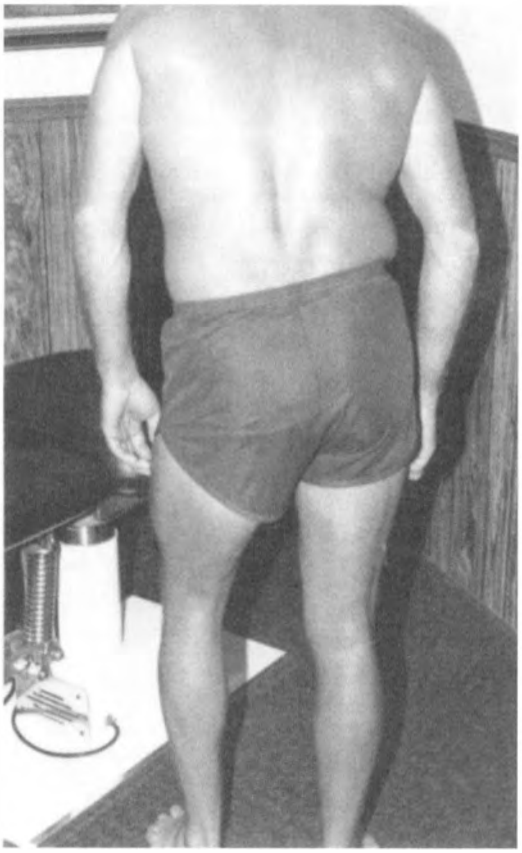


Figure 10.86. Néri's bowing sign.

Lumbosacral list has received a number of designations, including alternating lumbar scoliosis, alternating sciatic scoliosis, sciatic scoliotic list, trunk list, gravity-induced trunk list, "wind swept" spine, and lumbosacral list.

List hypotheses are as follows: (*a*) increasing back and leg pain with lateral leaning results from increased stretch of the nerve root in relationship to a disc herniation; (*b*) increasing pain with contralateral leaning implicates a medial herniation; and (*c*) increasing pain with ipsilateral leaning implicates a disc herniation lateral to the nerve root.

Radiologic study of the lumbosacral list may determine the segmental level of the disc herniation. Laterality of the lumbosacral list does not indicate the relationship of the disc herniation to the nerve root, either axillary or lateral. Nevertheless, the lumbosacral list remains an important clinical sign (134).

Medial disc protrusions have poorer clinical outcomes than lateral disc protrusions and show a higher incidence of cauda

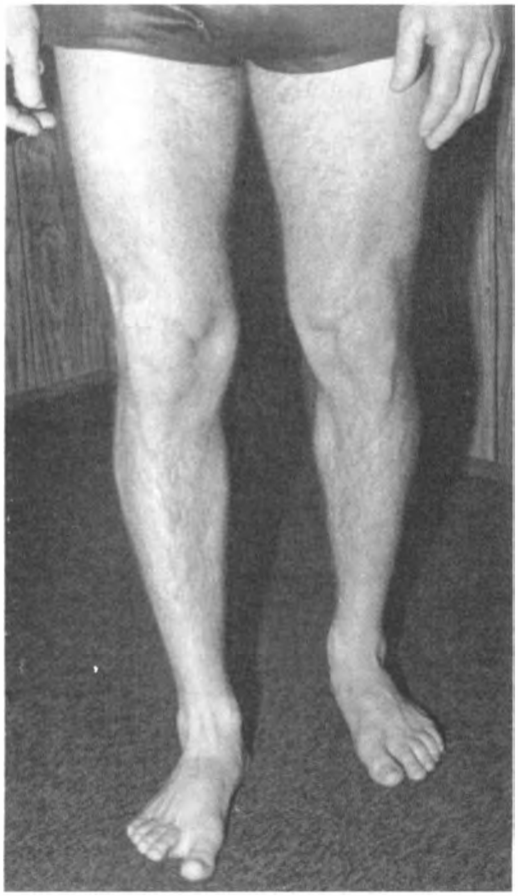


Figure 10.88. Gait.

equina syndrome. Up to 33% of lumbar disc herniations are midline. Also referred to as central or dorsal, they protrude through the strong central fibers of the posterior longitudinal ligament or the anulus. The midline herniation reportedly causes predominantly low back pain because of stretching or injury to the posterior longitudinal ligament and, rarely, sciatica. Passive straight leg raising typically produces back pain without radiation. Cauda equina syndrome, which can result from medial disc protrusion, has been reported extensively in the literature (135).

Lumbar lordosis (Fig. 10.90). Note whether the patient while standing reveals increased, decreased, or normal lumbar lordosis. The typical disc patient will have a loss of lumbar lordosis because this posture opens the dorsal intervertebral disc space, thus relieving the pressure of nuclear bulge on the involved nerve root or cauda equina.

Chronic pain patients exhibit *increased lumbar lordosis,* and acute pain patients exhibit *increased thoracic kyphosis and a forward head position* in the standing position. Sitting finds acute patients to have increased thoracic kyphosis compared with controls (136).

Pain on palpation (Fig. 10.91). Note the levels of pain that the patient experiences on deep digital pressure. Sometimes, not only the back pain but also a radiating sciatic discomfort can be elicited.

Percussion (Fig. 10.92). Tapping over the involved paraspinal and spinous process levels creates pain if inflammatory changes are present around the involved nerve roots.

Kemp's sign (Fig. 10.93). The test for Kemp's sign can be performed with the patient in either the standing or the sitting position. Sitting increases intradiscal pressure and, therefore, maximizes stress to the disc, whereas standing increases weightbearing and maximizes stress to the facets. The test for Kemp's sign should be performed in both positions. Kemp's sign can be positive for facet irritation or compression of a bulging nucleus against a nerve root. If both are present, low back pain is elicited. With a disc bulge, accentuation of the lower extremity radiculopathy is increased. Some patients with disc lesion experience only back pain with Kemp's sign. With a medial disc, Kemp's sign is usually positive when the patient is flexed either to the right or to the left in extension. Pain occurs because a medial disc can irritate a nerve root regardless of the direction in which the patient is posteriorly and laterally flexed. In medial disc protrusion it is expected that the patient will experience greater pain when flexed away from the side of pain or disc lesion, whereas in lateral disc protrusion, the patient will experience greater pain when flexed into the side of low back and lower extremity pain.

Goniometric measurements (Figs. 10.94–10.97). Goniometric measurements should be taken with the patient

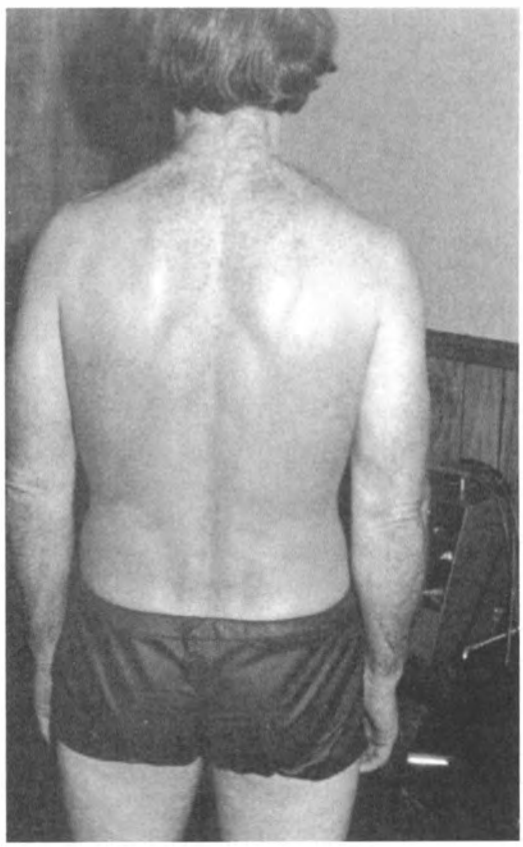


Figure 10.89. Lean of patient.

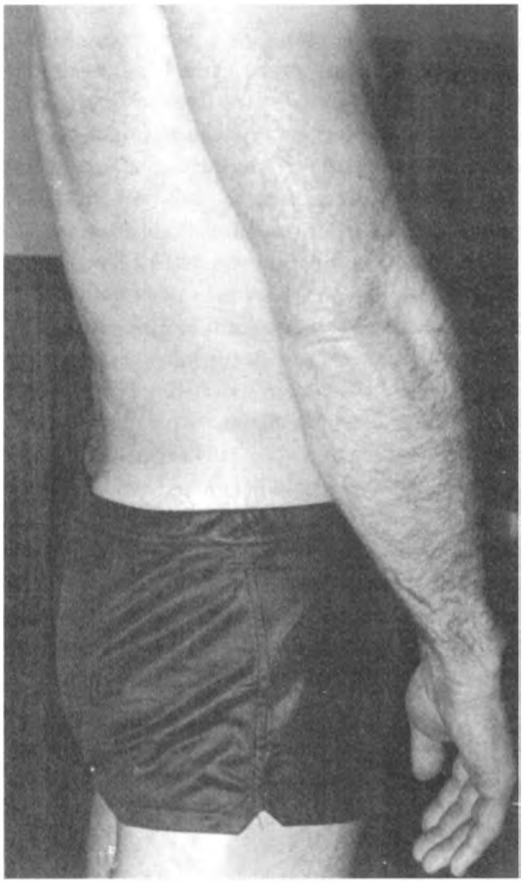


Figure 10.90. Lumbar lordosis.

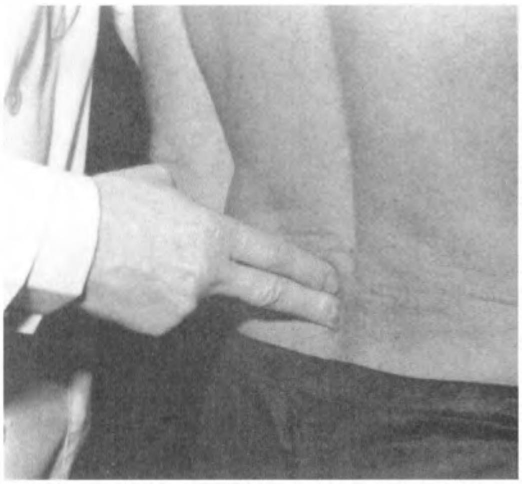


Figure 10.91. Pain on palpation.

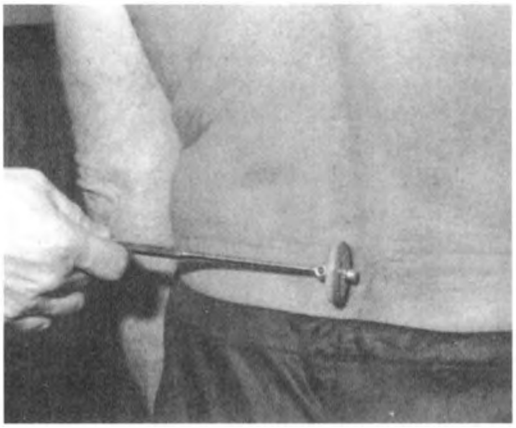


Figure 10.92. Percussion.

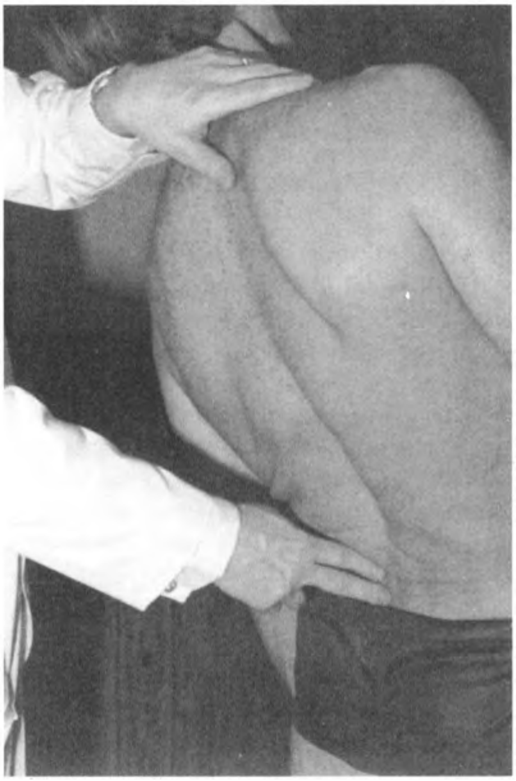


Figure 10.93. Kemp's sign.

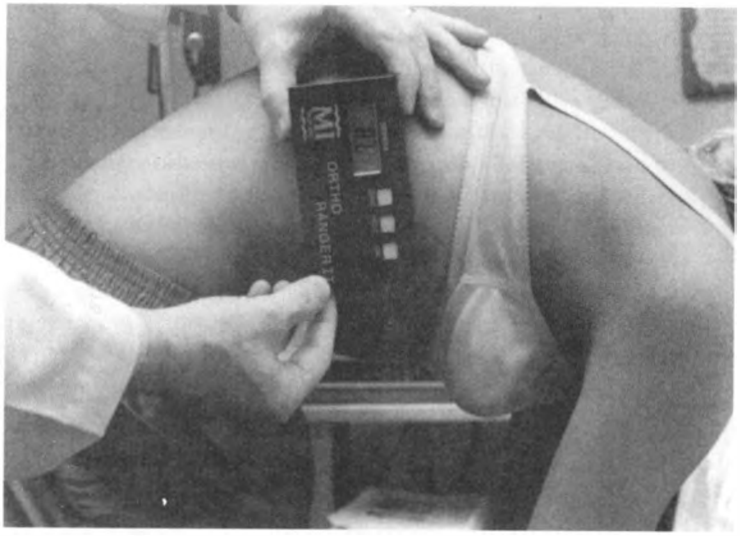


Figure 10.94. Flexion measured.

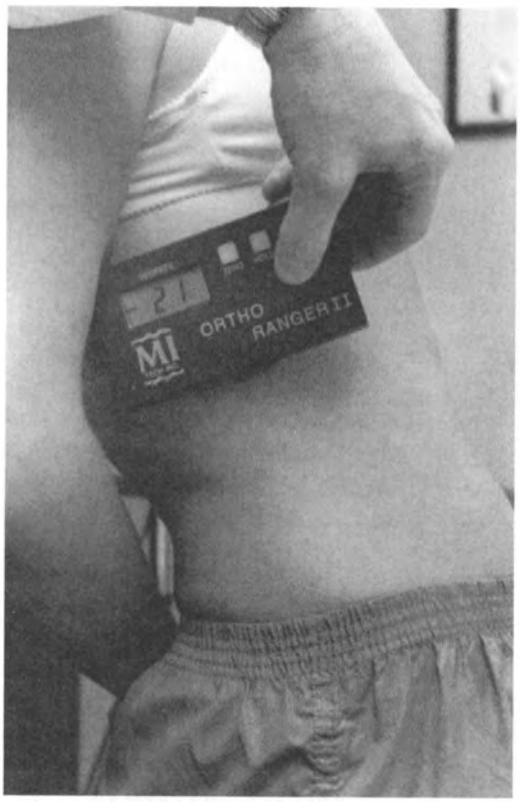


Figure 10.95. Extension measured.

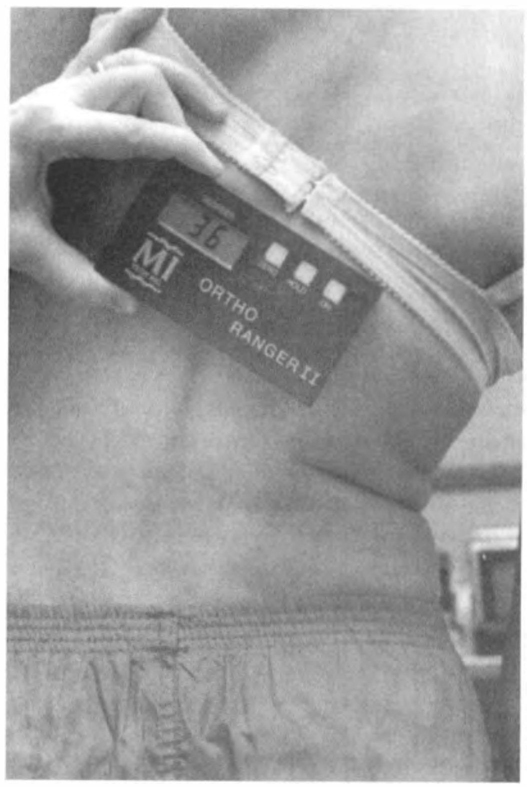


Figure 10.96. Lateral flexion measured.

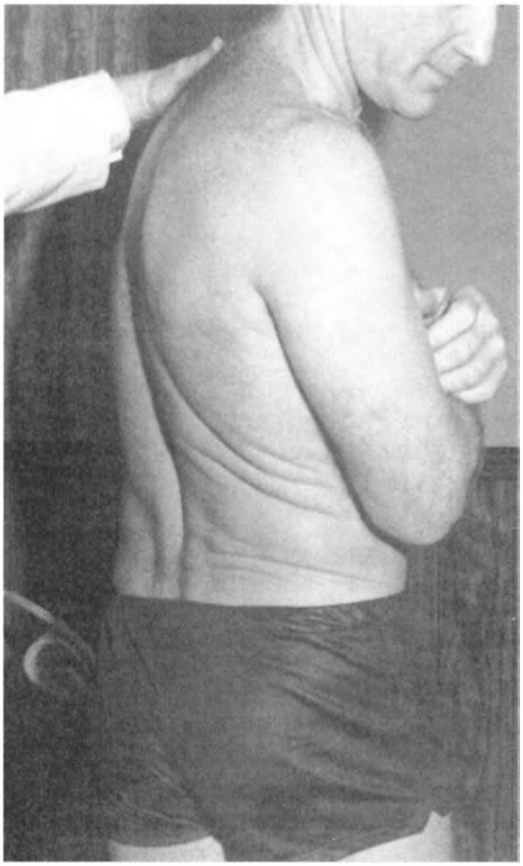


Figure 10.97. Rotation measured.

in flexion, extension, lateral bending, and rotation of the lumbar spine. These measurements provide a record of the ranges of motion for comparison with future measurements and for verification of patient response or failure to treatment.

Digital computerized goniometers have shown greater accuracy than older, handheld metal or plastic goniometers. The accuracy of goniometric measurement is critical, because range of motion status, coupled with improvement of the SLR sign, are the two tests used to determine the progress of a patient under care. Specifically, in our clinical practice, we feel that a patient must show at least 50% improvement within 3 to 4 weeks of conservative manipulative care or we change our treatment protocol and perform more diagnostic tests and entertain surgical consultation. Million et al. (137) found objective assessments of spinal motion and SLR to show a high degree of intraobserver reproducibility, thereby emphasizing their importance in evaluating the progress of the low back pain patient.

Range of Motion of the Thoracolumbar Spine

Repeated measurements were made of lumbar sagittal range of motion by 14 examiners using three different measuring instruments to determine the reliability of lumbar range of motion measurements among examiners and subjects, and to determine whether variance is caused by subject inconsistency, examiner inconsistency, differences between examiners, or differences between instruments.

No systematic difference resulted from instruments or posture condition. However, a statistically significant variance was found among examiners—a poor interexaminer reliability. Range of motion measurements must be interpreted with caution in clinical, research, and disability applications. Even when obtained with excellent instruments, results must be interpreted with caution (138).

Toe walk (Fig. 10.98). The inability to walk on the toes indicates an L5-S1 disc problem caused by weakness of the calf muscles supplied by the tibial nerve.

Heel walk (Fig. 10.99). The inability to walk on the heels indicates an L4-L5 disc problem caused by weakness of the anterior leg muscles supplied by the common peroneal nerve.

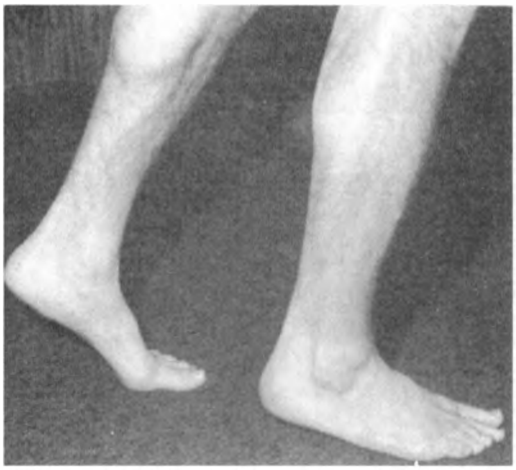


Figure 10.98. Toe walk.

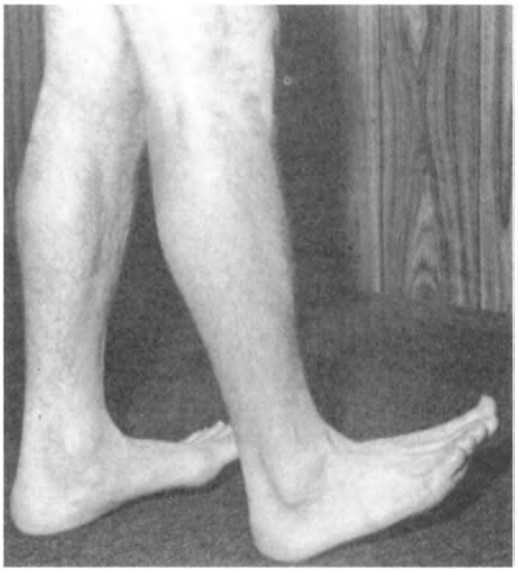


Figure 10.99. Heel walk.

Examination with the Patient in the Supine Position

Some of the following tests may be done with the patient in the prone position, depending on which position is more comfortable for the doctor and/or patient.

Lindner's sign (Fig. 10.100). The test for Lindner's sign (also known as the Brudzinski or Soto-Hall sign) is often performed in conjunction with the straight leg raising test or the Valsalva maneuver for maximal effect. Lindner's sign refers to stretching of the dural linings of the nerve roots behind the bulging disc material, which causes pain when performed.

Straight leg raising sign (Figs. 10.101 and 10.102). During straight leg raising the lumbosacral nerve roots move through their intervertebral foramina up to several millimeters, depending on the author quoted (139). Fisk states that the nerve roots move 2.5 cm (130). A great deal of traction is found of the sciatic nerve at the sacral ala and the sciatic notch, with movement first seen at the sciatic notch and later at the roots. If the patient feels pain soon after initiating the SLR maneuver, it can indicate either a large disc protrusion or nerve sensitivity at the sacral ala or sciatic notch. Movement of the sciatic nerve diminishes with age and proximity to the spinal cord.

In SLR, tension and movement develop first in the sciatic notch, then in the ala of the sacrum as the nerve passes over the pedicle, and finally at the intervertebral foramen itself. Movement of the nerve root through the intervertebral foramen has been cited to be 2 to 6 mm (20), 4 to 8 mm (140), and 2 to 5 mm (141).

It is important to remember that compressing or stretching a normal nerve is not painful. The SLR pain is a reflex or sensory input mechanism that protects a person from injury. The reason for SLR pain is explained as sensitivity of the dorsal roots caused by mechanical pressure. Perl (142) believes, however, that SLR pain is caused by a chemical noxious irritation by substances liberated by mechanical pressure.

Charnley (140) found SLR to be the best clinical or radiologic sign for diagnosing disc protrusion. Hakelius and Hindmarsh (143) found an inverse proportion to the degree of limitation of SLR and the percentage of positive disc herniation at surgery. Sprangfort (144) found that in young people the sign has no specific value for diagnosing disc herniation and that a negative SLR excluded disc herniation. After age 30, however, possible SLR is seen less often but its diagnostic value increases, and a negative SLR no longer excludes the diagnosis of disc herniation (144).

Laségue (145) described the painful effect in patients with sciatica of stretching the sciatic nerve by extending the knee with the hip flexed; he also described the relief from pain when the knee was then flexed. This is the classic leg raising sign. Variations of this sign, along with interpretations of its meaning, lend much more knowledge to the examining physician than merely noting that with a certain degree of leg raise the patient experiences either back or leg pain or both. On the examination form would be recorded whether the leg raising sign was positive and, if so, at what degree of elevation (Fig. 10.101*B*).

Figure 10.100. Lindner's sign.

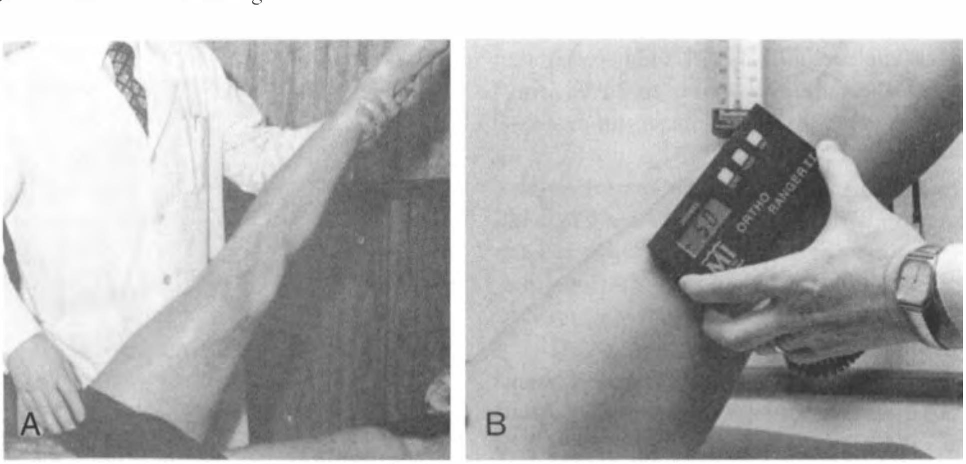


Figure 10.101. **A.** Straight leg raising (SLR) and medial hip rotation performed simultaneously. **B.** Goniometer measurement of angle at which SLR is positive.

Figure 10.102. Braggard's maneuver performed.

Breig and Troup (146) add a degree of sophistication to the SLR test. After noting the level of pain on straight leg raising, lower the extremity a few degrees to relieve the pain and then dorsiflex the ankle while medially rotating the hip. Medial hip rotation places greater stretch on the lumbar and sacral nerve roots and accentuates the SLR sign. These authors feel if the pain that limits straight leg raising is elicited by such dorsiflexion and medial hip rotation, increased root tension is indicated and the site of pain may help in locating the level of the disc causing the pain. Figure 10.101*A* shows medial hip rotation and Figure 10.102 shows dorsiflexion of the foot (Braggard's sign). Figure 10.101*B* shows goniometric measurement of SLR.

By stretching the lumbosacral nerve roots, the SLR sign proves that the first sacral nerve root allows the greatest movement.

In theory, the SLR should identify not only the presence of increased root tension but also, possibly, the site of such irritation. The production of pain on passive dorsiflexion of the ankle near the limit of the pain-free range of SLR confirms that the root is mechanically compromised. Pain on pressure in the popliteal fossa after flexion of the knee at the limit of SLR has a similar significance, and when the well leg raising test is positive, this pain is a strong confirmation of root involvement.

The angulatory stress exerted on the lumbar nerve roots during SLR was measured on cadavers within 4 hours of death (147). A short length of rubber tube was inserted between the disc and nerve root and the tension was monitored by semiconductor pressure transducers. Results of this testing were:

1. With the SLR, the pressure between the nerve root and the disc does not change until the leg is raised to about 30°, with a progressive rise occurring as the angle of the leg increases. The pressure increase is highest at the L5–S1 disc level and half as high at the L4–L5 level. The pressure increase on SLR at L3–L4 was one tenth of that at L5–S1.

   It can be concluded that (*a*) an SLR that is positive under 30° reveals a large disc protrusion. The nerve root is stretched here long before it normally would be. (*b*) SLR is most useful in identifying L5–S1 disc lesions, because the pressures are highest at this level. On SLR, L4–L5 is not as apt to give as much pain as is L5–S1, because the pressure between the disc and the nerve root is half that at L5–S1. Therefore, the L5–S1 disc lesion gives more pain in the low back and leg than does the L4–L5 disc lesion. (*c*) No movement on the nerve root occurs until SLR reaches 30°. (*d*) No movement of the L4 nerve root occurs during SLR (148).
2. Adduction of the hip on SLR increases the pressure on the nerve roots.
3. The second, third, and fourth lumbar nerve roots show no increase in tension during SLR but did show an increase during the femoral stretch test (149).

Straight leg raising and Lindner's signs (Fig. 10.103). Whenever the straight leg raising test produces a questionable result for pain, combine it with flexion of the cervical spine (Lindner's sign). This combination places the greatest pull and stretch on the nerve roots behind the intervertebral disc and often elicits pain. Along with this combination, dorsiflex the foot, have the patient cough, or perform the Valsalva maneuver. These maneuvers further accentuate intradiscal pressure and elicit pain that otherwise might be missed.

Swan and Zervas (150) found that simultaneous flexion of the neck and elevation of the contralateral leg produced pain in the ipsilateral (presenting) sciatic notch in five patients with either free fragments or herniated disc found at operation. Raising the contralateral leg alone elicited no pain in either leg.

Adduction and internal rotation of the leg while SLR is performed brings out the pain response more readily; this is called Bonet's phenomenon. Also performing dorsiflexion of the foot during SLR is called Braggard's sign; and extension of the great toe during SLR to accentuate the nerve root stretch is called Sicard's sign.

Well leg raising (Fajersztajn) sign (Figs. 10.104 and 10.105). The well leg raising sign (Fajersztajn sign) is exacerbation of pain down the involved or painful lower extremity when the opposite or noninvolved extremity is placed in straight leg raise. Hudgins (151) states that increased sciatica on raising the opposite or well leg (the cross straight leg raising sign) is associated with a herniated lumbar disc in 97% of patients. Myelography is unnecessary for the diagnosis of disc her-

Figure 10.103. Tests for straight leg raising and Lindner's signs, performed together.

Figure 10.104. Interpretation of the well leg raising sign in a case of lateral disc bulge.

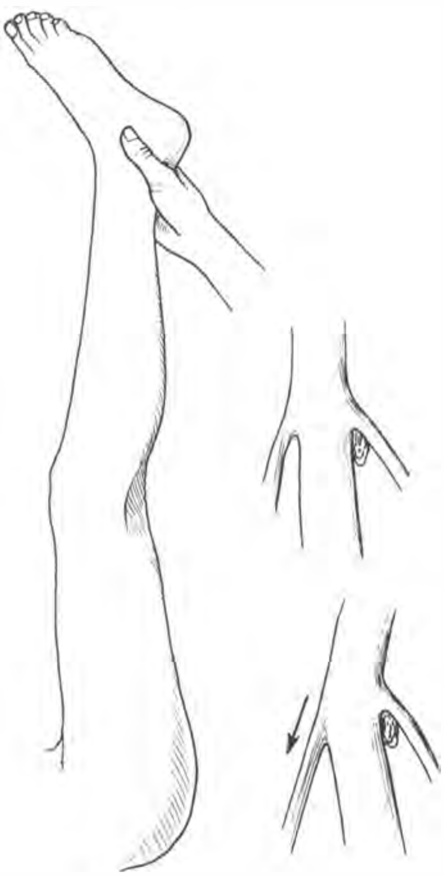


Figure 10.105. Interpretation of the well leg raising sign in the case of a medial disc bulge.

nia in patients with this sign. Although it is possible for patients with this sign to have a normal myelogram, nevertheless, 90% prove to have a herniated disc.

When the disc protrusion is displaced lateral to the nerve root (Fig. 10.104), raising the uninvolved leg actually pulls the nerve root away from the disc and can relieve back or leg pain.

When the disc protrusion is displaced medial to the nerve root (Fig. 10.105), raising the uninvolved leg actually pulls the nerve root into the disc bulge and causes radiculopathy down the involved leg.

Interpretation of the straight leg raising sign. In a study of 50 patients in a 2-year period, Edgar and Park (152) found that the pattern of pain on SLR was closely related to the central or lateral position of the disc protrusion. In addition to its use in the diagnosis and assessment of progress, the SLR sign may be helpful in localizing the protrusion by analysis of the distribution of the pain so induced. Clinically, myelographic and operative observations were carried out prospectively on 50 such patients to investigate the relationship between the pattern of pain in SLR and the site of the protrusion. In 80% of the patients, the following correlation was found:

Location of Protrusion	Back Pain	Leg Pain
Lateral protrusion		+
Medial protrusion	+	
Intermediate protrusion (subrhizal)	+	+

Therefore, a lateral protrusion causes a patient to experience leg pain; a medial protrusion, back pain; and a subrhizal protrusion, both back and leg pain.

The straight leg raising sign can provide a wealth of information; the level of pain can indicate the disc at fault; the presence of back pain, leg pain, or both can indicate the type of protrusion; and various combinations of Valsalva, cervical flexion, dorsiflexion of the foot, and medial hip rotation can aid significantly in diagnosis.

Macnab (153) demonstrated the bowstring sign as being the most reliable test of root tension in sciatica caused by an intervertebral disc lesion (Fig. 10.106).

Shiqing et al. (154) reported on a study of 113 patients that the distribution of pain on SLR allowed an accurate prediction of the location of the lesion in 100 (88.5%) of the cases. Central protrusions caused back pain, lateral protrusions caused lower extremity pain, and intermediate protrusions caused both.

Validity and importance of SLR in objective evaluation. Lastly, concerning the SLR, remember its importance

Figure 10.106. When eliciting the bowstring sign, the patient's foot should be allowed to rest on the examiner's shoulder with the knee slightly flexed at the limit of straight leg raising. Sudden firm pressure is then applied by the examiner's thumbs in the popliteal fossa. Radiation of pain down the leg or the production of pain in the back is pathognomonic of root tension. (Reprinted with permission from Macnab I. Backache. Baltimore: Williams & Wilkins, 1977.)

in the diagnosis and evaluation of progress of the patient under treatment for sciatic caused by a disc lesion. High degrees of reproducibility of interexaminer objective assessment were found for SLR (137). SLR has been found to be the most reliable and strongly recommended objective test in evaluating spinal manipulative response for low back pain (155).

Miller et al. (156) evaluated tests including gait, toe and heel walk, plantar flexion, cervical flexion, patellar and Achilles reflexes, SLR, and sensibility to pinprick and light touch, and found that the SLR had the best intra and interexaminer reliability. Figure 10.101*B* shows measurement of SLR with the digital goniometer for recording accuracy.

Importance of SLR

The straight leg raising test is regarded as probably the most important clinical test for evaluating lumbar nerve root tension caused by disc herniation. The incidence of a positive SLR test varies between 81 and 99%. A positive SLR test postoperatively correlates with inferior surgical outcome (157).

The straight leg lift was the most sensitive preoperative physical diagnostic sign (90%) for correlating intraoperative pathology of lumbar disc herniation (158).

Sock Test

Protrusions were not found in patients who could not reach to the ankle (the "sock test") and yet had an SLR greater than 40°. Neither was there a patient with a protrusion who could reach to the ankle or distal to the ankle and had a SLR less than 40° (159).

L5 and S1 Nerve Root Compressions More Likely Positive on SLR

Straight leg raising is more likely to be positive with an L4–L5 or L5–S1 disc herniation than with other high lumbar (L1–L4) herniations in which the test is positive in only 73.3% of patients. The likely reason for this is that the L5 and S1 nerve roots move 2 to 6 mm at the level of the neural foramen, whereas higher lumbar nerve roots show little excursion (160).

What Level of SLR Is Significant?

Tension is transmitted to the nerve roots once the leg is raised beyond 30°, but after 70°, further movement of the nerve is negligible. A typical positive SLR sign is one that reproduces the patient's sciatica between 30° and 60° of leg elevation (161).

The relationship between the SLR test and the size, shape, and position of the hernia was evaluated before inception of nonoperative treatment and then 3 and 24 months after treatment. The limitation of the SLR test was not related to size or position of the hernia. A decrease in hernia size over time, irrespective of shape, was not correlated to a concomitant improvement in SLR. It must be presumed that additional factors (e.g., inflammatory reactions affecting the nerve roots) are of importance for the magnitude of SLR (162).

Patrick's sign (Fig. 10.107). Patrick's sign refers to pain in the groin and hip area, which is common with disc lesion because of the irritation of nerve supply to these structures. Radiographic evaluation of the hip will rule out any hip disease.

Gaenslen's sign (Fig. 10.108). The test for Gaenslen's sign is performed by flexion of one knee upon the chest, while the other is placed in extension over the side of the table. This is a differential sign between sacroiliac and lumbar spine pain. When the test is performed, the pain will appear at the location of the lesion, whether it be in the sacroiliac or lumbar spine.

Cox's sign (Fig. 10.109). Cox's sign occurs when, during SLR, the pelvis rises from the table rather than the hip flexing.

Figure 10.107. Patrick's sign.

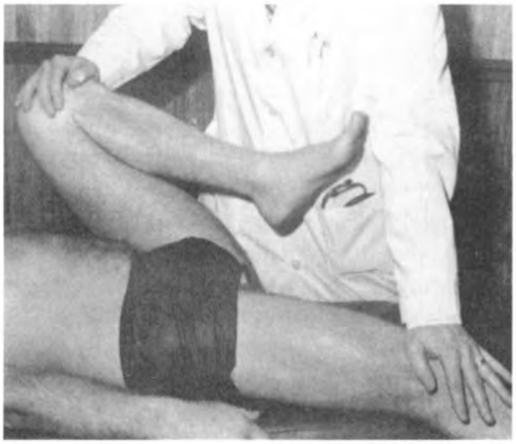


Figure 10.108. Gaenslen's sign.

Figure 10.109. Cox's sign.

I have noticed this occurrence in patients with prolapse into the intervertebral foramen—a grave condition.

Amoss' sign (Fig. 10.110). Amoss' sign is manifested by difficulty in rising from the supine position. The patient must use the arms to lift him or herself and prevent flexion or motion of the lumbar spine.

Dorsiflexion of the foot (ankle extension) (Fig. 10.111). The sciatic nerve is made up of tibial and common peroneal nerves. The common peroneal nerve divides into the superficial and the deep peroneal branch. Dorsiflexion as shown in Figure 10.111 depends on nerve supply via the deep branch of the peroneal nerve to the anterior tibialis muscle, the extensor hallucis longus muscle to the great toe, and the extensor digitorum longus muscle to the toes. The superficial peroneal nerve supplies the peroneal muscles that allow the foot to flex laterally at the ankle as well as flex upward (dorsiflexion). Dorsiflexion weakness in the foot at the ankle is indicative of fifth lumbar nerve root compression by an L4–L5 disc level lesion.

Figure 10.110. Amoss' sign.

Figure 10.111. Dorsiflexion of the ankle.

The inability of the patient to walk on the heels is also indicative of the same finding, but testing the patient's strengths as shown in Figure 10.111 is a much more intricate evaluation. The patient may be able to walk on the heels, yet demonstrate weakness of the muscle on dorsiflexion.

Dorsiflexion of the great toe (Fig. 10.112). Dorsiflexion strength of the great toe is determined by testing the strength of the extensor hallucis longus muscle. Dorsiflexion weakness of the great toe is indicative of L5 nerve root irritation by an L4–L5 disc lesion.

Goodall and Hammes (163) have developed a prototype of a meter used to establish differences in dorsiflexion strength of the great toe to detect early L5 nerve root lesions. The meter is accurate within 2%.

Plantar flexion or ankle flexion of the foot (Fig. 10.113). The tibial branch of the sciatic nerve supplies the posterior tibialis, gastroc soleus, flexor digitorum longus, and hallucis longus muscles. Weakness of plantar flexion of the foot is indicative of first sacral nerve root compression by an L5–S1 disc lesion.

A variation of this test is to ask the patient to walk on the toes. The inability to do so indicates the same finding as that of the plantar flexion sign. As in testing in dorsiflexion, testing the strength of one foot against the other is a much more reliable sign, because a patient may be able to walk on the toes and still have calf muscle weakness on one side.

Peroneal muscle testing. The peroneal muscles, which are the evertors of the ankle and foot, receive nerve supply from the first sacral nerve root. Test their strength by asking the patient to walk on the medial borders of the feet; or have

Figure 10.112. Dorsiflexion of the great toe.

Figure 10.113. Plantar flexion of the ankle.

the patient sit on the edge of the table and, as the patient attempts to pull the foot into eversion and dorsiflexion, oppose this by pushing against the head and shaft of the fifth metatarsal bone with the palm of your hand.

Plantar flexion of the great toe (Fig. 10.114). The flexor hallucis longus tendon is tested for strength in plantar flexion of the great toe. Weakness here is indicative of a first sacral nerve root compression by an L5-S1 disc lesion.

Thigh measurement (Fig. 10.115). Both thighs are measured at the same distance above the superior patellar pole. Differing sizes indicate atrophy.

Calf measurement (Fig. 10.116). Both calves are measured at the same distance below the inferior patellar pole. Different sizes indicate atrophy.

Milgram's sign (Fig. 10.117). The inability to hold the feet 6 inches off the floor while in the supine position indicates extreme nerve root irritation and is believed to be a sign of arachnoiditis caused by iophendylate dye as well as disc lesion.

Ankle jerk reflex (Fig. 10.118). The deep reflex of the ankle known as the "Achilles reflex" is diminished or absent in the presence of an L5–S1 disc irritation of the first sacral nerve root and, therefore, is of extreme importance in evaluating lower disc involvement. Note that the patient's foot is held in dorsiflexion while the ankle jerk reflex is elicited. Thus, not only the reflex but also the strength of the muscular contraction of the calf muscle is observed. This test can be performed with the patient prone or supine.

Figure 10.116. Calf measurement for atrophy.

Figure 10.114. Plantar flexion of the great toe.

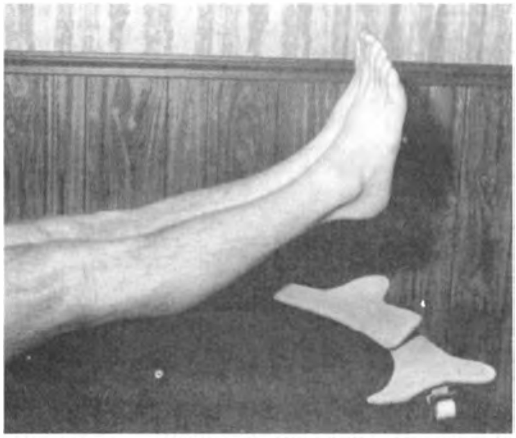


Figure 10.117. Milgram's sign.

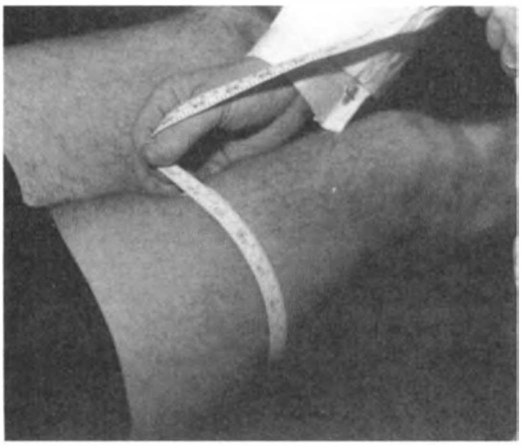


Figure 10.115. Thigh measurement for atrophy.

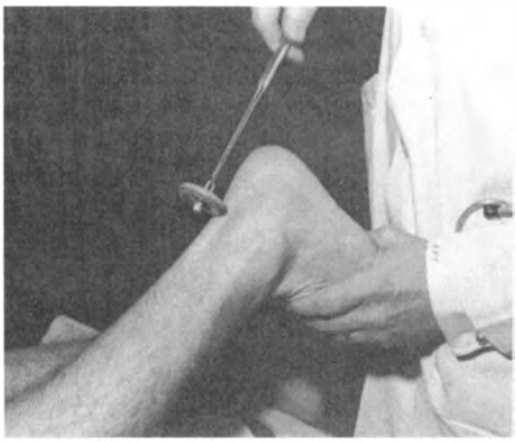


Figure 10.118. Ankle jerk reflex testing.

Absent Ankle Jerk May Be Normal

A significant number of "normal" adults have unilateral absence of an ankle reflex. Over the age of 40 years, in either sex, the proportion of patients with absent ankle reflexes increases; 1 to 10% of adults older than 40 years show unilateral absence of an ankle reflex. Unilateral loss is therefore a more useful neurologic sign and, where appropriate, will require further investigation, irrespective of age. Absent ankle reflex for herniated lumbar disc is reported to be approximately 90% between 20 and 45 years of age and 60% over the age of 50 years (164).

Patellar reflex (knee jerk) (Fig. 10.119). The patellar reflex sign indicates involvement of the L3 disc, which would affect the fourth lumbar dermatomes. Because discs other than the L4 or L5 are seldom involved, this is relatively useless in evaluating disc lesions in the lower extremity.

Pinwheel examination (Figs. 10.120–10.123). Pinwheel examination of the lower extremities is shown in Figures 10.120–10.123. The weight of the pinwheel is the only downward force applied to equalize the pressure of each leg. The same dermatome of each leg is stimulated, and the patient is asked which feels less sharp. Testing is shown of the fifth lumbar dermatome above the knee (Fig. 10.120); the L5 dermatome below the knee (Fig. 10.121); the dermatomes at the first sacral level of the thigh (Fig. 10.122); and the dermatomes at the first sacral level below the knee (Fig. 10.123). The first sacral dermatome is tested with the patient prone.

Figure 10.119. Patellar reflex (knee jerk) testing.

Figure 10.120. L5 dermatome.

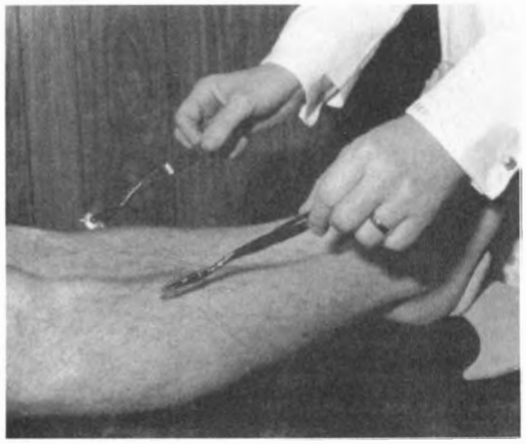


Figure 10.121. L5 dermatome.

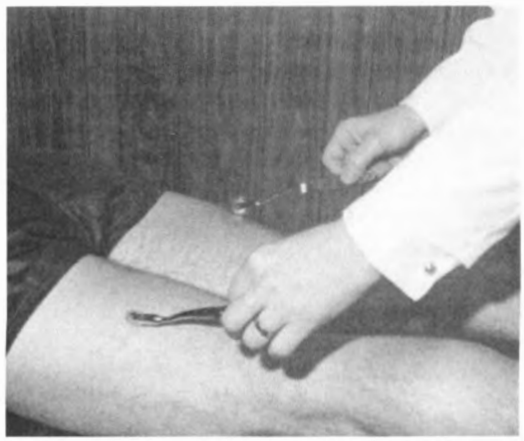


Figure 10.122. S1 dermatome.

Figure 10.123. S1 dermatome.

Figure 10.124. Tapping the origin of the inner hamstring muscles (semitendinosus and semimembranosus) at the ischial tuberosity to elicit the hamstring reflex.

Figure 10.125. Tapping the insertion of the inner hamstring muscles of the semimembranosus and semitendinosus tendons at the medial condyle and proximal portions of the tibia to elicit the hamstring reflex.

Vibratory sense. Vibratory sense can be tested; however, realize that older persons (aged more than 50 years) have a naturally decreased vibratory and temperature perception.

Tensor fascia femoris response. Macnab (153) discusses the reflex contraction of the tensor fascia femoris to plantar reflex and the loss of this response in S1 nerve root lesions.

Hamstring muscle reflex (Figs. 10.124 and 10.125). Loss of the hamstring reflex occurs in compression of the L5 nerve root by an L4–L5 disc protrusion.

Measurement of Lower Limb Circulation

Femoral artery (Fig. 10.126). Draw a line between the anterior superior iliac spine (ASIS) and the symphysis pubis; midway between these points, drop down 1 inch and that will be the femoral artery. Palpate the pulse and compare right to left for pulse strength.

Popliteal artery (Fig. 10.127). By Doppler or palpation determine the patency of the popliteal artery.

Posterior tibialis artery (Fig. 10.128). By Doppler or palpation compare the two pulses of the posterior tibialis arteries.

Dorsalis pedis artery (Fig. 10.129). By Doppler or palpation compare the pulse of the dorsalis pedis artery and its strength in the two extremities. This artery is located between the first and second metatarsal bones on the dorsum of the foot.

These pulses are important in differentiating intermittent claudication of ischemic cause from that of neurogenic cause. When these pulses are present and the patient has the cramp-like pains of claudication, the origin of pain is not vascular but neural. Look for discal lesions, ligamentous hypertrophy, stenosis, or peripheral neuropathy.

Moses' sign (Fig. 10.130). The test for Moses' sign is performed by grasping the calf of the patient's leg, which creates pain if phlebitis or vascular occlusion is present.

Examination with the Patient in the Prone Position

Nachlas' knee flexion sign (Fig. 10.131). On passive flexion of the knee, the patient lying in the prone position will experience pain in the low back or lower extremity. This sign is positive for sacroiliac, lumbosacral, and disc lesions.

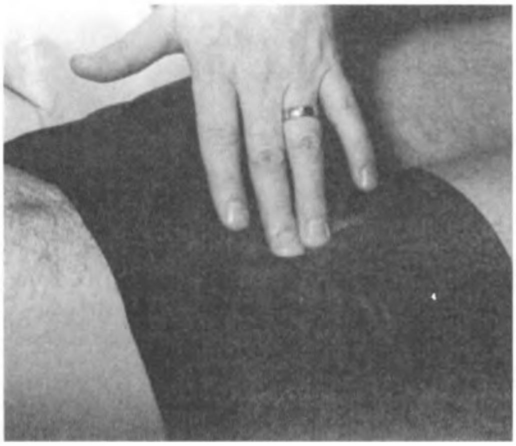


Figure 10.126. Femoral artery.

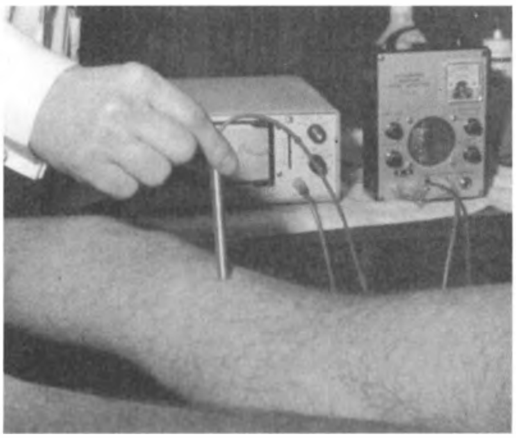


Figure 10.127. Popliteal artery.

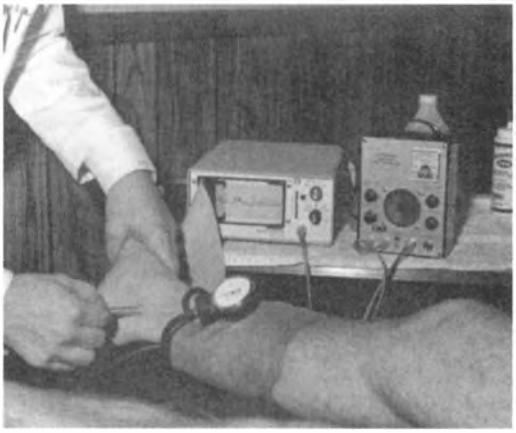


Figure 10.128. Posterior tibialis artery.

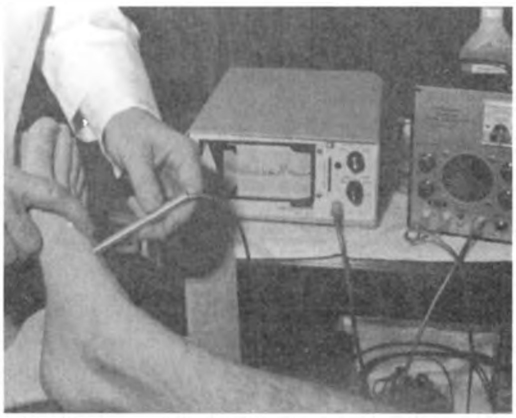


Figure 10.129. Dorsalis pedis artery.

Figure 10.130. Moses' sign.

The mechanism of producing sciatic pain by this test is unknown. It may be that knee flexion in the prone position stretches not only the high lumbar roots, but also, to a minimal extent, the lumbosacral roots; slight movements in the presence of severe nerve root compression could elicit sciatic pain. It is also possible that stretching of the lumbar plexus pulls on the sacral plexus through the interconnecting branches (165).

Yeoman's sign (Fig. 10.132). The test for Yeoman's sign is performed by applying pressure over the suspected sacroiliac joint to fix the pelvis to the table. The patient's leg, flexed at the knee, is hyperextended by lifting the thigh from the table. Increased pain in the sacroiliac is indicative of a lesion at that level.

Ely's heel-to-buttock sign (Fig. 10.133). The test for Ely's sign is performed by bringing the patient's heel to the opposite buttock by flexing the knee. Ely's sign identifies any irritation of the psoas muscle or a lumbosacral lesion.

Ely's sign also demonstrates contracture or shortening of the rectus femoris muscle. If contracture is present, the hip will flex and the buttock will rise from the table.

Prone knee flexion test (Fig. 10.134). Prone knee flexion provides provocative testing for lumbar disc protrusion (166). The pathophysiology of this test depends on compression of spinal nerves during hyperextension of the lumbar spine, which intensifies intervertebral disc protrusion into the spinal canal. Also, the lumbar intervertebral foramina are narrowed and the spinal canal cross-sectional area is decreased by lumbar

Figure 10.131. Nachlas' sign.

Figure 10.132. Yeoman's sign.

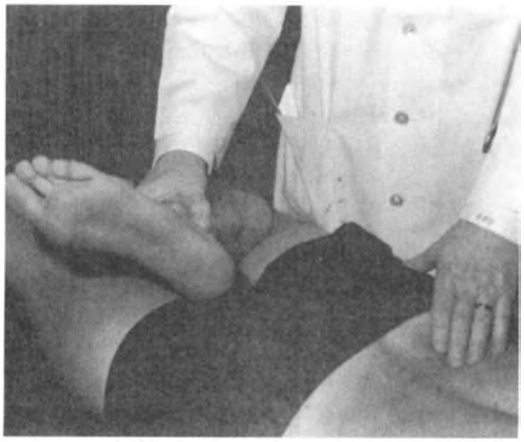


Figure 10.133. Ely's sign.

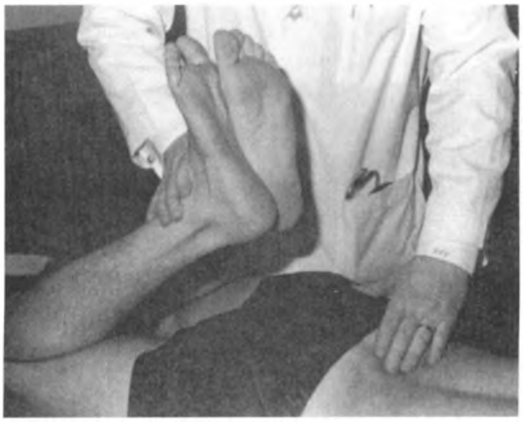


Figure 10.134. Prone knee flexion test.

Figure 10.135. Popliteal fossa pressure.

extension. Compression of a spinal nerve by lumbar disc protrusion may be intensified. Therefore, a protruded disc that has not produced sufficient neurocompression to cause weakness or reflex changes on testing with the spine normally aligned may be provoked by this test to produce changes that the examiner can elicit by testing in the prone knee flexion position.

The patient lies prone and the knees are hyperflexed, producing lumbar extension. The patient remains in the posture for approximately 45 to 60 seconds, and then the deep reflexes and muscle strength of the lower extremity are again evaluated. Weaknesses not observed prior to this maneuver may well be evident following it.

Popliteal fossa pressure (Fig. 10.135). In sciatica, the tibial branch of the sciatic nerve will be tender in the popliteal space on deep pressure, which is known as the "bowstring sign." According to Macnab (153), this is probably the single most important sign in the diagnosis of a ruptured intervertebral disc. The test for this sign can be performed with the patient in either the prone position (Fig. 10.135) or the supine position. With the patient in the supine position, the SLR is *performed until the patient experiences some discomfort. At* this level, the knee is allowed to flex and the patient's foot is allowed to rest on the examiner's shoulder. The test demands sudden firm pressure applied to the popliteal nerve. This action may startle the patient sufficiently to make him or her jump. Reproduction of pain in the leg or in the back is irrefutable evidence of nerve root compression.

Nonorganic Physical Signs (Malingering)

A patient with three or more of the following signs should be suspected of malingering. (For more information on psychological screening of patient, see the article by Waddell et al. [167]).

Libman's sign (Fig. 10.136). Deep palpation of the mastoid processes indicates the patient's pain threshold. Compare the patient's pain response to palpation of the mastoid pro*cesses to the pain response to examination of the low back. The* two of these pain sensitivities should be the same.

Tenderness to skin pinch (Fig. 10.137). With a pen lay out specific spinal segments on the patient's back. Then pinch the skin segment by segment, which should elicit pain in the pathway of the appropriate segment. If the patient complains of a generalized pain over many segments of the spinal nerve, symptoms are probably being exaggerated.

Mannkopf's sign (Fig. 10.138). Take the patient's pulse prior to deep palpation of a painful area. Such deep palpation should increase the pulse approximately 10 bpm if it is a true marked pain. If palpation does not accentuate the pulse, the patient may be exaggerating the symptoms.

Burns' bench sign (Fig. 10.139). Have the patient sit on a low stool and bend forward and touch the floor with the palms of the hands. If the patient claims not to be able to do this because of low back pain, suspect malingering, because flexion in this particular posture will not affect the low back specifically. Primary motion occurs at the hip joints and not the lumbosacral spine.

Flip test (Fig. 10.140). Have the patient sit on the examination table with the back straight and legs extended. If truly suffering from a disc lesion compressing the sciatic nerve, the patient cannot perform this test and will have to flex the knee or raise the hip from the table in order to relieve the sciatic stretch. If the test can be performed, the patient probably has no true sciatica or disc lesion and is malingering.

Figure 10.136. Libman's sign.

Figure 10.137. Tenderness to skin pinch.

Figure 10.138. Mannkopf's sign.

Figure 10.139. Burns' bench sign.

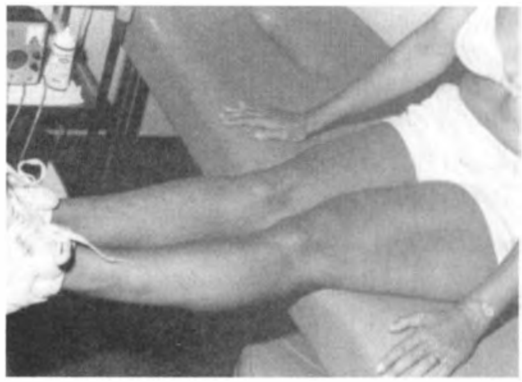


Figure 10.140. Flip test.

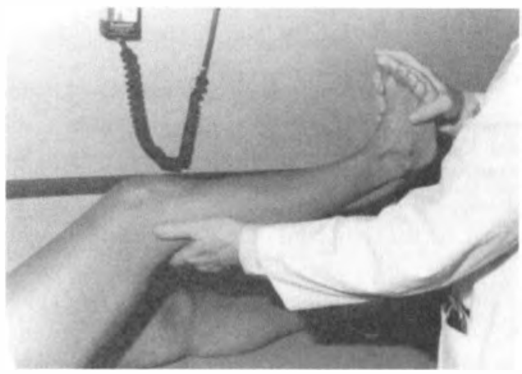


Figure 10.141. Plantar flexion test.

Plantar flexion test (Fig. 10.141). Ask the patient to raise the legs one at a time until low back or leg pain is felt. Note the angle at which the pain is elicited, and ask the patient to lower the leg. Then place one hand under the patient's knee and one under the patient's foot and raise the lower extremity, keeping the knee slightly flexed. Raise the leg to one half of the height at which pain was originally elicited and plantar flex the foot. If the patient says that this causes pain, suspect malingering.

Flexed hip test (Fig. 10.142). Place one hand under the patient's lumbar spine and the other under the patient's knee. Lift the knee, and if the patient claims to feels pain in the low back before the lumbar spine moves, suspect malingering.

Axial loading test (Fig. 10.143). Press the patient's cranium in a downward position. The axial loading may elicit pain in the neck but should not elicit pain in the low back. Suspect malingering if the patient says pain is felt in the low back.

Figure 10.142. Flexed hip test.

Figure 10.143. Axial loading test.

Rotation test of the shoulders and pelvis (Fig. 10.144). Have the patient turn the shoulders to rotate the entire spine. If complaint is made of low back pain, suspect malingering, because the patient is not truly moving the lumbar spine but rather is moving the spine from the thighs upward.

CORRELATIVE DIAGNOSIS OF LOW BACK PAIN

With the history and physical examination of the patient completed, including radiographic examination, findings can now be correlated.

Cox Clinical Classification of Low Back Pain Progression

The Cox system classifies back pain into 15 categories. Low back pain, in both its cause and progression, is well suited to placement in one (or a combination) of these categories. A description of each of these categories follows.

Category I—Anulus Fibrosus Injury

The patient with anulus fibrosus injury presents with the typical low back pain syndrome (i.e., the patient is young and usually on the first visit complains of low back pain following some flexion, twisting, or combined movement). Usually no leg pain is noted and relief is obtained within a few days. This type of pain can recur with progressive worsening of symptoms.

Clinically, the patient may present with muscle spasm, a loss of lordosis, and a positive Kemp's sign, but with no findings on the straight leg raising test and no altered motor or sensory changes of the lower extremity. Any leg pain is transient and not subjectively severe. Radiographs may reveal no change of discal space and no signs of discogenic spondylosis. This patient responds well to distraction manipulation and is usually satisfied with the clinical results.

The patient in category I has undergone tearing, cracking, or severe sprain of the anular fibers, causing irritation of the sinuvertebral nerve and resultant back pain. This patient is similar to the type I or type II patient described in the classifications of White and Panjabi and Charnley (31, 32).

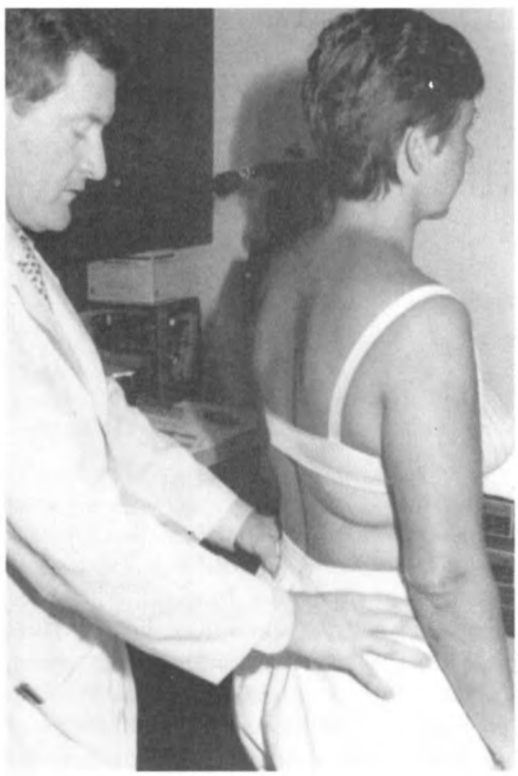


Figure 10.144. Rotational test of the shoulders and pelvis.

Category II—Nuclear Bulge

The patient with nuclear bulge presents with a worsening of low back pain and minimal leg pain.

Clinically, the patient may have paresthesias of the lower extremities but has no frankly altered deep reflexes. Findings include minimal irritation of the nerve root into the lower extremity, and demonstration of a more positive straight leg raising sign, Kemp's sign, and other orthopaedic tests for early disc protrusion. Déjérine's triad may increase the pain. Radiographs may show some early thinning of the disc space and discogenic and spondylitic changes, which may be minimal.

With prolonged exacerbation of low back and leg symptoms, the patient in category II requires a longer treatment period than does the patient in category I. At this stage, it is important that the patient wear a lumbosacral support to stabilize the low back for healing. Sitting must be strictly avoided to reduce the intradiscal pressure and allow the anulus to heal. Cox exercises to open the dorsal intervertebral disc space are most helpful at this time, and nutritional supplementation (Discat) may be incorporated into the treatment regimen.

The patient in category II shows progression of the tears and cracks of the anulus found in the category I patient, with the nucleus pulposus bulging into these anular fibers and causing further irritation of the sinuvertebral nerve and early and minimal irritation of the nerve roots that exit from the cauda equina within the vertebral canal.

Articular facets also become pain-producing entities because of disruption of the articular cartilage and fibrous capsule and the subluxation resulting from the loss of normal mobility of the motion segment. With increased intradiscal pressure or anular disruption, this patient is analogous to the type II or type III patient of Charnley's classification (32).

Category III—Nuclear Protrusion

The patient with frank nuclear protrusion may exhibit severe antalgia, marked lower extremity pain, and altered deep motor and sensory abnormalities.

Clinically, the patient demonstrates difficulty in straightening from a flexed position and a marked loss of lumbar lordosis. Radiographic studies show antalgia and possible discal change.

Depending on the medial or lateral relationship of the disc bulge to the nerve root, range of motion in the low back is markedly limited, and Kemp's sign is definitely positive.

The patient in category III requires prolonged treatment, and ambulation will be limited because of pain on weightbearing. It is mandatory that the patient wear a lumbosacral support and remain recumbent. At the outset of treatment, two or three visits per day may be necessary for maximal relief from pain. This patient is similar to Charnley's type IV classification (32).

Category IV—Nuclear Prolapse

The patient with nuclear prolapse primarily has lower extremity pain with minimal or absent low back pain. Nuclear material has completely torn through the anulus and lies within the canal as a free fragment severely irritating the nerve root and perhaps the cauda equina. The patient may have bowel and bladder problems. The decision regarding surgical treatment is based on the clinical differential diagnosis. If the patient does not show a 50% improvement within 3 weeks, surgery becomes imminent. This patient is analogous to Charnley's type V or type VI classification (32).

Category V—Discogenic Spondyloarthrosis

The patient with discogenic spondyloarthrosis (chronic advanced degenerative disc disease) has a history of intermittent low back pain (i.e., the patient is relatively free of pain except for acute exacerbations). The straight leg raising test is negative except for low back pain. Repeated motion of the spine, especially rotatory movements, causes low back pain. The patient must exercise care when bending and lifting. This patient is analogous to Charnley's type VII classification (32).

Category VI—Facet Syndrome

The patient with facet syndrome presents with hyperextension of the lumbar spine, which usually produces pain. Radiographs may well reveal a degenerative change of the facets, which follows degenerative disc disease. Macnab's line is positive. The work of Van Akkerveeken is important here to determine the stability of the facet syndrome. See Chapter 13, *Facet Syndrome,* for details on this diagnosis.

Category VII—Spondylolisthesis

Radiographic study is diagnostic in the patient with spondylolisthesis. See Chapter 14, *Spondylolisthesis,* for details on this diagnosis.

Category VIII—Lumbar Spine Stenosis

The patient with lumbar spine stenosis may present with symptoms of neurogenic intermittent claudication. For a full explanation of lumbar spine stenosis, see Chapter 4, *Spinal Stenosis.*

Category IX—Iatrogenic Back Pain

The patient with iatrogenic back pain, caused by either myelograms or surgery, suffers from irritation to the neural contents of the vertebral canal. The irritation is perhaps sufficiently severe to cause cauda equina symptoms. These patients are the most challenging to treat because of the difficulty in pinpointing the diagnosis and the consequent difficulty in arranging proper treatment. Many of these patients are failed back surgery syndrome (FBSS) patients whose biomechanics are so altered that relief from pain is difficult, if not impossible, to attain.

Category X—Functional Low Back Pain

The patient with functional low back pain often has personality aberrations and does not understand or will not understand the cause and treatment of low back pain. Sometimes emotional upset manifests itself through low back pain symptoms. Managing this type of patient is a challenge to both the surgeon and the nonsurgeon.

Category XI—Sprain and Strain

The patient with sprain or strain presents with an innocuous injury of nonrecurring frequency that seems to involve muscle

and ligament damage rather than discal or facetal damage. No nerve damage can be found. The pain may be present for several weeks following an athletic injury or automobile accident, but it is not chronic unless facet or disc damage has occurred.

Treatment consists of maintaining normal range of facet motion, restriction of motion in the early stages of injury, and rehabilitative exercises later.

Category XII—Subluxation

When a patient with subluxation presents with back pain, note the level and type of subluxation (e.g., a right lateral flexion subluxation of L5 on S1).

Category XIII—Tropism

In the patient with tropism, the level of asymmetry of the facet facings is marked. For a full explanation, see the discussion of tropism in Chapter 2, *Biomechanics of the Lumbar Spine.*

Category XIV—Transitional Segment

When a patient with transitional segment presents with back pain, ascertain whether there are 23 or 25 spinal segments to determine whether the patient has lumbarization or sacralization. See Chapter 6, *Transitional Segment,* for details on this diagnosis.

Category XV—Pathologies

Category XV is allowed for patients with any other pathology.

Establishing the Correlative Diagnosis

When the first three pages of the low back examination form (Table 10.7) are completed, the fourth page is used to arrive at a diagnosis within the 15 categories of low back pain causes just outlined. By following the *"Flow Chart for Correlative Diagnosis,"* findings are combined into a meaningful diagnosis of the patient's problem.

First, if the patient has sciatica, we use the algorithm at the top of the page entitled "Low Back and/or Leg Pain (Below Knee Diagnosis)." The dermatome involved, sciatic scoliosis, Déjérine triad, and leg pain intensity compared with the back pain are used to arrive at the side, type, and location of the disc protrusion to the nerve root compressed. The diagnosis will be either category III or IV disc lesion. Each of these findings has been covered in this chapter, so their meaning can be used to arrive at this clinical impression.

Second, under "Low Back Pain (No Leg Pain Below Knee) Diagnosis," the findings will flow into the other 13 categories of low back pain problems, as explained in this chapter or explained in other chapters in this textbook.

At the bottom of the last page is the "Correlative Diagnosis of Low Back Pain and Leg Pain." Here will be given the final diagnosis of disc and nondisc causes of back problems. In the treatment chapters, the use of these correlative diagnoses to establish the treatment regimen for the patient will be shown.

An example of a diagnosis, following the examination and completing the flow chart, might be "L5–S1 right medial disc protrusion with an unstable facet syndrome of L5 on the sacrum, a right lateral flexion subluxation of L5, and tropism of the L5–S1 facet joints."

Re-evaluation of Patient Response to Care

At least every 2 weeks after instituting distraction therapy, the patient's progress is re-evaluated. The following objective tests are repeated at this re-evaluation: straight leg raise (recumbent and supine), range of motion, Kemp's sign, deep tendon reflexes, motor testing, sensory testing, Déjérine triad, pain on palpation, and prone lumbar flexion. Subjective scoring is done by Oswestry, Roland Morris, visual analogue scale (VAS), and the Quebec disability score. VAS is scored for each subjective symptom (i.e., low back pain, leg pain, groin pain, and so on). This objective and subjective scoring allows modification of treatment plans, resetting of therapy goals, and detailed monitoring of patient progress.

SPECIAL DIAGNOSTIC CONSIDERATIONS

Disc Pain Distribution

The anulus fibrosus has nociceptive nerve endings in it (168), and therefore an anular tear can cause pain referral of purely discogenic origin into the low back, buttock, sacroiliac region, and lower extremity even in the absence of neural compression (17, 24).

Facet Joint Pain Distribution

The zygapophysial joints are well innervated, and facet arthropathy can cause low back pain and referred pain into the buttocks and lower extremities. Classic facet syndrome pain is in the hip and buttock, with cramping leg pain primarily above the knee, low back stiffness (especially in the morning with inactivity), and the absence of paresthesia. Classic signs are local paravertebral tenderness, hyperextension back pain, and no neurologic or root tension signs with hip, buttock, or back pain on straight leg raising.

Differentiating Disc from Facet Pain Distribution

Differential diagnosis of lower extremity pain of disc versus facet includes the fact that facet pain rarely extends beyond the calf, usually only into the thigh, and not into the foot. Radicular disc pain is potentially worse than back pain. In facet pain, the back pain is worse than the leg pain. Radicular pain is usually accompanied by neurologic signs in disc lesions but not in facet problems (169, 170).

Elevated Cerebrospinal Fluid Proteins

The protein concentration in the cerebrospinal fluid (CSF) is often increased in patients with sciatica, probably because of plasma proteins leaking through the blood–nerve root barrier into the cerebrospinal fluid. Significantly higher values of the

CSF:serum albumin ratio and the CSF:serum immunoglobulin G ratios were found in patients with positive SLR test results and paresis compared with patients with no clinical findings. Elevated CSF proteins seem to be an important indicator of the functional status of the nerve root and a measure of the degree of seriousness of sciatica (86).

Nerve root injury, as suggested by a positive straight leg raising test, appears to be neurochemically linked to altered CSF vasoactive intestinal peptide levels in patients with radicular pain symptoms caused by disc herniation and lumbar stenosis (171).

Differentiating Recurrent Disc Herniation from Scar Formation

Gradually increasing symptoms beginning a year or more after discectomy are considered more likely caused by scar formation, whereas a more abrupt onset at any interval after surgery is more likely caused by a recurrent herniated disc (172).

Symptoms and signs that best distinguish between recurrent herniation and fibrosus are pain on coughing, a severely reduced walking capacity, and a SLR test positive at less than 30°; the presence of two or more of these parameters was found in 16 of 22 patients with recurrent herniation compared with 5 of 18 patients with fibrosus (173).

Pathologic Change in Sciatic Foramen as Cause of Sciatica

Longstanding sciatic symptoms and signs should include pathologic changes in the sacral foramen by benign and malignant neoplasms as well as infection. CT scanning should include the sciatic foramen in longstanding, undiagnosed sciatica (174).

Dorsal Root Ganglion Compression Symptoms

Dorsal root ganglion compression can result in myalgia and tendinitis symptoms into the lower extremities (175) as well as intermittent claudication, sciatica, and groin pain (176).

Clinical Instability Defined

White and Panjabi (31) state that a narrowed disc space without spondylosis is a sign of instability. Clinical instability is defined as the loss of the spine's ability, under physiologic loads, to maintain normal relationships between vertebrae so that no damage and no subsequent limitation to the spinal cord or nerve roots occurs and no incapacitating deformity or pain develops from structural change.

Differentiating Contained from Noncontained Disc

When a disc lesion is present, a differential diagnosis between protrusion and prolapse is necessary. The sudden onset of leg pain and absence of low back pain indicate prolapse (category IV), whereas low back pain followed later by leg pain indicates protrusion (category III).

Sciatic Scoliosis Defines Disc Lesion Type

Relief of pain on lateral flexion may indicate whether the disc protrusion is lateral or medial to the nerve root (21) (Fig. 10.145).

Cervical Disc as Cause of Myofascitis and Leg Pain

Cervical disc herniations have been reported to cause myofascial pain and altered deep reflexes in the lower extremities; the myofascial pain caused by this irritation ceased once the mechanical cervical disc rubbing of the cord was surgically relieved (177).

Leg Length Effect on Low Back Pain

Leg length inequality alters gait efficiency and predisposes to low back pain and hip arthrosis (178).

THORACIC DISC HERNIATIONS

Pain on Side Opposite Herniation

A 37-year-old hospice nurse was evaluated for left midthoracic pain, and an MRI revealed a large right-sided thoracic disc herniation at T7-T8, with a moderate degree of cord compression.

All signs and symptoms need not necessarily occur on the side of the lesion. Thoracic disc herniations can cause neural compromise by direct compression or by an indirect effect, secondary to arterial and venous thrombosis. The dentate ligaments may also resist posterior displacement of the cord, leading to traction and distortion of the neural structures (179).

Thoracic disc herniations, which occur in less than 4% of all disc herniations, should be included in the differential diagnosis of patients with paresthesias and weakness of the lower extremities. Up to 70% of thoracic disc herniations have been found to calcify compared with 4% of normal studies (180).

Brennan (181) reported that thoracic disc herniation is uncommon in adults, comprising only 0.25 to 0.75% of herniations. Although it is extremely rare in children, he did present a paraparesis in an 11-year-old boy following minor trauma, which on MRI was found to be caused by to a T4-T5 small herniation. The appearance was normal on myelography and CT. Laminectomy revealed disc material adherent to the dura with postsurgical need of left knee-ankle-foot orthosis at discharge.

UPPER LUMBAR DISC HERNIATIONS—DIAGNOSTIC CHALLENGE

Presentation, diagnosis, and outcomes of upper lumbar disc herniations (L1–L2, L2–L3, L3–L4) are variable and difficult. Preoperative signs and symptoms are highly variable, as are sen-

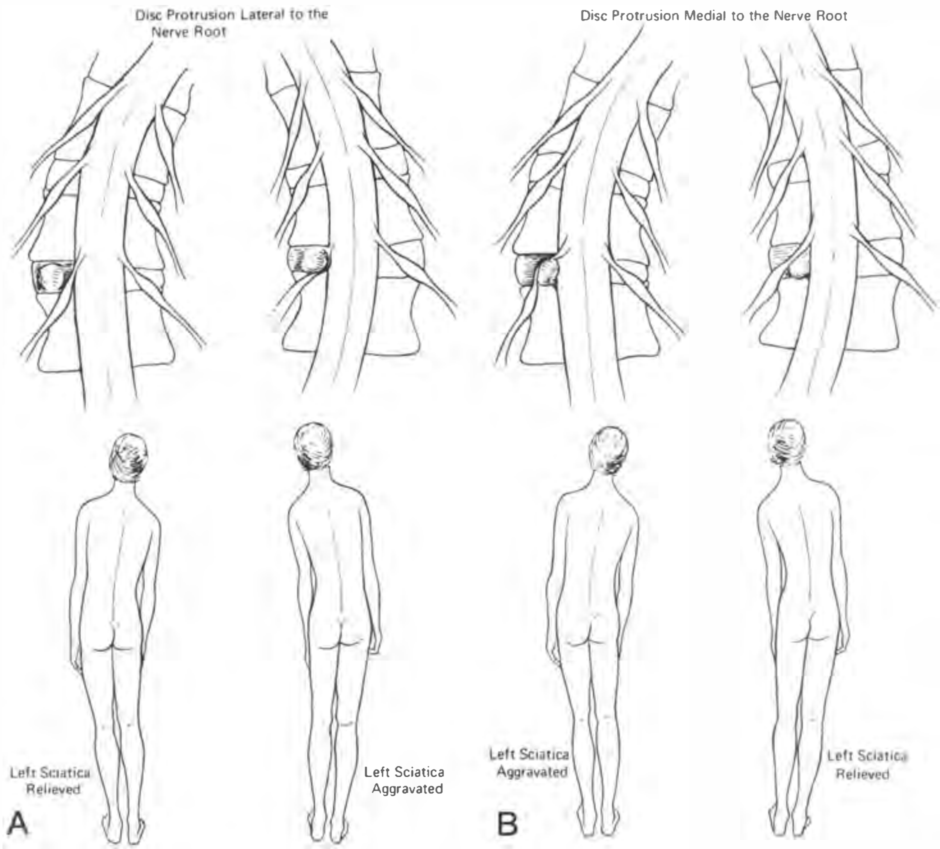


Figure 10.145. Sciatic scoliosis in a disc lesion. (Reprinted with permission from Finneson BE. Low Back Pain. 2nd ed. Philadelphia: JB Lippincott, 1980:302.)

sory, motor, and reflex testing, which can be potentially misleading in suggesting a level of herniation. Sensory, motor, and reflex deficits are weak predictors of the level of disc herniation. In analyzing radiographic studies (noncontrast CT, myelography, MRI) individually and using other radiographic studies and operative findings as a standard for comparison, a high false-negative rate is found for all studies when considered individually, especially at the higher L2–L3 level (182).

Recommended is postmyelogram CT and/or MRI in the workup of these patients, and intraoperative radiographs in all cases of decompressing upper lumbar disc herniation. Consider the differential possibilities of retroperitoneal tumor or hemorrhage, abdominal aortic aneurysm, diabetic femoral neuropathy, or lumbar plexopathy in the workup (182).

The sensitivity of CT scan at the L2–L3 level is 71% and at the L3–L4 level, 72%. For myelogram, the sensitivities are 50% at the L2–L3 level and 80% at the L3–L4 level. The sensitivities of MRI were found to be 75% at the L2–L3 level and 90% at the L3–L4 level (182).

Noncompensation patients had a significantly higher percentage of good or excellent results (86%) than those with compensation or legal claims pending (45% good or excellent results).

Upper lumbar disc involvement, with or without thoracic disc pathology, may be higher than previously reported. Many patients with upper disc pathology also have lower disc involvement, suggesting that upper disc pathology should be sought out in patients experiencing low back pain. The low level of suspicion continues to be the major difficulty in the diagnosis of thoracic spine disc pathology or high-level lumbar disc pathology (183).

Crossed Femoral Nerve Stretch Sign

A case is reported of L3–L4 far lateral disc herniation, in which the femoral stretching and crossed femoral stretching tests were positive. It is hypothesized that the crossed femoral stretching test may be a valid maneuver to help in the diagnosis of symptomatic disc herniation above L4 (184).

FAR LATERAL HERNIATED LUMBAR DISC HERNIATION

Age and Level of Occurrence

1. Far lateral herniated nucleus pulposus (HNP) occurs in older individuals more often than does the classic posterolateral HNP.
2. In far lateral disc herniations, 92% occur at L4–L5 or L3–L4, whereas 90% of posterolateral herniations occur at L4–L5 and L5–S1.
3. When the patient is initially seen, a more proximal root involvement is seen compared with that in classic posterolateral HNP (185).

Location and Appearance of Foraminal Lumbar Disc Herniations

Eighty-three patients were evaluated by CT and/or CT discography and operated on for foraminal lumbar disc herniation. Location and appearance of disc herniations are shown in Figures 10.146–10.148.

The reported incidence of foraminal disc herniation varies from 1 to 10%. Most far lateral disc herniations occur at the L3–L4 and L4–L5 levels, but in the study cited here, 35% of the patients had herniations at the L5–S1 level (186).

Clinical Picture of Foraminal Disc Herniation

The clinical picture of foraminal disc herniation is somewhat different from that of the usual disc herniation, especially for neurologic signs of root compression. Biradicular symptoms and neurologic signs of root compression were more frequent with foraminal herniations. Radiculopathy severity has been accredited to direct contact of the herniation with the posterior root ganglion. Figure 10.149 shows the clinical findings of foraminal disc herniation.

Postoperative Results in Treatment of Foraminal Disc Herniation

Postoperative results were good in 76% of the patients who received surgical treatment for foraminal disc herniation. The other patients felt mild residual radicular pain, although no residual root compression was found on postoperative CT. Only 21% of the patients who had a radicular deficit recovered totally.

Most foraminal lumbar disc herniations are reached through the interlaminar exposure extended to the upper lamina and medial facet without total facetectomy. An extra-articular approach should be reserved for extraforaminal herniations.

Foraminal herniations may be overlooked because of their low frequency among lumbar disc herniations and because even a moderate bulge of the disc may impinge the nerve root in the narrow space of the intervertebral foramen (186).

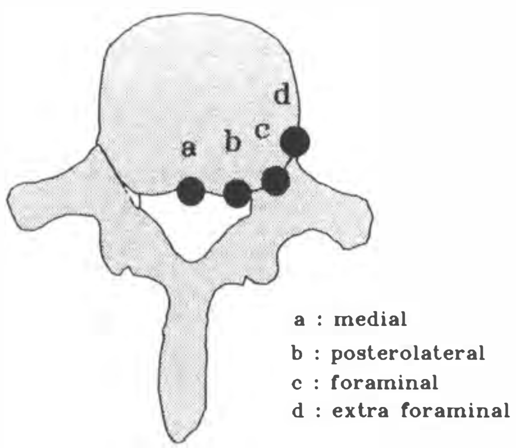


Figure 10.146. Classification of lumbar herniated discs. (Reprinted with permission from Lejeune JP, Hladky JP, Cotten A, et al. Foraminal lumbar disc herniation: experience with 83 patients. Spine 1994;19(17): 1905–1908.)

Extraforaminal Disc Herniation

Discography-CT was found to be accurate and useful in differentiating extraforaminal from foraminal lumbar disc herniation, even when "state-of-the-art" neuroradiologic postmyelographic CT failed. Because the lumbar nerve root sheath terminates near the dorsal root ganglion within the intervertebral foramen, disc herniations lateral to this foramen escape myelographic recognition. An accurate preoperative diagnosis, established by discography-CT if necessary, followed by a minimally invasive surgery is an effort to minimize surgical trauma and to expedite rehabilitation of the patient (187).

Extraforaminal Disc Prolapse Can Masquerade As a Nerve Sheath Tumor

A patient presented with an L3 radiculopathy in whom MRI demonstrated what appeared to be a nerve sheath tumor in an extraforaminal location on the L3 nerve root. A lateral inter-

Figure 10.147. Computed tomography at the L3–L4 level shows foraminal disc herniation. (Reprinted with permission from Lejeune JP, Hladky JP, Cotten A, et al. Foraminal lumbar disc herniation: experience with 83 patients. Spine 1994;19(17):1905–1908.)

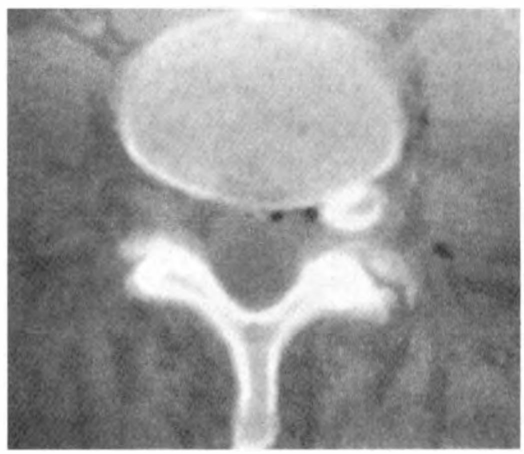


Figure 10.148. Computed tomographic discography demonstrates contrast extravasation in the left L5 foramina. (Reprinted with permission from Lejeune JP, Hladky JP, Cotten A, et al. Foraminal lumbar disc herniation: experience with 83 patients. Spine 1994;19(17):1905–1908.)

Clinical Findings in the Present Series of 83 Foraminal Herniations Compared With a Series of 100 Posterolateral Herniations

	No. of Patients Foraminal	No. of Patients Posterolateral
Biradicular symptoms	34	11
Motor weakness	40	15
Sensory impairment	42	29
Total relief of radicular pain after surgery	59	86

Figure 10.149. Clinical findings of foraminal disc herniation. (Reprinted with permission from Lejeune JP, Hladky JP, Cotten A, et al. Foraminal lumbar disc herniation: experience with 83 patients. Spine 1994;19(17):1905–1908.)

muscular approach was used to excise the lesion to preserve the facet joint. Histologic examination of the intraneural lesion revealed degenerative disc fragments. The structure of the anulus fibrosus in the upper lumbar region predisposes these regions to lateral herniation. Furthermore, it is proposed that the lateral disc herniation allowed the disc fragments to erode through the epineurium of the neural sheath. This case expands the differential diagnosis of fusiform enlargement of nerves to include disc herniation (188).

Case 6

A 36-year-old man suffered severe left anterior thigh pain of 1 month's duration. Quadriceps weakness, absent patellar reflex, hypoesthesia on pinwheel of the anterior thigh, and agonal type pain causing the thigh and knee to be flexed and held to the chest for relief was observed. Sleep, ambulation, and work were impossible.

Figure 10.150 is a CT scan performed prior to my seeing this patient. It shows an L4–L5 left extraforaminal disc prolapse (*arrow*). From this CT scan, surgery was recommended to remove the L4–L5 fragment.

Severe pain and indecision on the patient's part prompted a second opinion from me. MRI was ordered to include the entire lumbar spine, whereas the former CT was done from L3–L4 to L5–S1 only. Figure 10.151 shows the L4–L5 intraforaminal and extraforaminal prolapse (*arrow*).

However, in Figure 10.152, at the L2–L3 level (*arrowhead*) is shown a large free fragment located extraforaminally and lying within the osseoligamentous canal. Note that the dorsal root ganglion on the opposite side (*arrow*) is well visualized, whereas on the involved side it is obliterated by the disc fragment.

Figure 10.153 is the sagittal image which was invaluable also. It shows the fragment within the L2–L3 canal, which is filling most of it (*arrow*).

This case is a good example to teach the chiropractic physician, or any other physician, to look carefully at all possible levels of the lumbar spine for the location of disc compression of nerve root or dorsal root ganglion. The femoral nerve root origin of the patient's pain could have been the L4–L5 disc prolapse compressing the L4 dorsal root ganglion and nerve root. In this case, surgery performed at the L4–L5 level would have resulted in a patient not receiving relief of the left femoral radiculopathy with neurologic complications. Surgery to relieve the L2–L3 disc prolapse resulted in complete relief for this patient.

Intradural Disc Herniation

Intradural disc herniation is a rare disorder that occurs most often at the L4–L5 level in middle-aged men. The symptoms are severe and generally follow an acute event such as lifting. Persons with previous spinal surgery are more at risk. The preoperative diagnosis is difficult, and surgery is indicated to alleviate symptoms and relieve the neurologic deficit (189).

Thermography

Supporters of thermography state that (*a*) normal patients have normal thermograms of their lower extremities, and (*b*) patients with abnormalities (e.g., disc ruptures causing sciatica) have abnormal thermograms. The specificity of thermography (its ability to be negative in asymptomatic patients) was 45 and 48% for testing thermographers. Thermography is not useful as a diagnostic aid in sciatica (190), although this is an area of controversy.

Pressure Algometers

Pressure algometers are instruments that measure the amount of force (pressure) that induces pain or discomfort. The measure of pressure threshold (PTH) is simple and it can be accomplished in a few minutes. First the patient is asked to point with one finger to where the maximal pain is felt. The examiner palpates the area with his or her fingertip to identify precisely the maximal pain area—the most tender spot—and marks it. The meter is applied to this point, perpendicularly to the muscle surface, and the pressure is increased continuously at a rate of 1 kg/sec until the patient starts to feel pain. A 2 kg/cm^2 side-to-side difference in pressure threshold is considered abnormal.

Algometry assists the health practitioner in the crucial decision, namely, how much pressure sensitivity is abnormal and

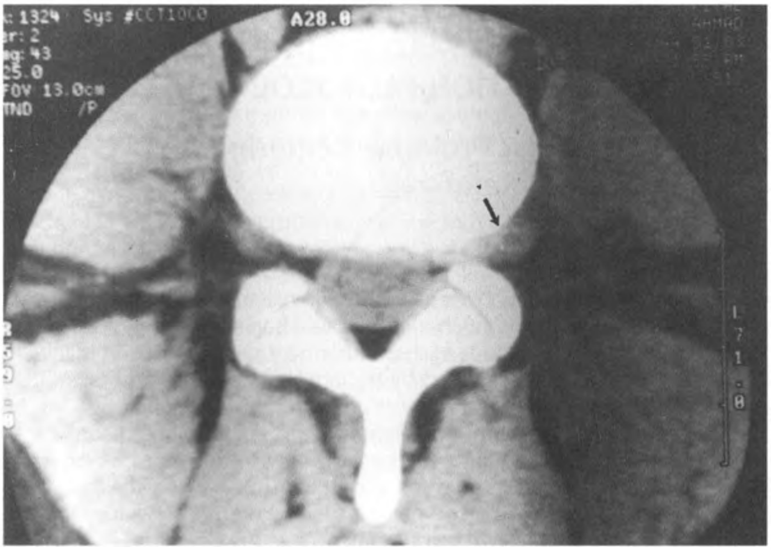


Figure 10.150. Computed tomography scan at the L4–L5 level shows an extraforaminal fragment (*arrow*).

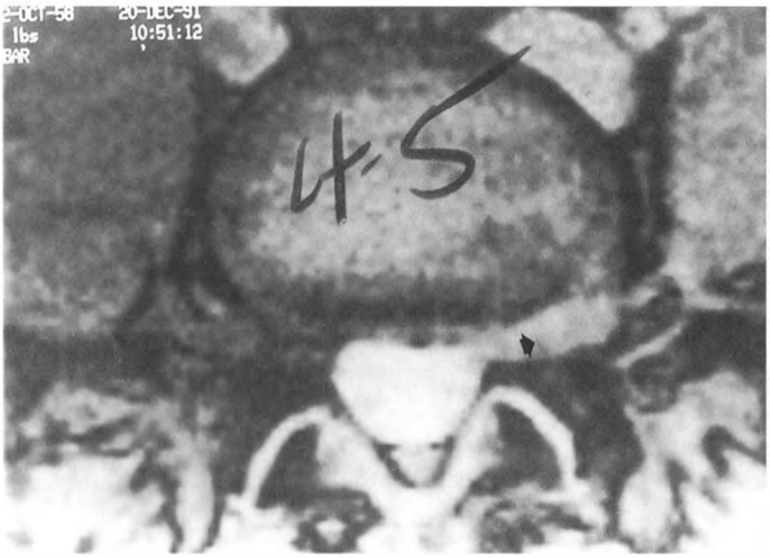


Figure 10.151. Magnetic resonance image shows the L4–L5 extraforaminal fragment (*arrowhead*) as seen in the computed tomography scan shown in Figure 10.150.

Figure 10.152. Axial magnetic resonance image at the L2–L3 level shows the left huge free fragment (*arrowhead*). Note the dorsal root ganglion on the opposite noninvolved right side is normal (*arrow*), whereas on the left involved side it is engulfed with the disc sequestration.

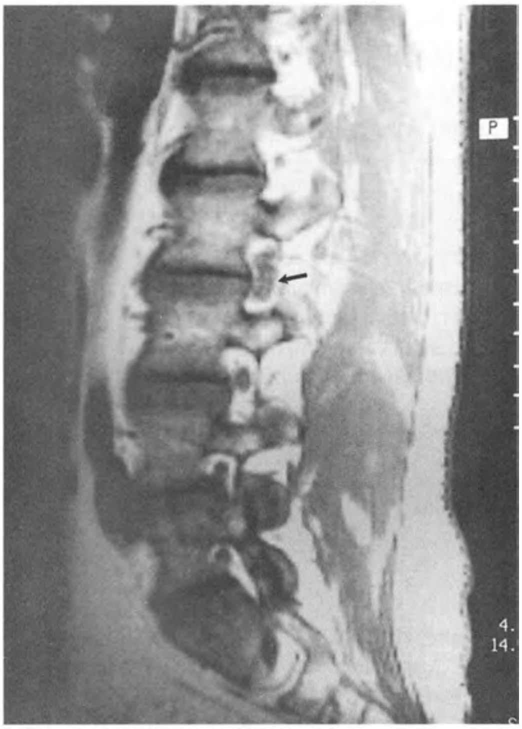


Figure 10.153. Sagittal magnetic resonance image shows the L2–L3 foraminal disc fragment *(arrow)*.

how much is diagnostic of trigger points, tender points, fibromyalgia, and muscle and joint dysfunction.

The tissue compliance meter (TCM) is a clinical mechanical instrument that consists of a force gauge ranging to 5 kg with a long shaft, which is fitted with a 1 cm^2 rubber disc. When the rubber disc is pressed into the examined tissue at a known force a disc fitted around the long shaft of a force gauge slides up indicating the depth of penetration, on a scale attached to the shaft. Normal values for TCM have been established and the reliability and reproducibility of results have been proved. Muscle spasm has been defined as a sustained involuntary, usually painful contraction, that cannot be alleviated completely by voluntary effort. The tissue compliance meter is the only clinical method that can objectively document the presence of a soft tissue abnormality (191).

Obturator Nerve Neuralgia

Two cases of obturator neuralgia, both affecting L1 roots by L1–L2 disc herniations were reported. L1 root compression can induce obturator neuralgia, and disc herniation should be included in the cause of obturator nerve palsy and obturator neuralgia, a fact not previously reported (192).

Piriformis Syndrome

Sciatica could be caused by a piriformis syndrome. In 10% of people, the sciatic nerve passes between the two parts of the tendinous origin of the piriformis muscle and internal rotation of the thigh compresses the sciatic nerve (193).

CASE PRESENTATIONS OF TYPICAL DIAGNOSES MADE USING AUTHOR'S EXAMINATION PROTOCOL

L5-S1 Disc Prolapse Requiring Surgical Removal

Case 7

A 28-year-old, well developed white man was seen who had suffered from low back pain off and on over the last 2 years. He had been treated by a chiropractor and had some relief, but the pain had reached a point where treatment did not result in relief. The patient was examined by his family doctor, who prescribed pain pills. He consulted another chiropractor, who, on seeing his low back, left S1 dermatome sciatica and severe antalgic lean with an accompanying limb, referred the patient to us.

Examination revealed a positive Cox sign on the left at approximately 30°. The patient walked with an obvious left limp, and the ankle jerk on the left was absent. Sensory examination revealed hypesthesia over the left S1 dermatome into the small toe side of the foot. An outstanding finding in this patient was

Figure 10.154. CT shows left disc protrusion of the L5–S1 disc *(arrow)* in a 28-year-old male with left S1 dermatome sciatica, an absent ankle reflex, and a marked right antalgic sciatic scoliosis.

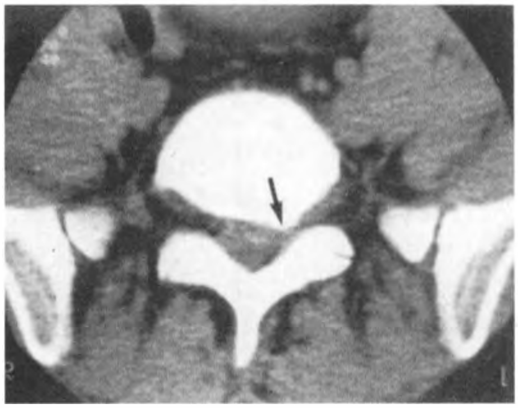


Figure 10.155. Another computed tomography cut at L5–S1 shows L5 inferior body plate hyperostotic bone exostosis *(arrow)* narrowing the left lateral recess and intervertebral canal sagittal diameter.

the gluteal skyline sign, as the left buttock hung well over 2 inches inferior to the right, with a marked flaccidity of the muscle on strength examination. Both the gluteus maximus and hamstring muscles were grade 4 of 5 strengths.

Because of the marked motor loss, the severe pain to the patient, the absent left ankle jerk, and the fact that prolonged chiropractic treatment had rendered no relief, the decision was made to send this patient for a CT scan (Fig. 10.154), which revealed a large L5–S1 disc protrusion on the left. An exostosis of bone on the left inferior L5 vertebral body plate was evident (Fig. 10.155).

Figure 10.156 shows the myelogram in the posteroanterior (PA) projection, and Figure 10.157 shows the oblique myelogram demonstrating the massive L5–S1 disc prolapse that is compressing the cauda equina and S1 and S2 nerve roots.

At surgery, this free fragment of disc material measured 3 cm by 1.5 cm. The patient had a good relief of sciatic pain and total return of motor power following this surgery.

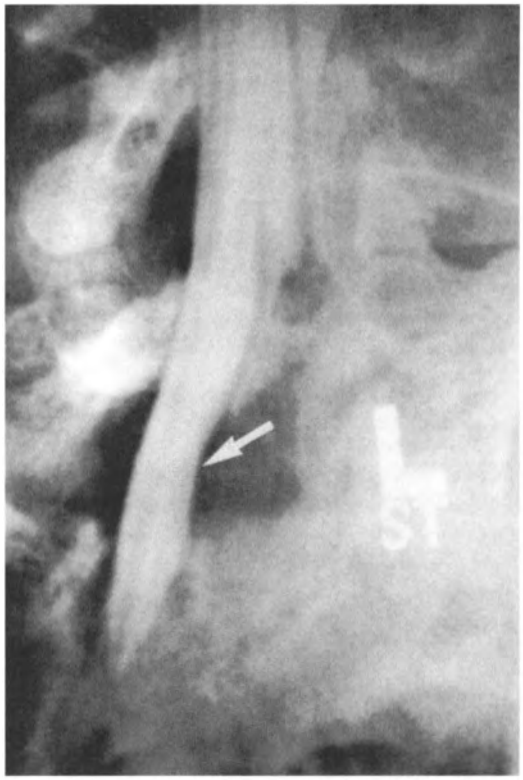


Figure 10.157. Oblique myelogram shows the defect into the myelographic dye column (*arrow*) by the disc protrusion at L5–S1.

L4–L5 Disc Protrusion with Foot Drop Treated With Manipulation

Case 8

A 44-year-old woman was seen complaining of 4 days of deep low back and right hip pain, which started following a sneeze. She stated that she felt better the following day, but the day before we examined her, she became markedly worse, and the pain radiated into the foot and into the sulcus of the toes.

Examination revealed pain at the L4–S1 levels. The right buttock, thigh, and anterolateral leg were painful to palpation. The straight leg raising sign was positive at 50° on the right, and the right ankle jerk was absent. However, the history revealed that 15 years previously this patient had had right sciatic pain and a rupture of the L5–S1 disc that had caused loss of the ankle jerk.

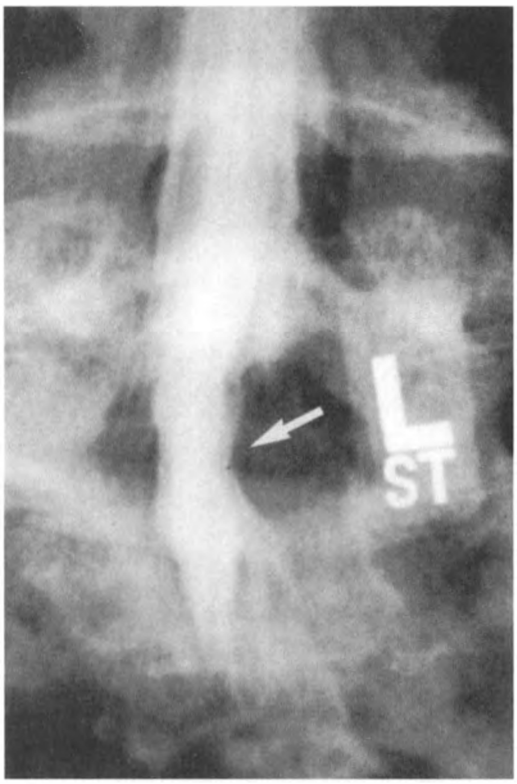


Figure 10.156. Posteroanterior myelographic study of the computed tomography-scanned patient seen in Figures 10.154 and 10.155 shows the large left filling defect into the dye-filled subarachnoid space by the large disc protrusion at L5–S1 (*arrow*).

The following day, the patient stated that she felt some relief in the right hip but that now the top of the foot had started to hurt. Three days later, the patient's condition had worsened until the SLR became positive on the right at 30°, with Braggard's maneuver positive. The left SLR was negative. Dorsiflexion weakness was now observed in the right great toe and foot at the ankle. The hamstring reflexes were +2 bilaterally. The ankle jerk on the right was still absent. The Déjérine triad was negative. The patient now had no low back pain, only leg pain.

Our impression 3 days following the first visit was that this patient had an L4–L5 disc prolapse and perhaps an L5–S1 extreme lateral disc lesion. Because of this dilemma, a CT scan was ordered that day. Figures 10.158 and 10.159 show the CT scan. A large osteophytic spur was seen from the posterior central vertebral body plate into the vertebral canal at L5–S1 in Figure 10.158. The radiologist felt that this was a probable cause of the patient's symptoms. The CT scan at the L4–L5 level did show a small disc asymmetric bulge on the right side (Fig. 10.159). Figures 10.160 and 10.161 reveal small myelographic filling defects at the L4–L5 level.

My impression was that the patient was suffering from an L4–L5 nuclear disc protrusion compressing the L5 nerve root causing radiculopathy into the right leg. The large osteophytic spur, in my evaluation, had probably been there for many years and was a result of an old anular irritation from the previous L5–S1 disc protrusion that had been treated years previously. We felt that the large osteophyte at the L5 level was really of no consequence at that time.

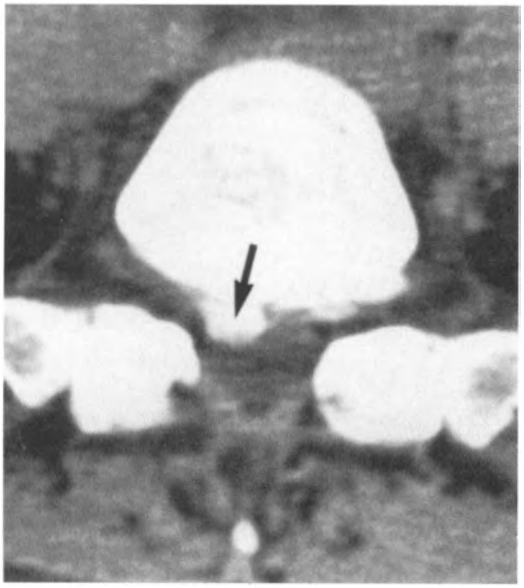


Figure 10.158. Axial computed tomography slice at the L5–S1 level shows a right posterolateral hypertrophic spur into the lateral recess and vertebral canal (*arrow*).

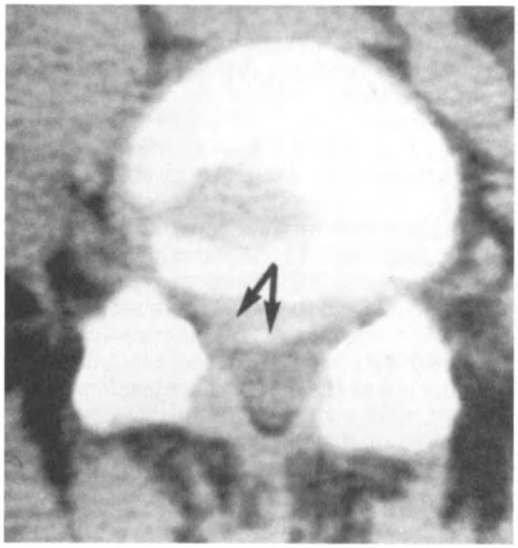


Figure 10.159. L4–L5 axial computed tomography slice shows a right central disc protrusion into the lateral recess (*arrow*).

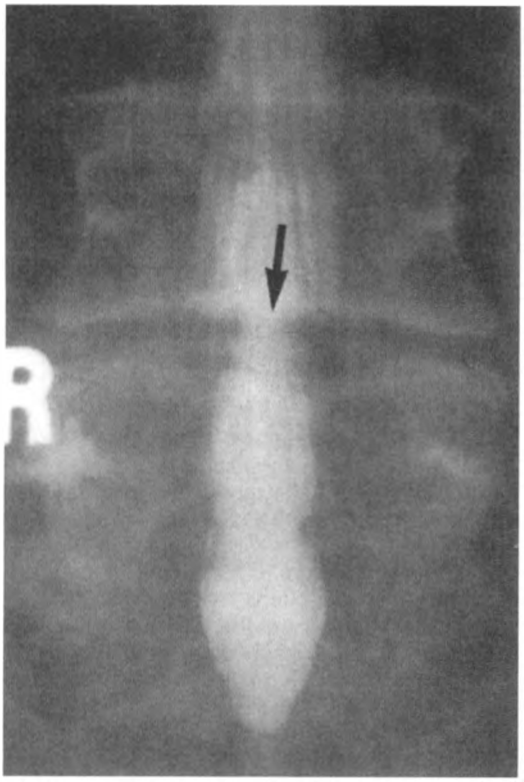


Figure 10.160. Myelogram posteroanterior view shows a minimal narrowing of the dye-filled subarachnoid space at the L4–L5 level (*arrow*).

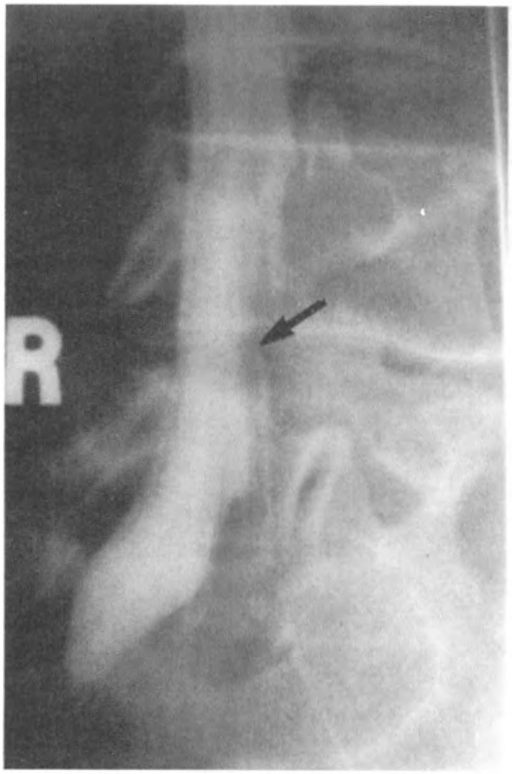


Figure 10.161. Left anterior oblique view reveals compression of the L5 nerve root by an L4–L5 disc protrusion (*arrow*).

Treatment was given consisting of flexion distraction at the L4–L5 disc level. Positive galvanism was applied over the L5–S1 disc, as well as over the course of the sciatic nerve and the buttock and popliteal space. Alternating hot and cold packs were applied to the spine.

This treatment resulted in gradual relief of the pain and the return of dorsiflexion strength in the right leg. At 6 weeks, the patient was able to walk on the heels and dorsiflex the great toe on the right.

This case is an excellent example of one in which the doctor could be misled by the large osteophyte at the L5 level that really was of no pathologic significance to the patient's symptoms at that time. The osteophyte had been there for many years before the present complaints. It may also be that the degeneration of the L5–S1 disc had shifted the movement to the L4–L5 disc and it was now placed under enough stress to lead to the new anular tearing and fresh disc bulge.

This case also shows that careful clinical correlation of the radiographic and examination findings is absolutely necessary to arrive at the proper conclusions. Further, in a patient with foot drop, one must be especially cognizant of the compression of the L5 nerve root. If this patient found the pain to continue for up to 1 or 2 weeks, with progressive weakening on dorsiflexion, a referral for a neurosurgical consultation would have been made. The doctor must be sensitive to the fact that dorsiflexion can be a permanent impairment if allowed to prevail too long before the nerve root is decompressed. Such dorsiflexion problems may well be a source of medicolegal trouble to a doctor. A word on this certainly should be sufficient to make the doctor aware that a case with dorsiflexion weakness is a good case to observe very closely and to get a second opinion.

L4–L5 Disc Prolapse Surgically Removed

Case 9

A 42-year-old single man, suffering from cerebral palsy, was seen complaining of low back and right leg pain with occasional pain into the left leg. This pain started 5 months previously following sleeping on a soft couch, after which he bent down to pick something up and felt immediate back pain. Two months after the injury, he developed severe right leg pain and minimal left leg pain. Approximately 1 month later, an MRI was performed with a diagnosis of an L4–L5 herniated disc and a possible L5 right herniated disc. He was treated with physical therapy for an additional 3 weeks and then sought care at our office.

Figure 10.162 is a picture of this patient standing upright, and Figure 10.163 is a PA radiograph showing the left antalgia of the thoracolumbar spine. When first seen, this patient was walking with a walker.

Physical, orthopaedic, and neurologic examination results were as follows: There was inability to lie down for the SLR examination. Ranges of motion were limited to 75° flexion, 0° right lateral flexion, 15° left lateral flexion, and 25° extension. The right ankle reflex was +1 and the left was +2, and the patellar reflexes were +2 bilaterally. Hypesthesia was present in the right S1 dermatome. Two days later, when able to die down, the patient's SLR was positive on the right at 35° and on the left at 65°.

This patient was placed on a treatment regimen that involved staying in our clinic and maintaining recumbency throughout the day to receive flexion-distraction treatment, and receiving physical therapy in the form of positive galvanism into the L4–L5 and L5–S1 disc, acupressure point treatment, and alternating hot and cold packs to the low back and the right lower extremity.

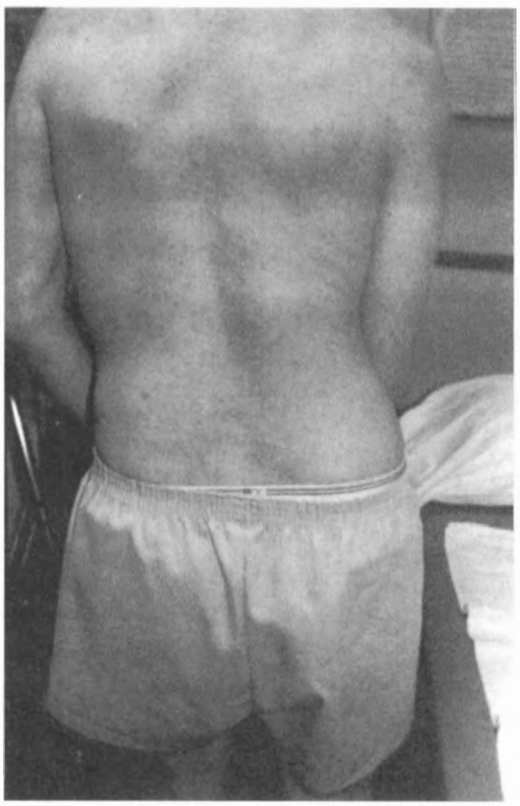


Figure 10.162. Left sciatic scoliosis of a patient with right sciatic radiculopathy.

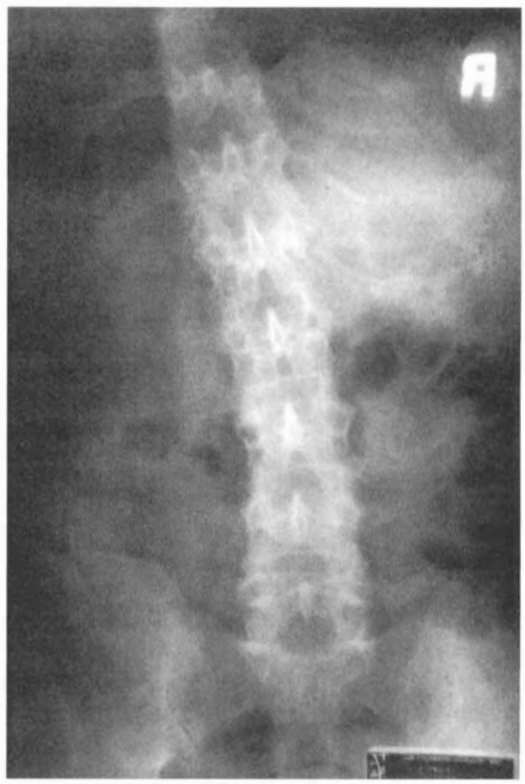


Figure 10.163. Left sciatic scoliosis of the spine of the patient shown in Figure 10.162.

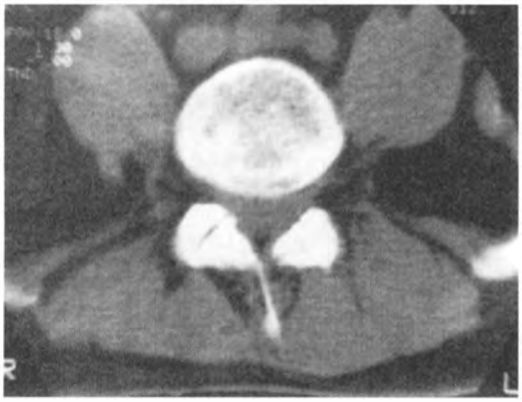


Figure 10.164. Axial computed tomography scan fails to reveal a definitive disc prolapse.

Treatment did not yield 50% relief within 3 weeks of care, and a CT scan was then ordered on this patient (Fig. 10.164). This scan was interpreted as showing a possible L4–L5 disc herniation, and a myelogram was recommended for further evaluation. The myelogram in Figures 10.165 and 10.166 reveals an extremely large extradural defect at the L4–L5 posterior disc space that creates a marked filling defect into the dye-filled subarachnoid space. A huge free fragment at the L4–L5 disc space on the right side which was underlying the L5 nerve root was surgically removed, and the patient had excellent relief of pain.

Following relief of pain, a pelvic radiograph was taken to evaluate femoral head height, because the patient continued to show a marked right short leg. This x-ray study (Fig. 10.167) reveals a 30-mm short right femoral head. Figure 10.168 shows a 15-mm lift placed under the patient's short right leg, which actually is an overcorrection. Ultimately a 9-mm lift was placed under the right heel and sole, which leveled the femoral heads. This combination of treatment gave this patient total relief from his low back and sciatic pain.

Extraforaminal Disc Prolapse Surgically Removed with Complications

Case 10

This case is from the records of David Taylor, DC, and it represents a case of the "far out syndrome" in which a free fragment of disc was found to have extruded into the intervertebral foramen on the left side. Figures 10.169 and 10.170 represent the PA and oblique views at the L4–L5 level following facetectomy to remove the free fragment of disc. Note that the left L4–L5 facets have been surgically removed. It actually appears as if discitis had occurred following surgery, but certainly a left lateral flexion subluxation is seen with extreme vertebral body plate sclerosis and irregular outline of the inferior L4 and superior L5 vertebral body plates. Note the marked hyperostosis of the bone margins of L4 and L5. This patient still has extreme low back and leg pain following surgery.

This is a good example of the removal of facets and the accompanying collapse of the intervertebral disc on the side of facet removal.

Disc Degeneration May Be Nutritional

Case 11

A 32-year-old woman has left first sacral dermatome sciatica. Figures 10.171 and 10.172 are sagittal T2-weighted and axial T1

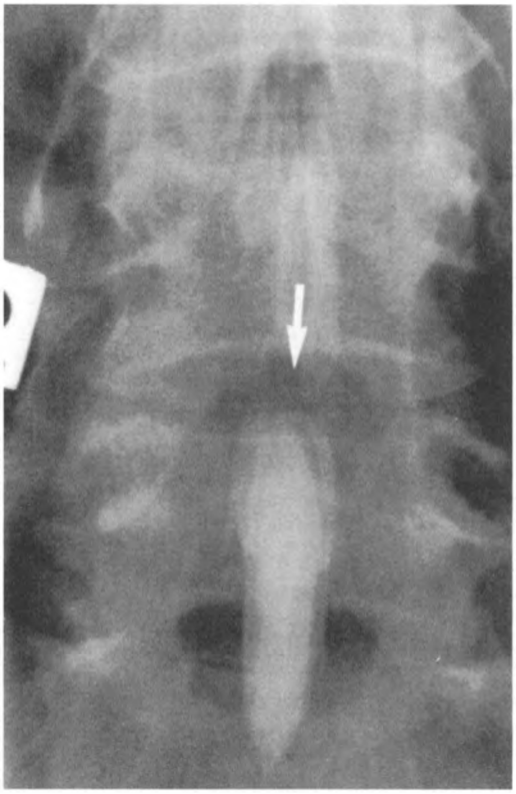


Figure 10.165. Posteroanterior myelogram reveals a large compression filling defect of the cauda equina at the L4–L5 level (*arrow*).

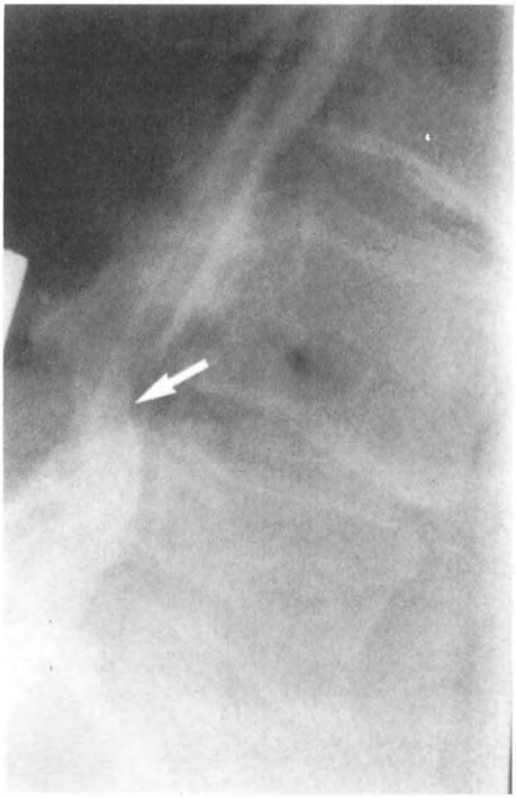


Figure 10.166. Lateral myelogram reveals flexion subluxation of L4 on L5 with an anterior defect of the dye-filled subarachnoid space (*arrow*).

Figure 10.167. The right femoral head is 30 mm inferior to the left on this upright Chamberlain's view taken to evaluate leg length deficiency.

Figure 10.168. A 15-mm lift under the right heel and sole levels the femoral heads.

images showing both L4–L5 and L5–S1 discs to be hypointense on sagittal image. It has been stated that disc degeneration is also a systemic disease, meaning it has a nutritional basis, which could explain the multiple level disc degeneration so often seen as opposed to single level degeneration. Perhaps the reason so many patients are seen with more than one disc showing degenerative change while only having one disc herniated, is because of the systemic lack of glycosaminoglycan coupled with the fact that the lower discs are required to perform the greatest degree of flexion and extension movement, while rotation movement seemingly places great stress on these discs as well.

Figure 10.172 reveals a left central disc herniation that contacts the left S1 nerve root and mildly contacts the thecal sac (*arrow*). Again, the importance of this case is that it shows the degenerative change not of just the disc that is herniated, but rather the disc adjacent to it as well. It has long been felt that the increased stress on the adjacent disc by the shift of motion and stress by the degenerating disc leads to degeneration. However, we must be aware that disc disease is considered to be a systemic disease as well as a traumatic event of stress.

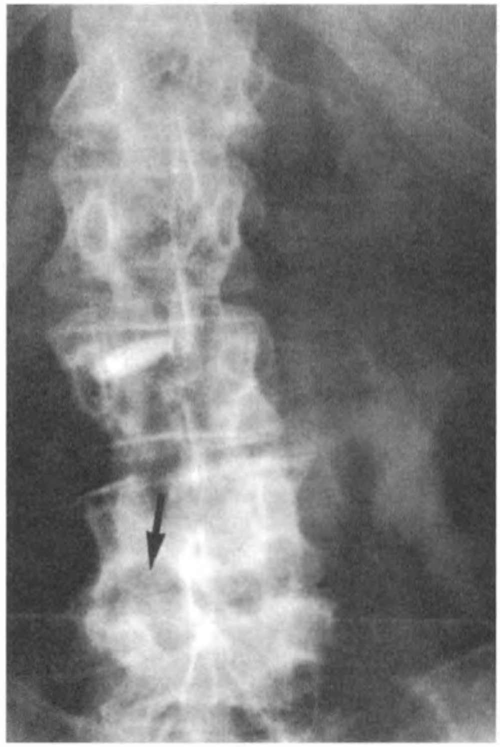


Figure 10.169. The left L4 inferior facet and L5 superior facet have been removed to enable surgical removal of a free fragment of L4–L5 disc within the L4–L5 intervertebral canal. Note the surgical bone removal (*arrow*). L4 is in left lateral flexion subluxation. (Case courtesy of David Taylor, DC.)

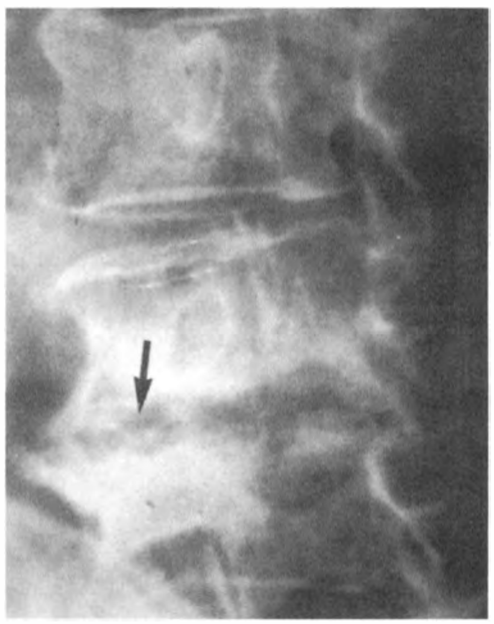


Figure 10.170. Note the marked loss of the L4–L5 disc space with irregularity of the opposing body plates having the appearance of discitis (*arrow*).

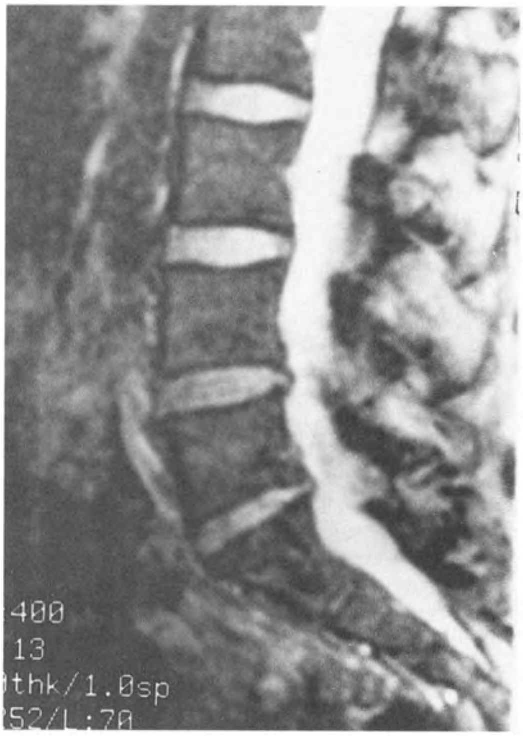


Figure 10.171. Note the loss of signal intensity of both the L5–S1 and L4–L5 discs.

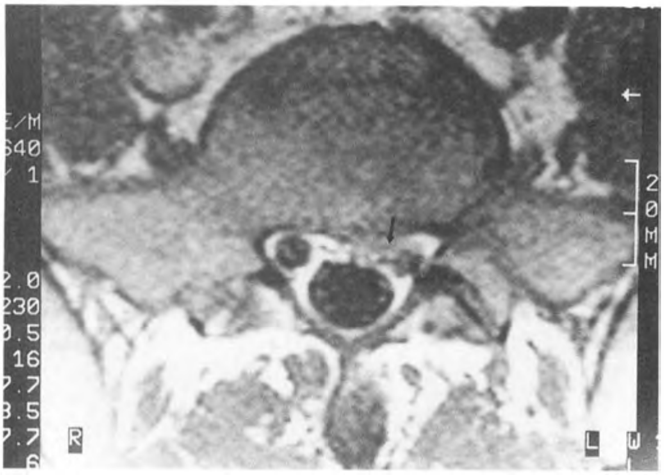


Figure 10.172. Note the left central disc herniation at L5–S1 (*arrow*).

Figure 10.173. The L5–S1 disc shows loss of signal intensity, type I marrow changes of the L5 vertebral body, a large anterior disc herniation, and a small posterior disc herniation. Anular irritation, as seen here, is documented to radiate pain into the groin, buttock, thigh, and flank.

Figure 10.174. A small central L5–S1 disc herniation is seen on the sagittal image shown in Figure 10.173.

Anterior Disc Herniation As a Cause of Referred Pain

Case 12

Figures 10.173 and 10.174 are T1-weighted sagittal and axial images showing L5–S1 loss of signal intensity, a small posterior central disc herniation, and a large anterior herniation. Such anterior disc irritation can refer pain into the flank, groin, buttock, and thigh because of anular fiber irritation.

Sequestered L5-S1 Fragment Conservatively Treated

Case 13

A 27-year-old insulin-dependent diabetic presented with right thigh pain extending to the knee in the distribution of the first sacral nerve root. No motor or sensory findings were seen and surgery was recommended to remove the L5–S1 disc herniation, but the patient chose conservative care.

Figures 10.175 and 10.176 are sagittal T1-weighted MRI images showing a large L5–S1 fragment, paracentral to the right side, which compresses the right first sacral nerve root (see arrow) on axial image. Note the extension of the free fragment posterior to the first sacral segment on sagittal view (*arrow*).

Limbus Vertebra As Seen on Plain and MRI Imaging

Case 14

Figures 10.177 and 10.178 show plain lateral x-ray imaging of an L3 anterosuperior plate ununited apophysis (*arrow*) with comparison of Figure 10.178 showing the trapezoid shaped defect filled with disc intensity material (*arrow*) on sagittal MRI image. This is the discal invagination of the apophysis and replacement of the vertebral body because of apophyseal failure to develop. Also note the Schmorl's nodes into the inferior L3 vertebral end

Figure 10.175. Note the large free fragment of L5–S1 disc material lying posterior to the first sacral body (*arrow*).

Figure 10.176. Note the large free fragment of L5–S1 disc material lying within the right posterolateral vertebral canal, compressing the right first sacral nerve root (*arrow*).

Figure 10.177. Plain x-ray film shows the anterior limbus vertebra at L3 (*arrow*).

Figure 10.178. Magnetic resonance image shows the appearance of the limbus vertebra (*arrow*). Also note the appearance of the L3–L4 Schmorl node defects that are not appreciated on plain x-ray study.

Figure 10.179. Right lateral flexion showing the left L3 accessory rib and its articulation with the L4 transverse process as a pseudoarticulation.

plate and superior end plate of L4 (*arrowheads*); these are not appreciated on the plain x-ray film in Figure 10.177. Also note the L4-L5 level stenosis formed by the posterior ligamentum flavum thickening and the posterior disc protrusion.

Lumbar Rib

Case 15

Figures 10.179 and 10.180 are right and left lateral bending studies of an accessory rib between the L3 and L4 lumbar transverse processes on the left side. Note also the movement of the pseudoarticulation of the rib with the L4 transverse process. Little wonder that this patient experienced much pain on motion.

Developmentally Enlarged L5–S1 Foramen

Case 16

Figure 10.181 is a lateral plain x-ray study showing an enlarged L5–S1 osseoligamentous canal that extends posteriorly into the lamina of L5 and the facet and lamina of the sacrum. The posterior L5 vertebral body is not viewed completely and a semilunar appearing posterior border suggests an erosive effect. The total canal measures more than 3 cm in diameter.

Figure 10.182 is an enhanced sagittal MRI image showing the L4 and L5 nerve roots to be well visualized within the enlarged canal. The canal has definitive margins and no sign of signal change indicative of bone hyperintensity or hypointensity. Final diagnosis was an anomalous formation of the osseoligamentous canal at L5–S1, which was of no clinical significance.

Figure 10.180. Note the accessory rib from Figure 10.179 showing motion at the L4 pseudoarticulation.

Figure 10.181. Plain lateral x-ray study shows enlargement of the L5–S1 intervertebral osseoligamentous canal that extends posteriorly into the lamina of L5 and sacrum with a semilunar appearance of the L5 posterior vertebral body. This suggests an erosive defect measuring in excess of 3 cm.

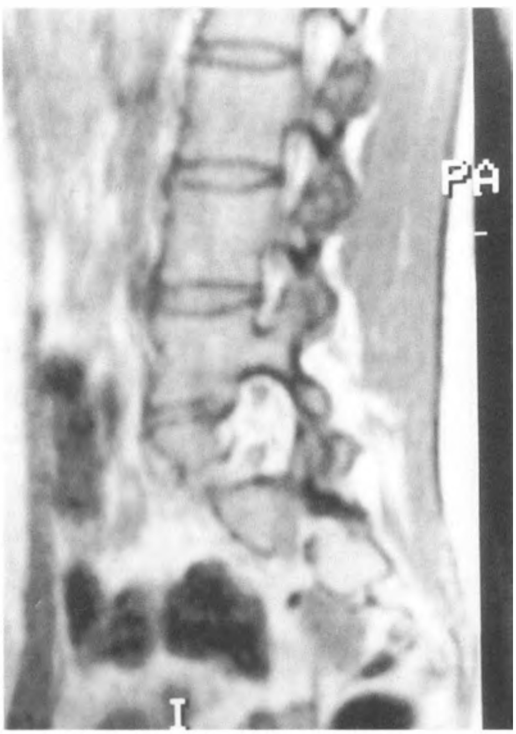


Figure 10.182. Magnetic resonance image with enhancement shows the L4 and L5 nerve roots to be well-visualized within the abnormally enlarged canal.

PATHOLOGIC CAUSES OF LOW BACK PAIN AND SCIATICA

The chiropractor is confronted with patients whose low back pain and leg pain are caused by organic diseases. These cases must be diagnosed and referred for proper comanagement. Examples of such conditions diagnosed in chiropractors' clinics will be presented.

Ependymoma

Figure 10.183 is a T1-weighted sagittal enhanced MRI image showing spinal cord widening with a hyperintense mass below the conus medullaris of a 21-year-old woman with low back pain and gait disturbance. Her symptoms had been considered somatoform prior to this MRI study. Surgery confirmed this to be an ependymoma.

Staghorn Calculus of Kidney

Figure 10.184 shows a large staghorn calculus within the collecting system and pelvis of the left kidney, which was producing pain in this patient.

Paget's Disease

Figure 10.185 shows the mixed lytic and blastic changes within the bones of the pelvis with right-sided thickening and sclerosis of the pelvic brim (*arrow*). Also note that the fourth lumbar vertebral body is expanded and appears sclerotic, which is commonly seen in Paget's disease.

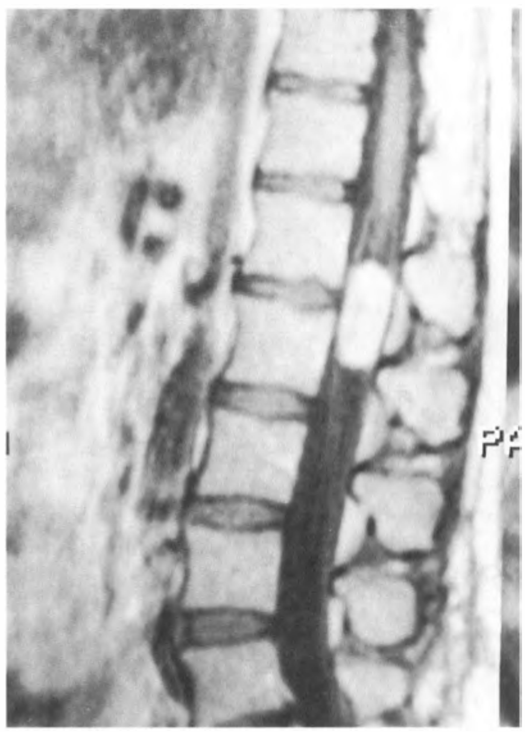


Figure 10.183. Ependymoma.

Figure 10.184. Staghorn calculus.

Figure 10.185. Paget's disease.

This pathology can be treated with chiropractic adjustment using low force distraction, always carefully testing the patient's tolerance before applying the manipulation.

Facet Fracture

After a fall, a patient was found to have a fracture through the left L4 inferior facet. See the arrows in Figures 10.186 and 10.187.

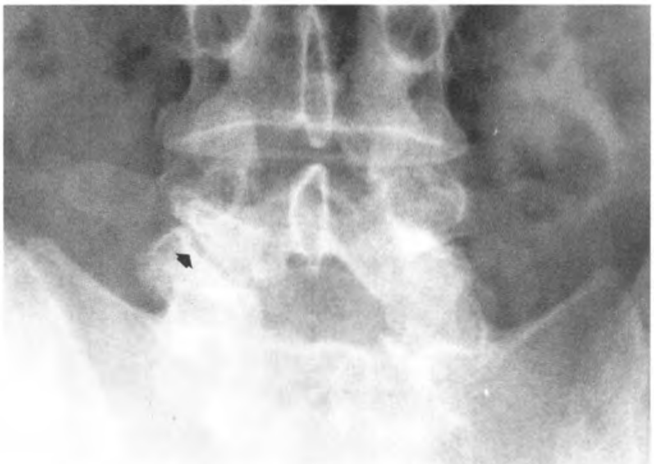


Figure 10.186. Fracture of the left L4 inferior facet (*arrowhead*).

Figure 10.187. Oblique view of Figure 10.186 showing the facet fracture (*arrowhead*).

Forestier's Disease

Diffuse idiopathic skeletal hyperostosis, or Forestier's disease is a condition is seen in 6 to 28% of autopsies with a ratio of men to women of 2:1, it is found mostly in whites and rarely in blacks. High percentages of these patients (30%) have diabetes mellitus. Morning stiffness, which dissipates within an hour but recurs later in the day, is typical.

Figures 10.188 to 10.190 show the preserved disc spaces with the flowing "candle wax" calcification along the anterolateral aspects of many vertebral bodies (*arrowheads*), which is typical of this condition. Note the preservation of the facet joint spaces (*arrows*). The sacroiliac joints show no erosion, sclerosis, or fusion. Because the posterior elements of the spine were not affected, the patient had good range of motion. Also note the thin radiolucent line separating the vertebral body from the calcification anterior to it (*arrow* on oblique view).

Figures 10.191 and 10.192 show the irregular, thick calcification anteriorly and laterally to the vertebral bodies of L4 and

Figure 10.188. Anteroposterior lumbar spine radiograph showing the flowing "candlewax" calcification of the anterolateral aspects of the vertebral bodies at the anterior ligament (*arrowhead*).

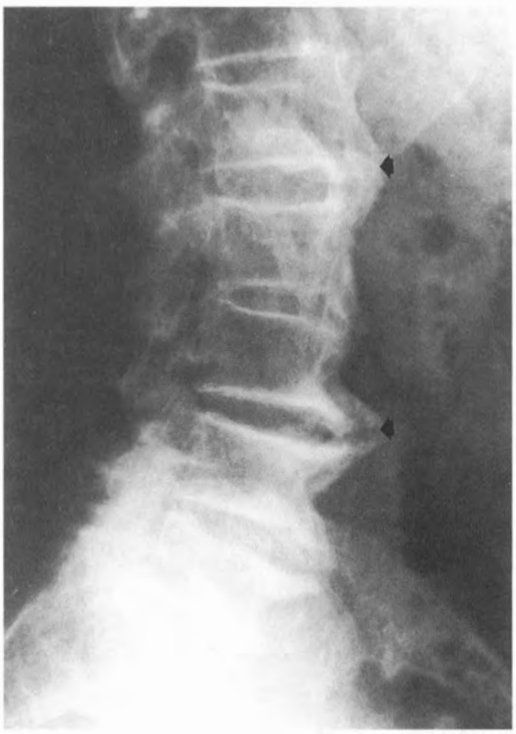


Figure 10.189. Lateral radiograph showing the preserved disc spaces and anterior flowing calcification of the anterior ligament (*arrowheads*).

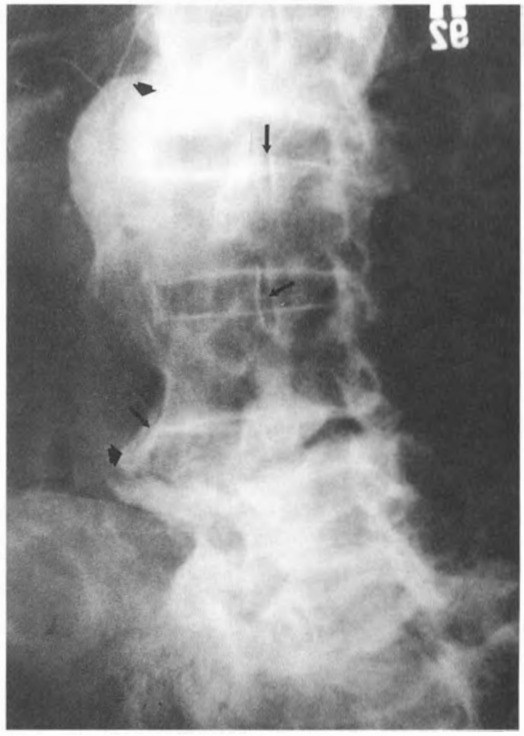


Figure 10.190. Note the preserved facet joint spaces (*arrows*) and the radiolucent thin line separating the vertebral body from the calcification anterior to it (*arrow*). The high anterior ossified ligament is noted (*arrowheads*).

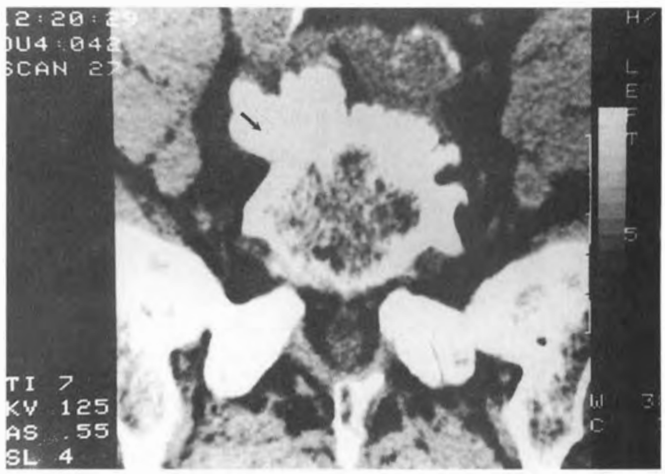


Figure 10.191. Computed tomography scan shows the thick, irregular calcification of the anterior ligament at the L5–S1 level (*arrow*).

Figure 10.192. Computed tomography scan shows the same changes at the L4–L5 levels as seen at L5–S1 in Figure 10.191 (*arrow*).

Figure 10.193. Computed tomography scan of the huge cervical spine anterior ligament hyperostosis that caused dysphagia in this patient.

L5 (*arrows*). Figure 10.193 shows the marked hyperostosis of the anterior cervical spine that caused dysphagia for this patient.

Treatment for these patients is range of motion adjusting after carefully testing for tolerance to the technique. Because the posterior elements are spared from fusion, motion can be elicited, often to the relief of the patient. Forceful adjusting is not tolerated by these patients, but distraction adjusting with lateral flexion, rotation, and circumduction motions gently applied is tolerated and helpful.

Hemangioma

Hemangiomas are benign neoplasms often seen incidentally on routine plain x-ray films and MRI studies. Most commonly they are solitary lesions, but they can be multiple and vary in size from small areas to total vertebral body involvement (Fig. 10.194). They can expand also beyond the confines of the vertebral body and even extrude into and compromise the spinal canal. They can weaken a vertebra and result in compression fracture, although this is uncommon (194). An autopsy study showed them to occur in 11% of patients (195).

Hemangiomas appear as hyperintense on both T1- and T2-weighted MRI images, which is explained by the mixture of angiomatous tissue and adipose tissue between the prominent trabeculae. The fat content accounts for the high-intensity T1 signal (196).

Ankylosing Spondylitis

A 19-year-old man complained of low back pain and stiffness, which was progressive for a 3-year period. Figures 10.195 and 10.196 were originally read as normal by a radiologist except for mention of loss of definition and increased sclerosis of the sacroiliac joints bilaterally.

Closer reading of these x-rays films shows missed information. Laboratory HLA-B-27 testing was positive and the impressions of psoriatic arthropathy, Reiter's syndrome, and more remotely rheumatoid arthritis were ruled out in favor of the diagnosis of ankylosing spondylitis. A lesson from this case is do not trust reports coming to you until you check the details of the study yourself. The overlooked subtle syndesmophyte also helped lead to the proper diagnosis of this case.

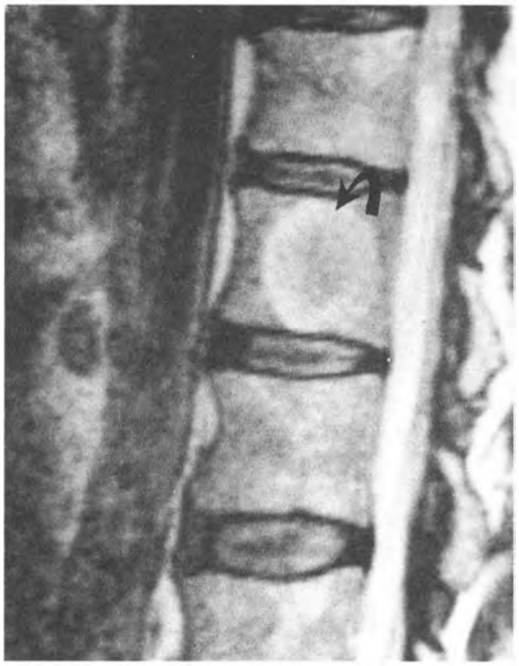


Figure 10.194. Hemangioma (*curved arrow*).

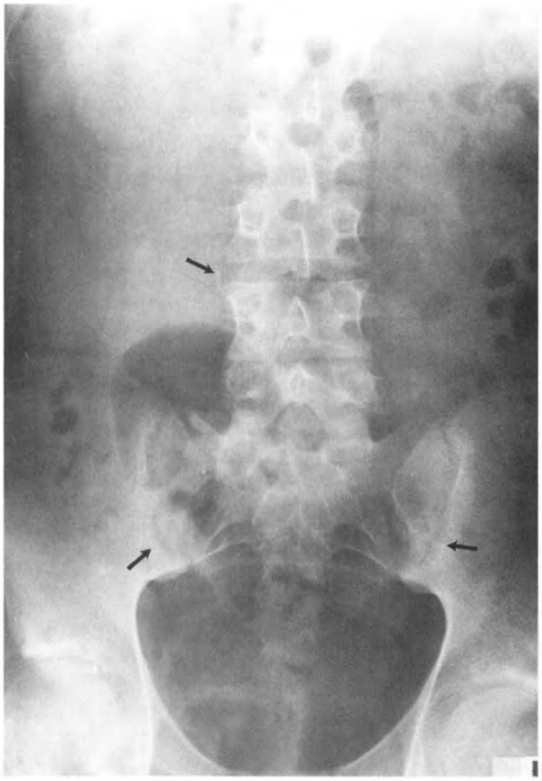


Figure 10.195. Ankylosing spondylitis. Note the left L3–L4 syndesmophyte formation (*arrow*) and the sacroiliac irregularity, widening joint space, and sclerosis (*arrows*).

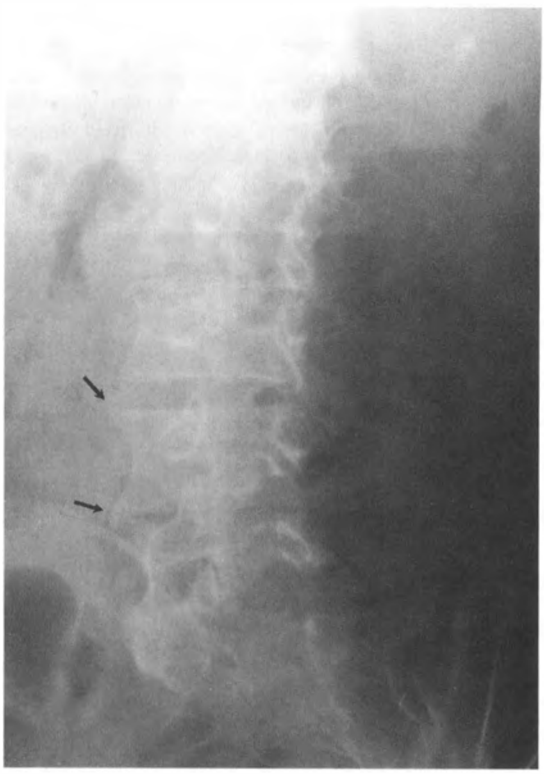


Figure 10.196. Oblique view of Figure 10.195 showing syndesmophyte formation at the L3–L4 and L4–L5 levels (*arrows*).

Unilateral Spondylolysis with Multilevel Spinal Stenosis

A 69-year-old woman is advised to have decompression surgery at the L3–L4 and L4–L5 levels to remove a disc protrusion, hypertrophic bone formation, and ligamentum flavum hypertrophy that had combined to form spinal stenosis at both lumbar levels, resulting in low back and leg pain. The patient chose chiropractic care first. Figure 10.197 shows L4–L5 left posterolateral disc protrusion and fragmentation (*arrow*) and bilateral ligamentum flavum thickening (*arrowheads*), which combined to form stenosis. Facet arthrosis is also noted. Figure 10.198 at the L3–L4 level shows left facet hypertrophy (*arrowhead*) and posterolateral bone plate hypertrophy, creating spinal and canal stenosis. Figure 10.199 shows left L5 unilateral spondylolysis, which I feel caused instability and added stress to the stenotic changes at the superior two levels.

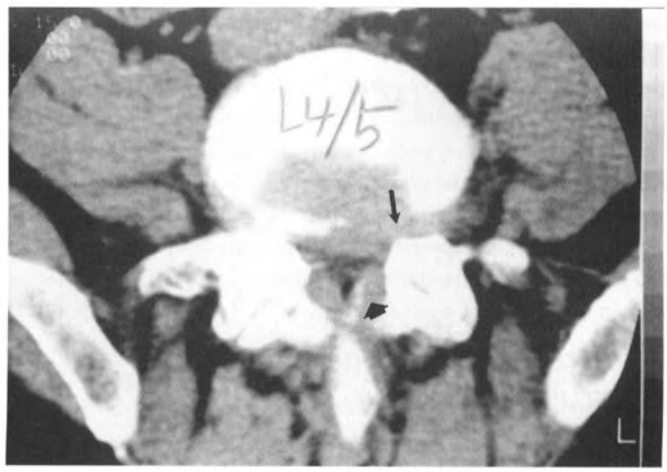


Figure 10.197. Computed tomography scan shows L4-L5 level spinal stenosis caused by ligamentum flavum hypertrophy (*arrowhead*) and left posterolateral disc herniation (*arrow*).

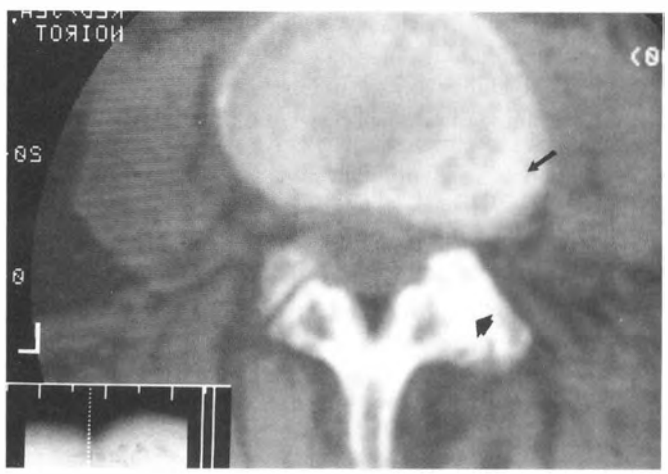


Figure 10.198. Computed tomography scan shows L3-L4 level stenosis caused by facet arthrosis (*arrowhead*) and posterolateral end plate hypertrophic changes (*arrow*).

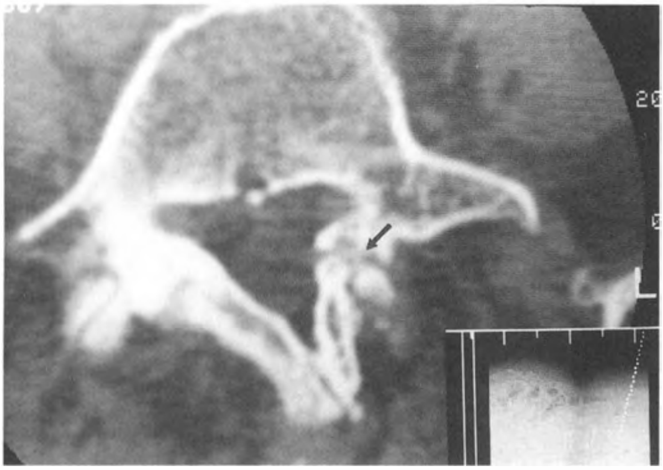


Figure 10.199. Computed tomography at L5–S1 shows left unilateral spondylolysis (*arrow*), an area of instability.

Treatment consisting of distraction manipulation, positive galvanism and heat, tetanizing current and ice, followed again by heat and acupressure point therapy resulted in good relief of this patient's pain so that 2 1/2 weeks of daily care resulted in total relief of the leg pain, with only low back pain persisting. This is an example of conservative care accomplishing satisfactory relief of patient pain without surgical intervention. Some of these cases that appear to be so stenotic remarkably respond to basic conservative distraction adjusting.

Undetermined Myopathy, Possible Muscular Dystrophy

A 51-year-old man complained of low back pain and bilateral leg pain with pain extending to the great toe on the left side and to the knee on the right. Blood triglycerides and creatine kinase were greatly elevated. See Figures 10.200–10.202, which are transaxial as well as coronal image sequences of the lumbar spine. Much unusual fatty replacement and muscle atrophy of the posterior back muscles is seen. See figure legends for the interpretation of findings.

The Mayo clinic worked up this case but no final diagnosis was forthcoming other than a type of muscular dystrophy.

Neurilemoma of Sciatic Nerve

Tumors of the nerve sheath should be included in the differential diagnosis of neurogenic pain in the lower extremity. MRI is probably the diagnostic modality of choice when a lesion of the sciatic nerve is suspected (197).

Compartment Syndrome

There are 46 compartments in the human body, 38 of which are located in the extremities where about 80% of compartment syndromes occur. A compartment is a space enclosed by inelastic fascia. A compartment syndrome is defined as an increase in pressure within a myofascial compartment that compromises capillary flow and, subsequently, neuromuscular function. Two types of compartment syndrome are found (198): (*a*) acute type and (*b*) recurrent, exertional, or chronic type, a disorder that results in intermittent periods of high pressure in the compartmental area sufficient to cause ischemic pain and impaired neuromuscular function.

The leg has traditionally been described as being composed of four compartments (e.g., anterior, lateral, superficial posterior, and the deep posterior). More recently, literature has added a fifth compartment, the posterior tibial. See Figure 10.203.

In general, if tissue pressures rise within a compartment to 30 to 40 mm Hg, capillary circulation can be compromised.

Figure 10.200. Axial T1-weighted image at the midlumbar spine reveals hyperintensity of the erector spinae muscles, labeled on the image as *1* (multifidees), *2* (longissimus), *3* (iliocostalis), and *4* (quadratus lumborum muscle). Compare the normal right quadratus lumborum muscle density with the hyperintense left side.

Figure 10.201. Axial section of the lower thoracic spine reveals relative normointensity of the multifidees muscles bilaterally (see number 1), whereas the longissimus muscles, shown at the number 2, reveal more normal intensity of the left side and hyperintensity indicative of fatty replacement of muscle tissue on the right side.

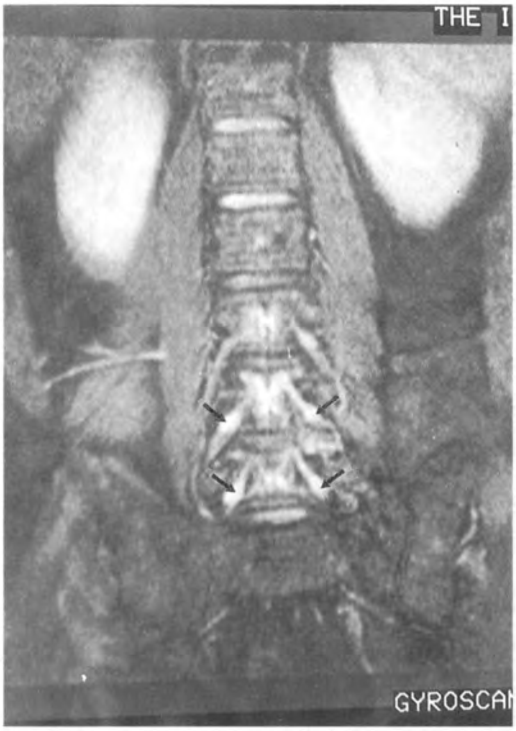


Figure 10.202. This coronal section through the vertebral and osseoligamentous canals shows the dorsal root ganglia (*arrows*) located intraspinally and intraforaminally in their course from the origin at the cauda equina to their exit at the outer limits of the osseoligamentous canal. This is an informative study showing the location of the nerve roots and ganglion and their vulnerability to stenosis by disc herniation, facet arthrosis, or even ligamentum flavum hypertrophy.

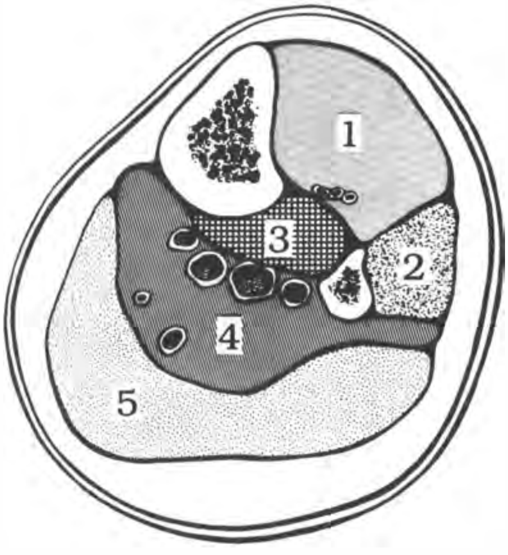


Figure 10.203. A diagram depicting the five compartments of the lower leg (*1*, anterior; *2*, lateral; *3*, posterior tibial; *4*, deep posterior; and *5*, superficial posterior). The drawing was patterned after Bourne R, Rorabeck C. Compartment syndromes of the lower leg. Clin Orthop 1989;240:98. (Reprinted with permission from Gerow G, Matthews B, Jahn W, et al. Compartment syndrome and shin splints of the lower leg. J Manipulative Physiol Ther 1993;16(4):245–252.)

Should this pressure remain elevated for extended periods of time, irreversible muscle and nerve injury can occur by capillary blood ischemia, producing an anoxia in the muscles and nerves in this region—the acute form of compartment syndrome.

The second variety of compartment syndrome, the chronic form, is more common and it is generally found in persons in their 20s who are active athletes. Chronic compartment syndrome is also known as recurrent, subacute, and exertional compartment syndrome, as well as intermittent claudication in athletes. The chronic anterior compartment syndrome is generally a synonym for the anterior tibial syndrome (198).

Shin Splint

The anterior shin splint syndrome involves musculotendinous inflammation or injury to the dorsiflexors of the foot, including the tibialis anterior, extensor hallucis longus, and extensor digitorum longus. The most common cause of anterior leg pain is periostitis, followed in decreasing prevalence by chronic compartment syndrome and superficial peroneal nerve entrapment. The soleus syndrome, one type of posterior shin splint, is caused by unequal pull of fascia, which occurs when the foot is in the pronated position.

Conservative management procedures in the treatment of acute shin splints include rest, physiotherapy, and cryotherapy. Microcurrent therapy and bracketing the involved region may also be of benefit. Once the acute phase is over, the following treatment may be used: massage, heat, trigger point therapy, foot orthotics, heel cord stretching of the nonballistic variety, ultrasound, local heat, shoe modifications, alterations in training program, and taping procedures. The athlete should continue to be taped for 1 month after resuming activity (198).

Epidural Hematoma

Spontaneous epidural hematoma can result from tearing of fragile epidural veins lying adjacent to the displaced anulus or nucleus (199).

Figure 10.204 shows the intraosseous and extraosseous vertebral venous system of the lumbar spine. Abnormalities or pathologic change of this venous network may give rise to symptoms similar to or mimicking lumbar disc herniations or spinal stenosis. Figure 10.205 is from a thrombosed dilated epidural vein case (200). Figure 10.206 demonstrates the differential findings of epidural hematoma from herniated disc material.

The proposed mechanism for hematoma formation is that disc herniations obstruct the epidural venous flow leading to phlebothrombosis (201). With minimal neurologic findings, or evidence of an early resolution of the hematoma and neurologic deficits, a conservative, nonoperative approach to therapy may be indicated (202). There should be an awareness of a possible link between aspirin and spinal epidural hematoma (203).

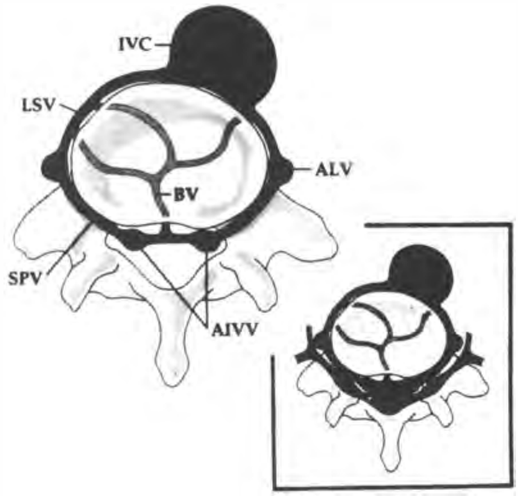


Figure 10.204. Axial illustration of the epidural venous plexus system of the lumbar spine. Note the intimate relationship with the overlying elements of the cauda equina and nerve roots. Elements of the venous network include the basivertebral vein (*BV*), the anterior internal vertebral veins (*AIVV*), the supra- and infrapediculate radicular veins (*SPV, IPV*), the ascending lumbar veins (*ALV*), and the lumbar segmental veins (*LSV*), which drain into the inferior vena cava (*IVC*). (Reprinted with permission from Hanley EN, Howard BH, Brigham CD, et al. Lumbar epidural varix as a cause of radiculopathy. Spine 1994;19(18):2122–2126.)

Lower Extremity Thrombus Prevention with Vena Cava Filter Screen

A 64-year-old man is seen with a history of lower extremity blood clots that resulted in the filter screen placement in the inferior vena cava to prevent thrombus formation from reaching his heart. Although this is an unusual finding on lumbosacral x-ray study, it is presented to alert the clinician to its anatomic location and physical features. See Figures 10.207 and 10.208.

Ligamentum Flavum Hematoma

Few cases of hematoma in the ligamentum flavum causing lumbar root compression have been described (204). Two patients presenting with signs and symptoms suggestive of nerve root compression secondary to extradural masses were found to have ligamentum flavum hematomas (205). Such hematomas must be considered in the differential diagnosis in a patient with back or leg pain, especially when trivial trauma is involved. On MRI, a mass continuous with the ligamentum flavum, compressing the dural sac and roots, is found. Removal of ligamentum flavum is the treatment of choice (204).

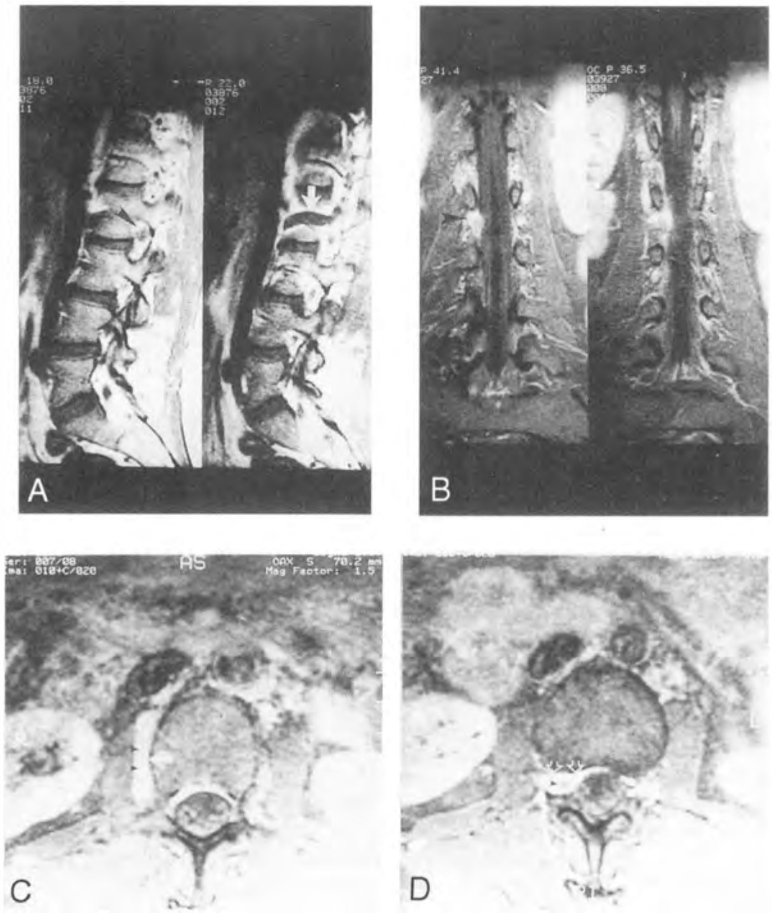


Figure 10.205. **A.** Right parasagittal magnetic resonance image (MRI) T1 (TR500/TE11). Spin echo image demonstrates enlarged lumbar segmental vein with intraforaminal extension (infrapediculate vein) intimately encasing the exiting nerve root. **B.** Coronal MRI spin echo T1 (TR750/TE12) with fat saturation after intravenous gadolinium. An enhanced mass with a central low signal defect extends into the right foramen and into the dilated adjacent ascending lumbar vein. Slight medial mass effect is present on the thecal sac. **C.** Axial MRI spin echo T1 weights (TR750/TE13) with fat saturation after intravenous gadolinium. The right lumbar segmental vein is dilated with residual central thrombus. Slight asymmetry is seen in the anterior internal vertebral veins. **D.** Axial MRI spin echo T1 (TR750/TE13) image with fat saturation after intravenous gadolinium. The anterior internal vertebral vein is dilated on the right with residual free-floating thrombus. Moderate mass effect on the thecal sac and displacement of the nerve root are identified. (Reprinted with permission from Hanley EN, Howard BH, Brigham CD, et al. Lumbar epidural varix as a cause of radiculopathy. Spine 1994;19(18):2122–2126.)

Figure 10.206. Magnetic resonance images demonstrating degenerative disc disease at the L3–L4 level with central disc herniation (*arrowheads*). In addition, a ventral and right-sided epidural mass (*arrows*) is revealed, with an intensity different from that of the disc herniation. **Left**: Sagittal T1-weighted image (RT 700 msec, TE 15 msec). **Center**: Axial proton-density image (TR 2168 msec, TE 15 msec). **Right**: Sagittal T2-weighted image (TR 2168 msec, TE 90 msec). (Reprinted with permission from Zimmerman GA, Weingarten K, Lavyne MH. Symptomatic lumbar epidural varices: report of two cases. J Neurosurg 1994;80:914–918.)

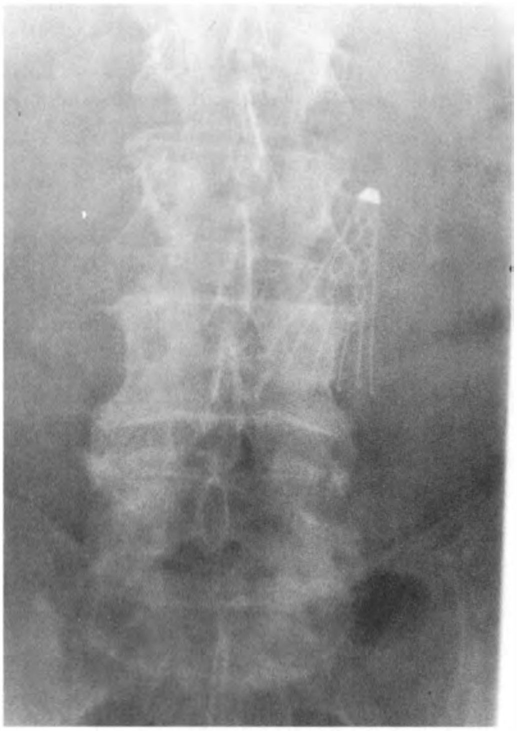


Figure 10.207. Parachute filter screen placed in the inferior vena cava to prevent thrombus from reaching the heart.

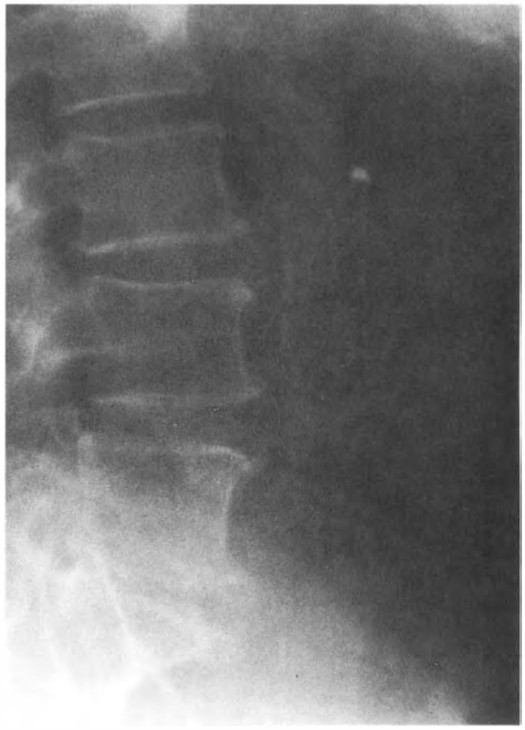


Figure 10.208. Lateral view of the filter shown in Figure 10.207.

Sacral Tarlov Cysts

Seventeen percent of patients undergoing myelography for the investigation of low back pain with radiculopathy show Tarlov cysts on myelography. A certain unknown percentage of which will cause symptoms such as sciatica or bowel and bladder dysfunction.

No significant difference was found in size between symptomatic and asymptomatic cysts in these patients. A striking disparity in the context of communication with the subarachnoid space is reported: five of five asymptomatic cysts were shown to communicate on MRI flow studies, whereas seven of seven symptomatic cysts were not shown to communicate (206).

Cysts of the S3 nerve root have been reported in patients who complained of neurogenic bladder and perianal sensory disturbance as well as buttock pain (207).

A case study of perineural cysts involved an 83-year-old woman complaining of low back pain and bilateral anterior thigh pain after a fall. Prior colon cancer resection 2 years previously was reported. Range of motion of the thoracolumbar spine was impossible because of the pain, SLR was normal recumbent, Patrick signs were normal, and the deep tendon reflexes of the lower extremity were +2 bilaterally and equal.

Generalized osteopenia of bone was seen on plain x-ray film with a 50% compression of the L4 vertebral body anteriorly with preserved height posteriorly. Blood tests were negative for multiple myeloma or malignancy.

Figure 10.209 is a T1-weighted sagittal image showing relatively homogeneous decreased signal intensity of the L4

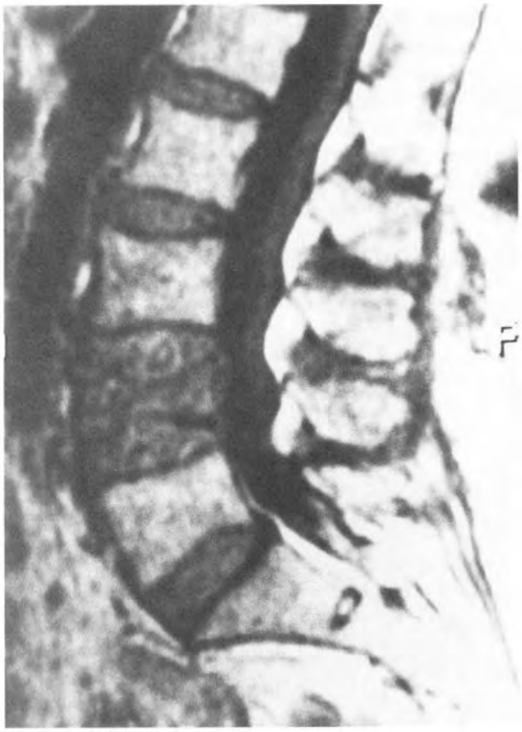


Figure 10.209. Sagittal T1-weighted image shows decreased density of the L4 vertebral body that is homogeneous throughout.

Figure 10.210 Sagittal T2-weighted image shows the signal intensity of the vertebral bodies and sacrum to be hyperintense and unremarkable. Note the small hemangioma of the L3 vertebral body (*arrows*). At the arrows are shown perineural cysts (Tarlov cysts) appearing as hyperintense on T2 weighting. These cysts involve the L4, L5, and sacral nerve roots.

Figure 10.211. A T1-weighted sagittal image showing the Tarlov perineural cysts as hypointense areas (*arrows*) compared with the appearance of the T2 images in Figure 10.210.

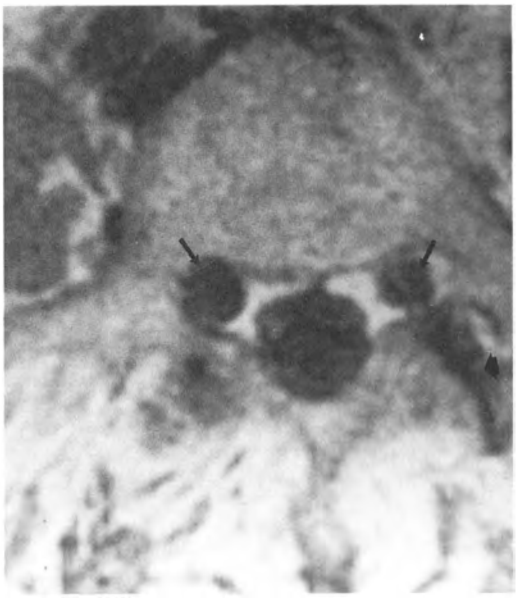


Figure 10.212. Axial T1-weighted image shows the large perineural Tarlov cysts (*arrows*) within the lateral recesses of the vertebral canal.

vertebral body, whereas a T2-weighted image (Fig. 10.210) shows mildly hyperintense body signal. The L4 vertebral body changes were felt to be a benign compression fracture. Note the ectasia of the upper sacral nerve root sleeves incidental to perineural Tarlov cysts at the L4 and L5 and upper sacral levels shown on Figures 10.210–10.212.

Treatment in this case was epidural blocks with steroid medication, which were not of benefit to the patient. Gentle flexion-distraction manipulation of the lumbar spine was given. Isometric contractions of the thigh and calf muscles to stimulate circulation were instituted as she did develop lower extremity swelling because of inactivity. Gradual relief of pain took place within 4 weeks of care.

Conjoined Nerve Roots

The thecal sac is the origin of lumbar nerve roots, with a nerve root exiting at the disc interspace, coursing downward and laterally to pass under the pedicle of a vertebra and exiting through the osseoligamentous canal at the level below the nerve root origin from the sac. In 1 to 2% of humans, instead of being individual nerve roots at each interspace, two nerve roots will join and exit at the same level. This is most commonly seen at the L5–S1 level by a conjoined L5 and S1 root, and, less commonly, at the L4 and L5 root level and the L3 and L4 root level.

The conjoined nerve root is a developmental abnormality in which two nerve roots arise together, sharing a common dural sleeve, and then separate within the vertebral canal in the lateral recess to exit through their own specific foramen. A triangular thecal sac extension may be seen on CT or MRI, suggesting a conjoined root.

Computed tomography appearance of conjoined nerve roots is that of cerebrospinal fluid because the dural sac surrounds the conjoined roots. Other tissues such as disc herniation or disc sequestration would be more hyperintense than a conjoined nerve root. This finding is important in diagnosing conjoined nerve roots. Myelography can be beneficial as a contrast study showing both sleeves lying within the CSF-filled sheath (208–211).

The significance of conjoined nerve roots is simply that two nerve roots lie within one sheath, and any irritation, such as a herniated disc, can cause intense pain for the patient. The differentiation of the density of a conjoined nerve root being more closely aligned with that of CSF is important to differentiate it from the more hyperintense changes of bone hypertrophy, herniated discs, or extruded discs. Figures 10.213 and 10.214 are CT and MRI studies showing the characteristic findings of conjoined nerve roots.

Tethered Cord

The tethered cord refers to the conus medullaris being in a lower position than its usual T12–L1 level and accompanied by a thick filum terminale (212). Tethered cord is occasionally seen as a solitary problem or associated with a lipoma or other dystrophic findings. Such dystrophic congenital neural tissue diseases as dermoid cysts, lipoma, diastematomyelia, or teratoma are included with the tethered cord. Tethered cord with lipoma is encountered in the lumbar spine in approximately 33% tethered cord incidences. Although most common in chil-

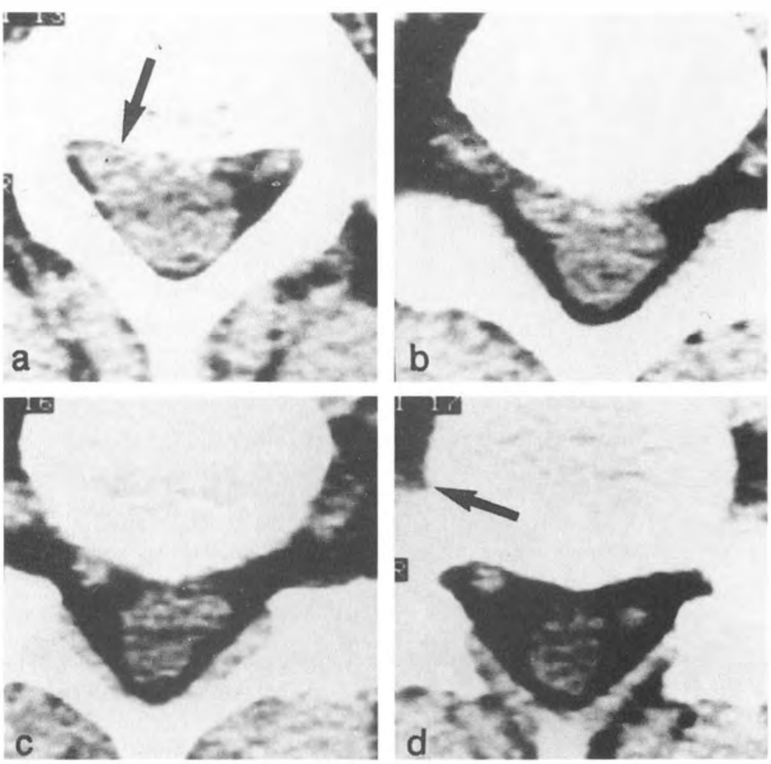


Figure 10.213. Conjoined root; characteristic computed tomography (CT) and magnetic resonance image (MRI) appearance. Consecutive CT scans (**A–D**) of L5–S1 demonstrate the classic conjoined root on the right. **A.** Cephalad to the anulus, the triangular extension (*arrow*) from the sac fills the right recess. The L5 and S1 roots are within this mass, which has the same CT density as the sac. **B.** The right L5 root has separated from the right S1 root, which is still connected with the sac. **C.** Both right roots are separated; the L5 root is in the foramen and the S1 root is still in the canal. The left S1 root is emerging from the sac. **D.** The right L5 root (*arrow*) has just emerged from under the pedicle. Note the characteristic asymmetry of the two S1 roots caused by the usual emergence of a conjoined root at a point between the usual sites of origin of the two roots. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:483–512.)

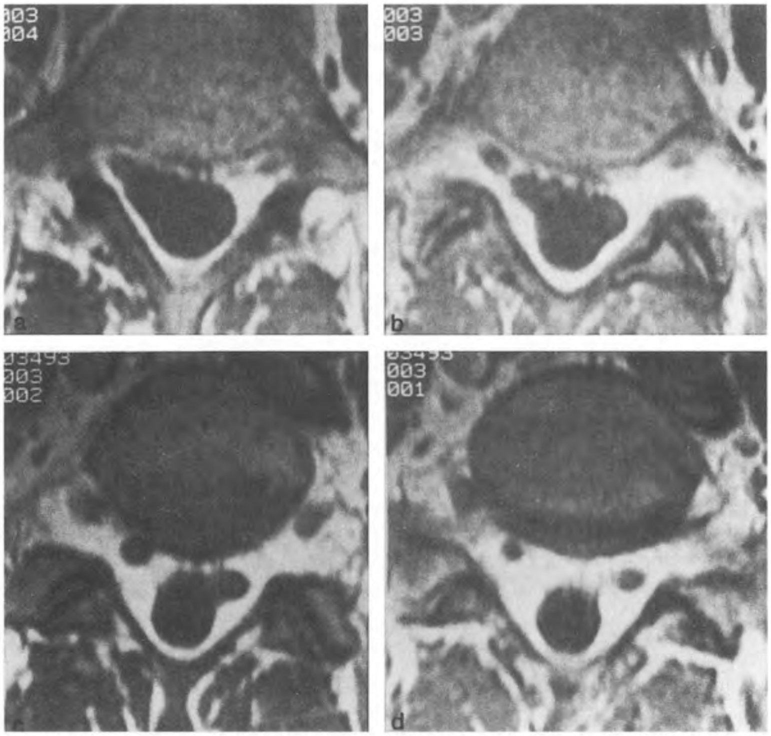


Figure 10.214. Conjoined root; characteristic computed tomography (CT) and magnetic resonance image (MRI) appearance. The axial MRI scans (**A–D**) of L5–S1 correspond closely to the CT scans in Figure 10.213 and clearly show the conjoined right root and its separation into the L5 and S1 roots. Sagittal MRI scans are inadequate for demonstrating or diagnosing a conjoined root. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:483–512.)

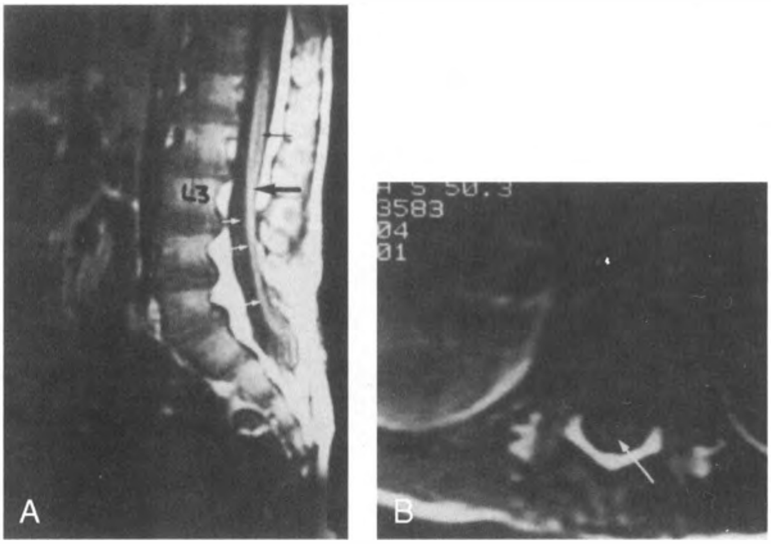


Figure 10.215. Tethered cord and hydromyelia magnetic resonance image. This young woman had an Arnold-Chiari malformation and callosal agenesis. **A.** The T1-weighted sagittal section shows the conus medullaris (*large black arrow*) extending down to L3, a finding consistent with a tethered cord. A long, somewhat thickened posterior root (*white arrows*) is seen extending from the conus to the S1 level. A low-signal linear density (*small black arrow*) in the cord from L1 to L2, which has a high signal on T2-weighted images, is characteristic of hydromyelia. **B.** An axial T1-weighted image of upper L2 shows the low signal fluid in the enlarged central canal (*arrow*) within the high-signal cord. MRI is clearly the best modality for imaging both tethered cord and hydromyelia. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:483–512.)

dren, these conditions may be encountered in adults as well, especially when back or leg symptoms warrant an examination in which tethered cord may be discovered.

Magnetic resonance imaging is the desired modality to study the conus medullaris and the disclosure of tethered cord. The position of the conus medullaris may be from the T12–L1 level to L2–L3, whereas location of the conus from L3 or caudal is considered a tethered conus. Accompanying lipoma is easily diagnosed from T1 and T2 images with the high signal on T1 and low signal on T2 image of fat (213–216). Figures 10.215–10.218 are tethered cord examples.

Calcified Disc Herniation

It is common to see calcification within a herniated nucleus pulposus, especially in children (217–219). Teplick (220) defines five major types of calcification within the disc herniation as seen on CT scan:

1. Linear calcification representing partial calcification of a disc herniation, which may be calcification of the posterior longitudinal ligament or anular material.
2. Focal areas of calcification within a herniation, usually rep-

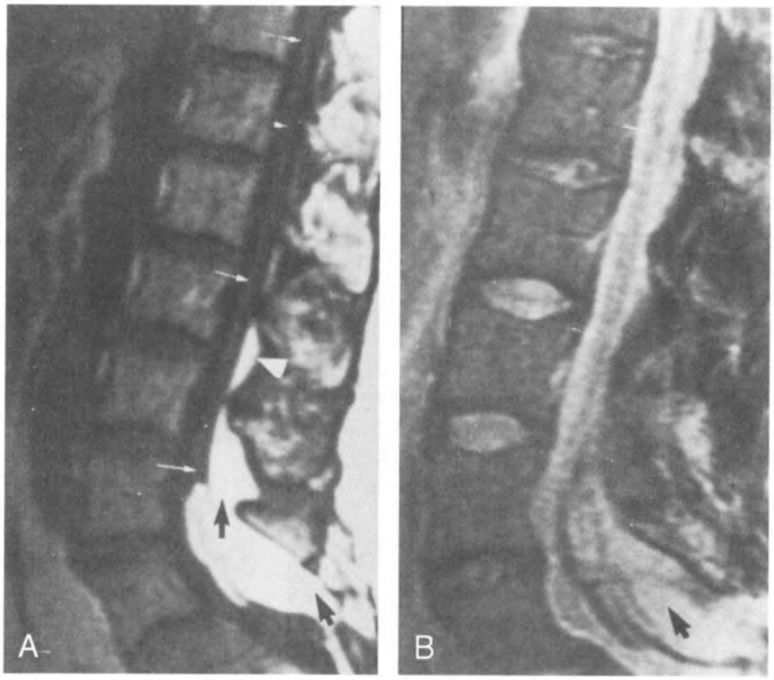


Figure 10.216. Magnetic resonance image of lumbosacral lipoma and tethered cord. Sagittal T1- (**A**) and T2- (**B**) weighted images of a 40-year-old man show the spinal cord (high signal on T1- and low signal on T2-weighted sections; *small white arrows*) extending down to a large high-signal lipoma (*black arrows*) in the posterior canal that is displacing the sac anteriorly from mid L4 to S1. The lipoma itself extends as high as L2–L3 (*arrowhead*). The canal is greatly widened from L3–L4 to S1 by the bulky lipoma. Note the typical decreased signal of the lipoma and other fatty tissue on the T2-weighted image (**B**). (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:483–512.)

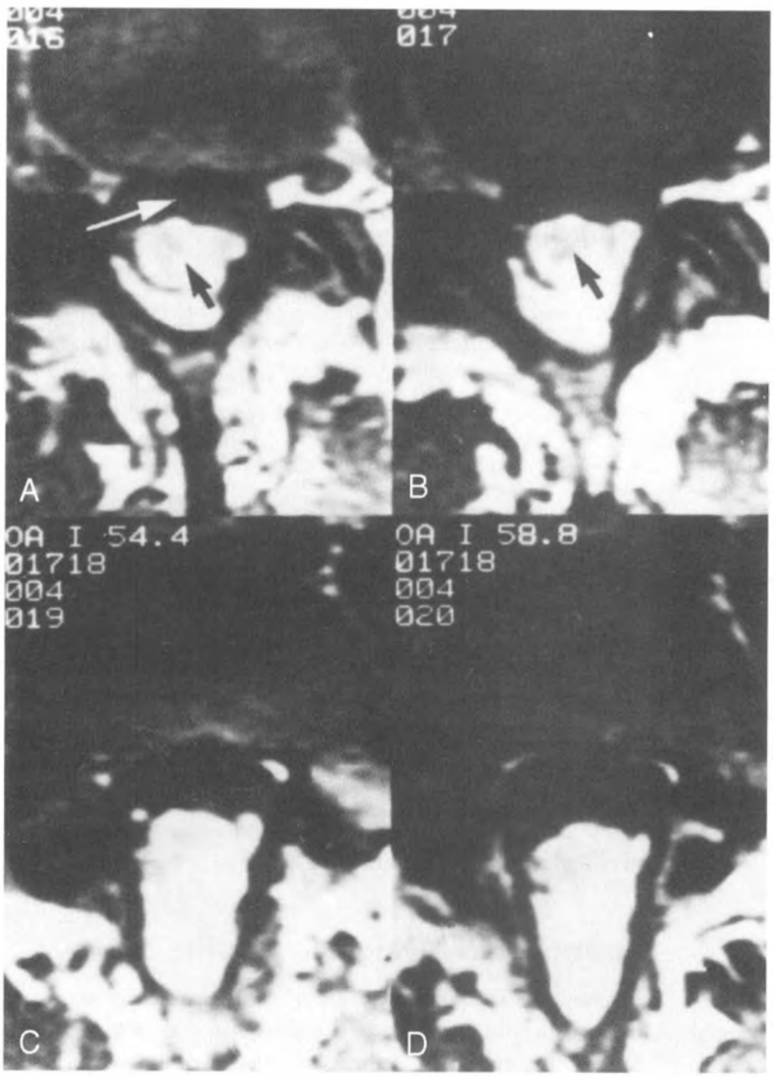


Figure 10.217. Magnetic resonance image of lumbosacral lipoma and tethered cord. **A** and **B.** Sequential axial images of L4–L5 (**C**) and L5–S1 (**D**) show the enlarged elongated canal and the large, somewhat irregular lipoma compressing the sac into the anterior canal (*white arrow*) and also extending itself intradurally into the sac (*black arrows*). The lipoma is clearly both intra and extradural. The extreme low position of the cord, the absence of any clearly defined conus, and the intradural–extradural lipoma are the characteristic findings in this condition. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:483–512.)

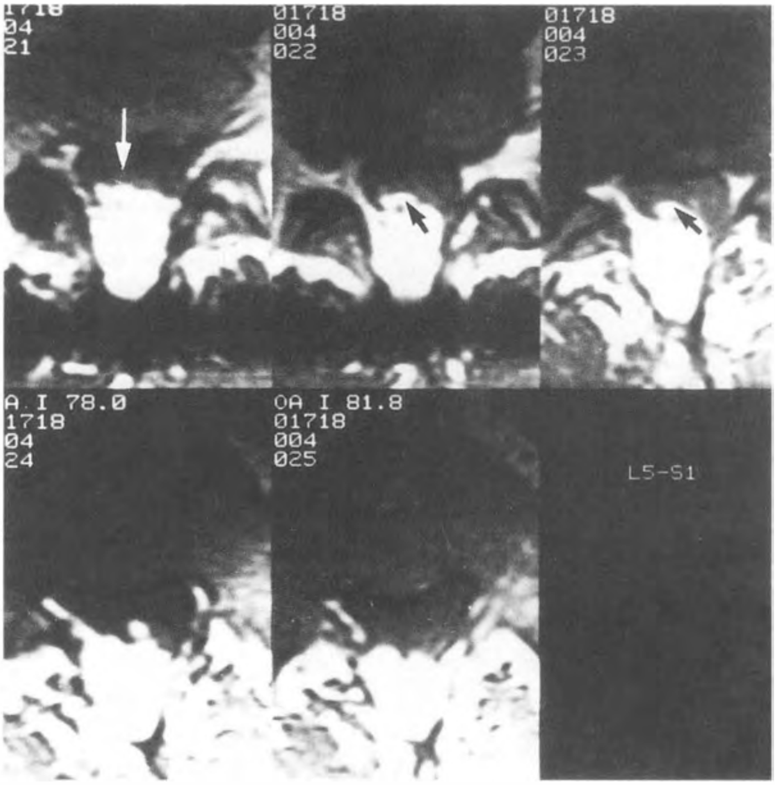


Figure 10.218. Magnetic resonance image of lumbosacral lipoma and tethered cord. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:483–512.)

resenting a longstanding condition of more than a few months.

3. Diffuse stippled calcification of a herniation, which occurs in a shorter period of time, perhaps days. Usually, these have corresponded to an acute onset of back pain with trauma, and have occurred in young males in their teens. The mechanism of this diffuse calcification is obscure.
4. Dense calcification of an entire herniation, which is difficult to differentiate from dense bone hypertrophy.
5. Calcified herniations associated with a calcified nucleus pulposus. They are uncommon in the lumbar spine. They are more common in children within the cervical and thoracic spines; in adults, they are usually seen in the thoracic spine.

It is important to note that CT may be necessary to differentiate calcification as MRI can confuse calcification with bone spur. MRI studies can fail to identify calcification (220).

Figures 10.219 and 10.220 are examples of disc herniation calcification.

Lateral Sacral Artery Aneurysm

A young woman reportedly developed acute cauda equina syndrome from a ruptured aneurysm of the lateral sacral arteries bilaterally. Angiography and partial embolization of the vascular supply and contrast-enhanced high-resolution CT were essential in the diagnosis and treatment of this unique aneurysm (221).

Snapping Hip

The diagnostic test for a snapping hip is to extend the knee, and adduct and flex the hip. A snapping in the hip is a positive sign. The most common cause of snapping hip is a tight band in the fascia lata. This fibrosus commonly follows repeated intramuscular injections of substances such as vitamins, antibiotics, and analgesics, either as treatment for chronic illness or because of drug abuse (222).

Back Mouse

The "back mouse" is a tender, fibrous, mobile, rubbery, size-altering, fatty subcutaneous nodule found in the lumbosacral area in up to 16% of people. "Back mice" are commonly found in people aged 25 to 65 years and in about 25% of women. These fat nodules are the result of herniations of fatty tissue through the neurovascular foramina from the deep fascia into the superficial fascia around the iliac crest and sacroiliac joints. They can cause local pain. The successful treatment is dry needling to reduce distention (223).

Eosinophilia-Myalgia Syndrome

Eosinophilia-myalgia syndrome (EMS), thought to be caused by ingestion of contaminated tryptophan products, is characterized by myalgias, arthralgias, and prominent peripheral

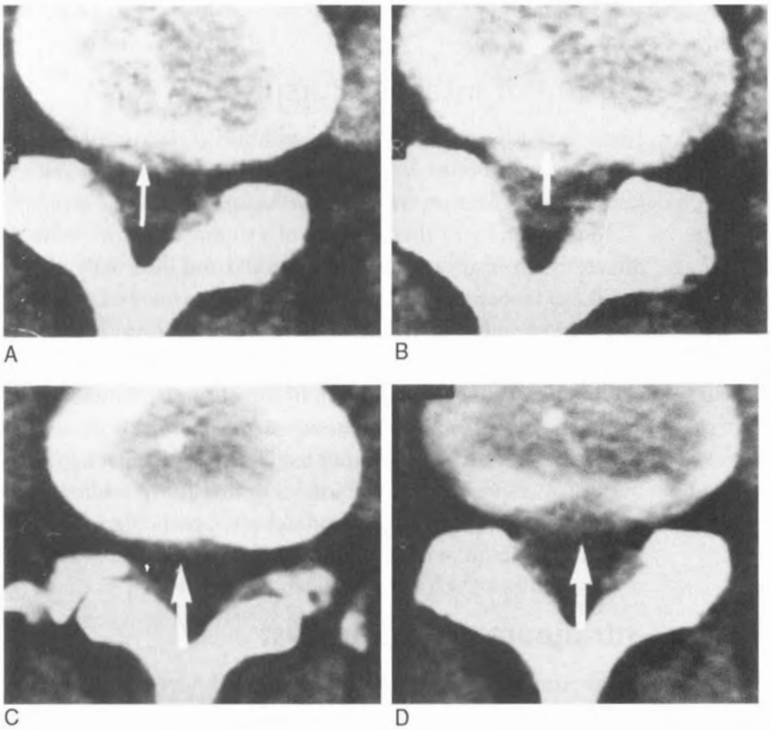


Figure 10.219. Calcified herniation; regression of herniation; and disappearance of the calcification; computed tomography. **A** and **B.** Computed tomography scans through the anulus at L4–L5 disclose a calcified central-right herniation (*arrows*). The low back symptoms and radiculopathy improved rapidly with bed rest. **C** and **D.** Corresponding computed tomography sections made about 4 months later show that the herniation has become smaller, but inexplicably the calcium in the herniation had completely disappeared.Computed tomography sections made 2 years later (not shown) disclosed that the herniation at L4–L5 has completely disappeared; no trace of calcification was seen. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:148.)

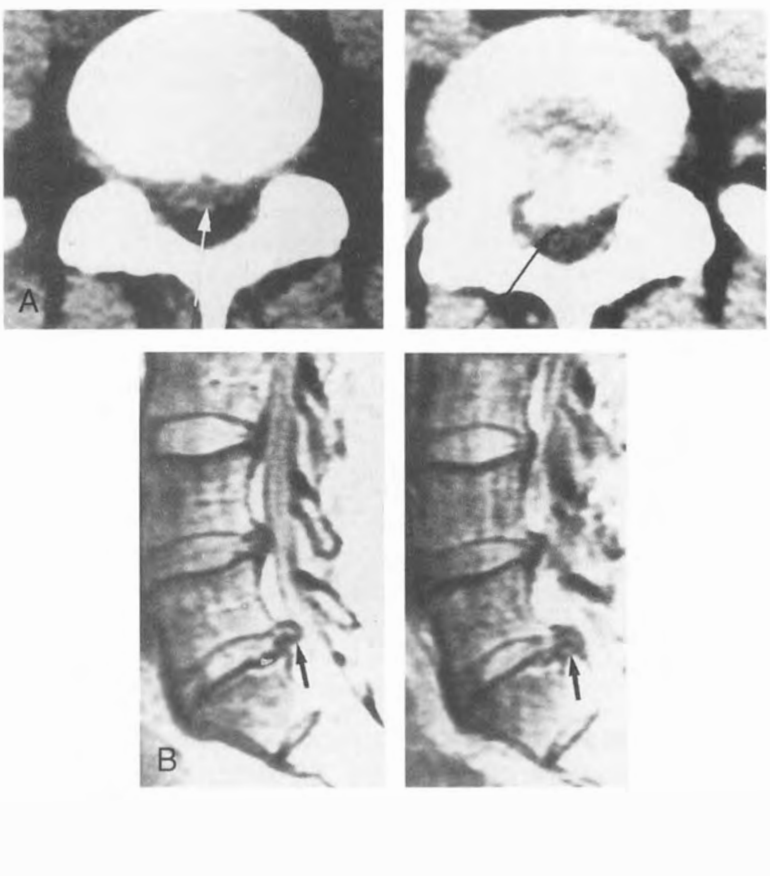


Figure 10.220. Calcified herniated nucleus pulposus (HNP) at L5–S1: Computed tomography versus magnetic resonance imaging (MRI) in two cases. **A.** Computed tomography of L4–L5 shows a large central HNP (*white arrow*) that contains dense calcification (*black arrow*) on the contiguous 3 mm slice. **B.** The sagittal MRI section (proton density and T2-weighted) shows the herniation (*arrows*), but the low-density border is not a conclusive finding for calcification. In these patients, the CT was necessary to conclusively demonstrate calcification. Awareness of calcification in a herniation is important if chemonucleolysis or percutaneous discectomy is being considered. In medicolegal litigation, a calcified herniation uncovered shortly (weeks or several months) after a traumatic episode is usually considered unrelated to the trauma. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:99.)

blood and tissue eosinophilia. Signs and symptoms include rash, dyspnea (often the presenting symptom), edema, neuropathy, leukocytosis, and elevated serum aldolase.

As of February 1991, 1543 cases had been reported from virtually every state and 28 deaths had occurred. The EMS outbreak resulted from the ingestion of a chemical constituent associated with specific practices used in the manufacture of tryptophan at one Japanese firm, Showa Denko. Tryptophan has been prescribed for management of insomnia, premenstrual syndrome, obsessive-compulsive behavior, and depression. The impurity may have resulted from the use of a new strain of organism, *Bacillus amyloliquefaciens* strain V, and/or the use of less powdered carbon in the manufacturing process (224).

Epidemiologic data, together with supportive results of studies in animals and rechallenges of patients with EMS with nonimplicated L-tryptophan sources, provide evidence that virtually all cases of EMS in the United States were linked to L-tryptophan produced by a single Japanese supplier (225).

Non-Hodgkin's Lymphoma of Epidural Space

Two patients developed sciatica caused by non-Hodgkin's lymphoma involving the spinal epidural space. Systematic investigation revealed no evidence of lymphoma in other sites. Non-Hodgkin's lymphoma typically affects the central nervous system late in its course. Involvement of the central nervous system occurs in approximately 10% of all cases, with compression of the spinal cord being the most serious complication. Central nervous system involvement as a presenting feature of lymphoma is rare. Although rare, isolated extradural non-Hodgkin's lymphoma should be considered in the differential diagnosis of sciatica (226).

Malignant Melanoma

Fifteen patients with symptomatic metastatic melanoma had severe back pain, and seven presented with neurologic findings. The interval between spinal involvement and death was 5.9 months (227).

Sarcoidosis

The possibility of intramedullary sarcoidosis presenting as a tumor should be included in the differential diagnosis of mass lesions of the spinal cord (228).

Sickle-Shaped Ligament Compression of L5 Nerve

Extraforaminal compression of the L5 nerve has been well documented. The lumbosacral ligament can cause this compression by entrapping the L5 nerve as it crosses over the sacral ala. The lumbosacral ligament was termed the "sickle-shaped ligament" by Danforth and Wilson in the original description of this structure. Surgical release of the sickle-shaped ligament has been advocated by Wiltse via a posterior paraspinal approach (229).

Obturator Internus Bursitis

Irritation of the obturator internus bursa (OIB) is identified as a common but thus far overlooked focus of myofascial irritability in association with low back pain.

In the maneuver that consists of a supine SLR test, with the affected extremity maximally adducted and internally rotated as the leg is straightened, the obturator internus and piriformis muscles are supporting the limb both stretching and contracting. This maneuver may produce irritation of the sciatic nerve at its pelvic outlet and irritation of the obturator internus muscle, the obturator internus bursa, or the piriformis muscle.

Tenderness in the anatomic locus of the obturator internus bursa, which presumably reflects obturator internus bursitis, is a common accompaniment of low back pain, particularly low back pain in association with regional myofascial irritability (230).

Intraneural Ganglion Cyst

Intraneural ganglion cyst of the peroneal nerve, diagnosed by ultrasound, which also gives the exact definition of its size and location, has been confirmed at operation (231).

Psoas Muscle Hematoma

Hematomas of the psoas muscle are a frequent complication of anticoagulant treatments (7%). The particular feature of hematomas in this site concerns the associated neurologic complication of femoral nerve paralysis. Although femoral nerve paralysis generally resolves, three cases have been reported that emphasize the occasionally serious outcome of these femoral nerve lesions. In two of these patients, the motor deficit only partially recovered, and in the third, the hematoma led to fatal hemorrhagic shock (232).

Subacute Bacterial Endocarditis

One third to one half of all patients with bacterial endocarditis have arthralgia, arthritis, low back pain, and myalgias that typically develop early, often preceding other manifestations of endocarditis. When musculoskeletal symptoms first appear, bacterial endocarditis would particularly be included in the differential diagnosis if the patient is older and has had a previously diagnosed heart murmur. Almost one third of patients with bacterial endocarditis have low back pain (233).

Prostatic Cancer

Although it is not a specific cause of sciatica, prostatic cancer can be implicated with low back pain and sciatica and deserves consideration in this section. Even with no treatment at all, less

than 10% of patients with localized disease die of it, and patients with a low-grade tumor have an even better prognosis. Unfortunately, once prostate cancer spreads beyond the gland, progression and death can occur in a matter of a few months, despite treatment (234).

Three tools for screening asymptomatic men have been proposed: digital rectal examination, prostate-specific antigen (PSA) determination, and transrectal ultrasound (234). In confirmed cases of bony metastasis from a prostatic primary carcinoma, serum acid phosphatase levels are normal in 20 to 25% of patients (235).

Figures 10.221–10.223 show an example of prostatic metastasis to bone in a 75-year-old patient with low back pain, left anterior and posterior lower extremity pain, and bilateral hip pain. Orchectomy had been performed for the prostate cancer and radiation treatment given for colorectal cancer. Figure 10.224 is another example of a sacral vertical alar fracture.

This patient was given chiropractic distraction adjustments, which relieved his lower extremity pain. Certainly, tolerance testing prior to manipulation as well as gentle technique was used; however, the case does illustrate the benefit of spinal adjustments in patients with advanced pathologies as long as the techniques are adapted to the condition.

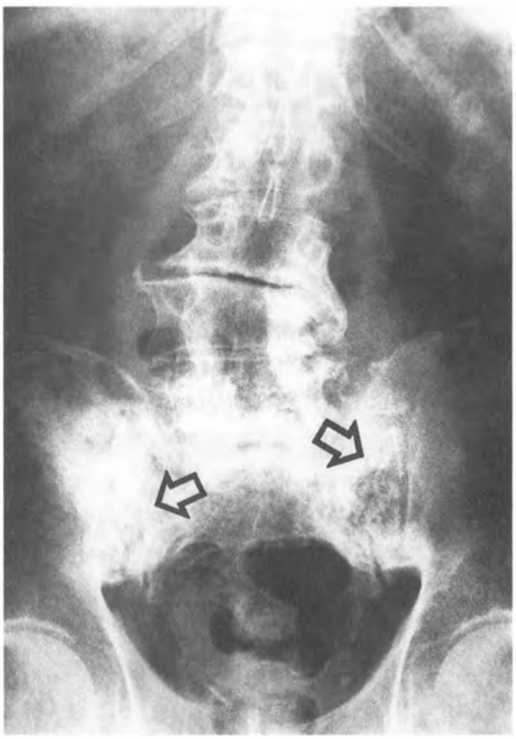


Figure 10.221. Anteroposterior lumbar spine and pelvic radiograph shows decompression laminectomy of the L3 to L5 levels with osteoblastic changes within the sacrum and right sacroiliac joint, indicating probable past radiation necrosis for colorectal cancer and prostatic metastasis. Also note the vertical oriented fracture lines through the sacrum (*arrows*) and see the computed tomography scan in Figure 10.223 for better observation of them.

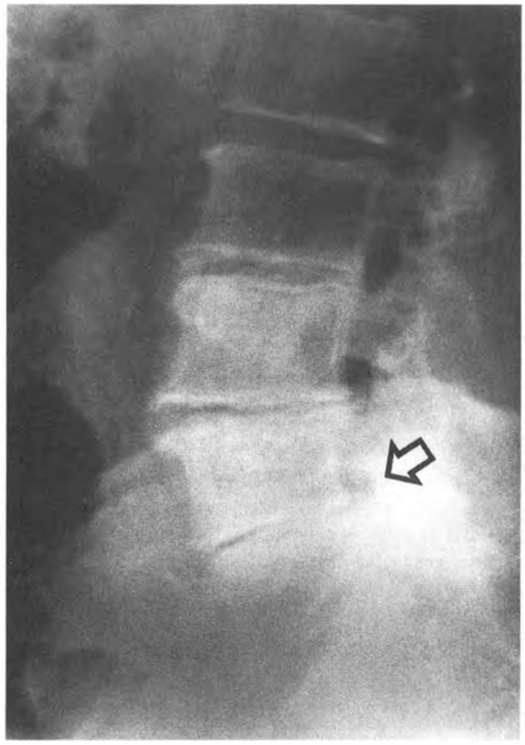


Figure 10.222. Lateral plain x-ray film shows the extensive disc degeneration and degenerative spondylolisthesis of L4 on L5 (*arrow*).

Figure 10.223. Computed tomography scan of the pelvis shows osteoblastic and radiation necrosis changes of the sacrum and ilia (*arrowheads*) as well as the vertical fracture line that parallels the sacroiliac joint on the right side (*open arrow*) and a suggestion of one on the left that is not as well delineated.

Polymyalgia Rheumatica

The mean age of onset of polymyalgia rheumatica is 70 years, and the disease is unusual in persons under the age of 50. About twice as many women as men are affected. Polymyalgia rheumatica is not a rare disorder. Prevalence has been estimated to be about 500 cases per 100,000 persons over the age of 50.

Patients with polymyalgia rheumatica usually present with

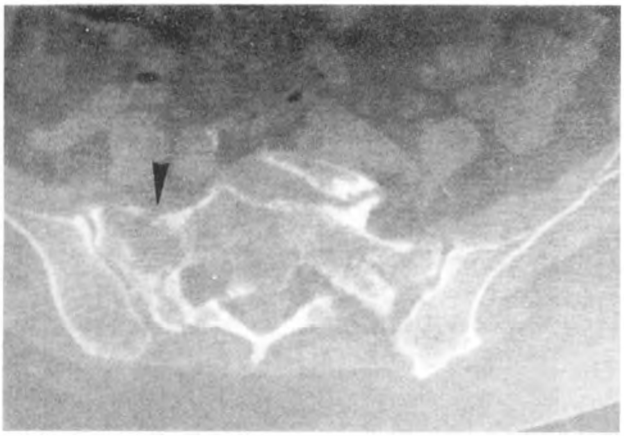


Figure 10.224. Representive computed tomography image through the first segment of the sacrum demonstrates a vertical right alar fracture (*arrow*) at window settings appropriate for bones. (Reprinted with permission from Leroux JL, Denat B, Thomas E, et al. Sacral insufficiency fracture presenting as acute low back pain. Spine 1993;18(16):2502–2506.)

acute pain in the shoulder and hip girdle that lasts for several months. They feel systemically ill and have morning stiffness, occasional weight loss, fever, and malaise. Evidence may be seen of mild synovial inflammation in the large joints, and even a rheumatoid arthritislike pattern of joint involvement. Temporal arteritis most commonly presents with headache with polymyalgia rheumatica. Pain is usually localized near the involved temporal artery, which may be tender to palpation and nodular (81).

Diabetic Radiculopathy

Diabetic radiculopathy commonly presents with severe unilateral pain of sudden onset that is usually located in the lower extremity, frequently in the proximal segments. Occasionally, bilateral asymmetric pain may be observed. Weakness of hip or thigh muscles, decreased sensation and hypo or areflexia are commonly observed. The clinical picture can resemble that of high lumbar disc herniation. Electrodiagnostic and radiologic studies can help differentiate between the two conditions (236).

Herpes Zoster Radiculopathy

Motor neuron involvement can occur in 1 to 5% of patients, and along with the radicular distribution of pain, it can mimic other clinical conditions including disc herniation, tumor infiltration, or infection. Urinary bladder involvement has been described in a few cases (237), and the dorsal root ganglion has been involved as well. Cutaneous lesions may or may not be present (238). Radiating pain, paresthesia, and motor and sensory loss may be seen as the virus inflames the sensory ganglia and posterior gray matter of the spinal cord. The cutaneous lesions of herpes may not be seen for 3 to 4 days after the onset of radicular symptoms. Early clues to diagnosing herpes are itching, burning, and tingling of the dermatome. Acute urinary retention may be present (239).

Ganglion Cyst of Posterior Longitudinal Ligament

Low back pain, bilateral L4 and L5 dermatome paresthesia, quadriceps weakness, and intermittent claudication occurred over a 2-year period in a 40-year-old man. CT showed a space-occupying, lobulated, gas-appearing lesion on the posterior wall of L3 vertebral body at the pedicular level (Fig. 10.225).

A 0.8 cm^2 well-encapsulated gas-filled cyst arising from the lateral edge of the posterior longitudinal ligament next to the pedicle of L3 was surgically removed. A ganglion cyst should be included in the differential diagnosis of the space-occupying lesions in this area (240).

Gas-Containing Lumbar Disc Herniation

Figure 10.226 is from a patient with bilateral leg pain, shown by CT scan and surgery to be an L4–L5 gas-containing disc herniation (241).

Intradiscal gas is associated with tumors, infection, trauma, therapeutic and diagnostic spinal procedures, and disc degeneration. The existence of gas within the spinal canal has been seen on 17 occasions of which 13 were associated with discal hernias (242).

Spina Bifida Occulta

Patients with spina bifida occulta (SBO)-S1 show a higher incidence of posterior disc herniation that can be explained by instability. Posterior disc herniation at L4–L5 or L5–S1 can be expected in most patients older than 18 years with low back pain or sciatica associated with SBO-S1 (243).

Endometriosis of Sciatic Nerve Causes Sciatica

When a sciatica is closely related to menses, consider cyclic sciatica resulting from endometrioma as a differential diagnosis

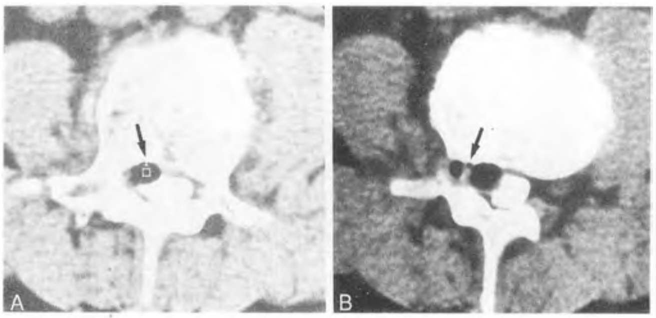


Figure 10.225. **A.** A low-density cystic lesion was noted on the posterior wall of the L3 vertebral body at the pedicular level. The density of the cystic content was extremely low with the absorption coefficient approximate to gas (*arrow*). **B.** The cyst was noted to be lobulated in the contiguous section (*arrow*). (Reprinted with permission from Lin RM, Wey KL, Tzeng CC. Gas-containing "ganglion" cyst of lumbar posterior longitudinal ligament at L3: case report. Spine 1993;18(16):2528–2532.)

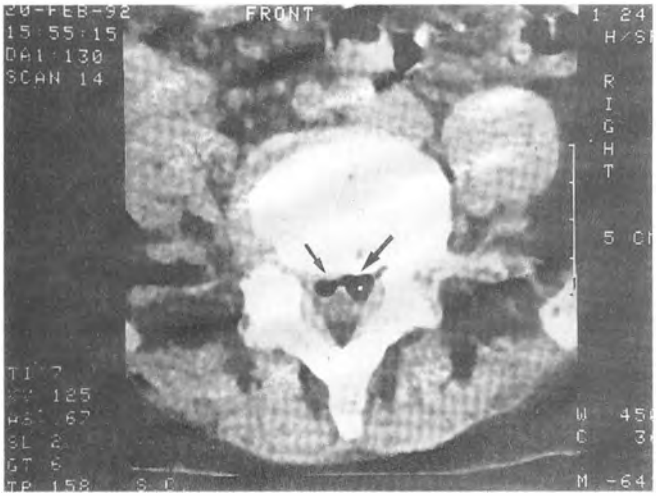


Figure 10.226. Lumbar computed tomography scan shows a paramedian bilobate low-density region of gas collection on this axial view (*arrows*). This gas escaped from the intervertebral nucleus pulposus, where the phenomenon of "vacuum phenomenon" is fairly common. (Reprinted with permission from Pierpaolo L, Luciano M, Fabrizio P, et al. Gas-containing lumbar disc herniation: a case report and review of the literature. Spine 1993;18(16):2533–2536.)

(244). Endometriosis of the sciatic nerve is rare, but must be included in the differential diagnosis of sciatic mononeuropathies. MRI may permit a specific diagnosis of this unusual cause of sciatica by showing a hemorrhagic mass in the region of the sciatic nerve (245).

Epstein-Barr Virus as Cause of Lumbosacral Radiculopathy

Six patients—five with lumbosacral radiculoplexopathy and one with femoral neuropathy—are reported in whom the neurologic symptoms coincided with elevation of antibody titers to various Epstein-Barr virus antigens (246).

Brown Tumor of Hyperparathyroidism Causes Sciatica

The first manifestation of hyperparathyroidism was a unilateral intraspinal cystlike lesion adjacent to the lamina and facet joint at the L4–L5 level producing sciatica. Histologic examination revealed multinucleated giant cells suggesting a brown tumor (247).

Cardiac Surgery as a Cause of Sciatica

In approximately 13% of patients undergoing cardiac surgery damage occurs to the peripheral nerve structures, usually in the upper limb, and brachial plexus lesions account for almost one half the total.

All the patients with sciatic nerve lesions had compromised blood flow through the femoral artery because of either an intraaortic balloon pump or a femoral artery thrombosis (248).

Posterior Apophyseal Ring Fracture

Posterior apophyseal ring fracture (PARF) of the lumbar spine is an uncommon injury thought usually to occur in adolescence (Fig. 10.227). Patients present with low back pain or sciatica caused by disc protrusion at L4–L5 or L5–S1. This is felt to be caused by relative weakness of the osteocartilaginous junction and firm attachment of the anulus fibrosus by Sharpey's fibers.

Figure 10.227 is from a 20-year-old woman with bilateral sciatica. Although usually found in adolescents, it has been described often in adults. It can occur without trauma or even strenuous exercise (249).

The radiologic appearances in young athletes with low back pain aged between 7 and 18 years were reviewed; 486 of 1696 patients had a total of 764 lumbar end plate lesions, 37(4.8%) of which arose from the posterior region of the lumbar end plate. In children and adolescents an end plate lesion appears to be caused by osteochondrosis of tissues that have been subjected to repetitive stress (250).

Idiopathic Epidural Lipomatosis

Pathologic overgrowth of epidural fat in the spinal canal has been described and reported almost exclusively in patients having long-term steroid treatment for a variety of clinical disorders. Idiopathic spinal epidural lipomatosis rarely is found in the absence of steroid treatment for obvious endocrinopathy.

Spinal epidural lipomatosis is most commonly found in the thoracic region, producing spinal cord compression. The second most common region in which it is found is the lumbosacral spine. For a patient with radicular pain or progressive paralysis who is obese, spinal epidural lipomatosis should be considered as a causative factor (251).

Primary Nerve Sheath Tumor

Nerve sheath tumors are the most common primary spinal tumors. In contrast, metastasis to the spinal nerve roots is rare. Metastatic tumors can clinically simulate other disease, and metastasis to spinal nerve roots can clinically mimic other diseases (252).

Cystic Meningioma

A 56-year-old hypertensive woman presented with low back pain of 3 week's duration with radiation to both legs. She had been prescribed nonsteroidal anti-inflammatory drugs and muscle relaxants without relief. Over the week before admission, she complained of worsening leg pain and weakness while walking. The deep tendon reflexes were decreased at the knees and absent at the ankles. Straight leg raising was limited to 45° bilaterally. The leading clinical diagnosis was a herniated nucleus pulposus (Fig. 10.228).

The pathogenesis of cyst formation in meningiomas remains obscure. Postulated mechanisms include central necrosis and cystic degeneration, active secretion of fluid by tumor cells,

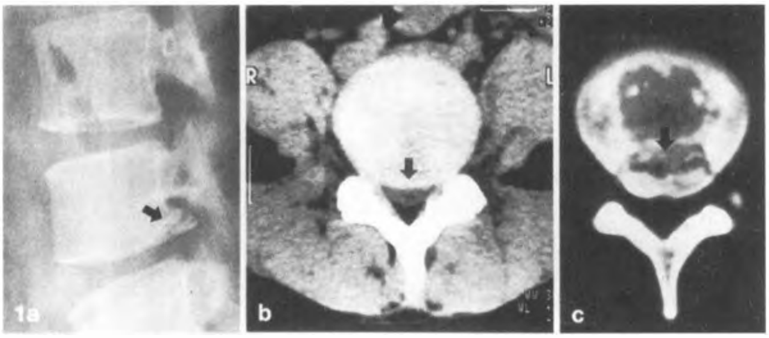


Figure 10.227. Posterior apophyseal ring fracture (*PARF*) at the center of the inferior rim of L4. **A.** Lateral radiograph shows PARF (*arrow*) involving the inferior rim of L4. **B** and **C.** Computed tomography at the disc level (**B**) and above (**C**) show diffuse disc protrusion (*arrow*) and a large broad-based bone fragment protruding into the spinal canal from the central aspect of the posterior margin (*arrow*), respectively. (Reprinted with permission from Yang IK, Bahk YW, Choi KH, et al. Posterior lumbar apophyseal ring fractures: a report of 20 cases. Neuroradiology 1994;36:453–455.)

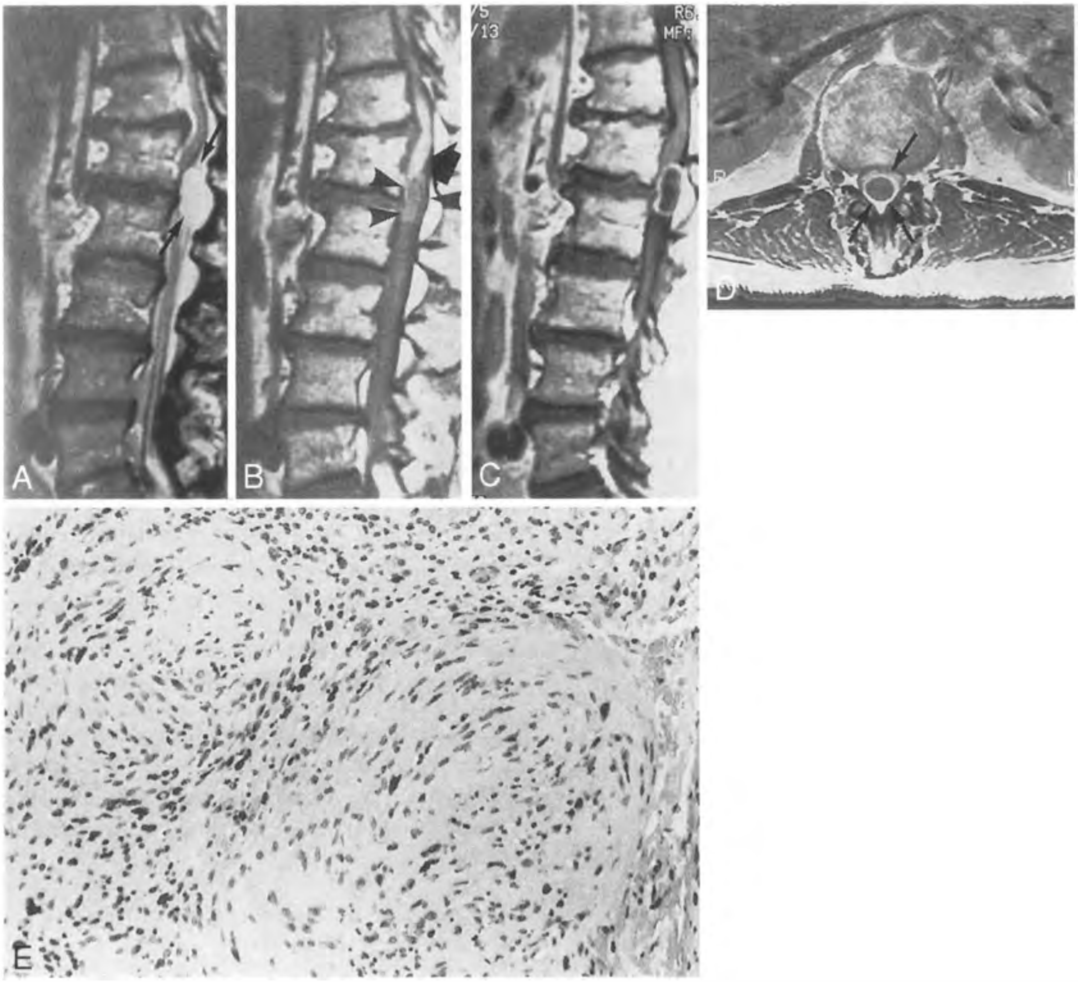


Figure 10.228. Cystic meningioma. **A.** A sagittal T2-weighted (2200/96) image showing a sharply delineated intradural lesion, with "capping" (*arrows*) on the superior and inferior aspect, at L1–L2. **B.** A sagittal T1-weighted (650/11) image showing that the mass (*arrowheads*) gives a slightly higher signal than the cauda equina and a lower signal than the conus medullaris, which is displaced anteriorly (*arrow*). **C.** The T1-weighted image after intravenous diethylenetriamine pentaacetic acid (Gd-DTPA) showed a ring-enhancing mass with low signal cystic center. **D.** The axial contrast-enhanced T1-weighted (750/15) image showing a well-defined enhancing ring (*arrows*) with a center of similar intensity to cerebrospinal fluid. **E.** A photomicrograph of the wall of the tumor showing whorls of meningiothelial cells with indistinct cell borders with intranuclear inclusions, characteristic of syncytial meningioma (hematoxylin and eosin magnification x29) (Reprinted with permission from Chynn EW, Chynn KY, DiGiacinto GV. Cystic lumbar meningioma presenting as a ring enhancing lesion on MRI. Neuroradiology 1004;36:460–461.)

and proliferating glial cells, evolutions of cerebral edema, and loculation of CSF (253). Sciatica can be the chief complaint of meningioma (254).

Multiple Myeloma Diagnosed with MRI

Multiple myeloma is a proliferation of malignant plasma cells that usually affects the bone marrow. The ability of MRI to depict changes in the bone marrow has been well documented. On T1-weighted images, 79% of the lesions were hypointense relative to muscle, and the remainder were hyperintense. MRI may be promising for assessing response to treatment, especially in patients with nonsecretory myeloma (255).

Baker's Cyst Compresses the Tibial Nerve

Baker's cysts, which are commonly found in severe polyarthritis, develop when strong positive pressures produced within the knee result in the rupture of the joint capsule, resulting in compression of the tibial nerve or the nerve to the medial belly of the gastrocnemius muscle (256).

Acquired Immunodeficiency Syndrome in Acute Lumbosacral Polyradiculopathy

Twenty-three patients with acquired immunodeficiency syndrome (AIDS) had acute lumbosacral polyradiculopathy. Neurologic complications are common in patients with human immunodeficiency virus (HIV) infection. Patients present with rapid progression of bilateral leg weakness that sometimes leads to paraplegia within several days. Leg areflexia, sphincter dysfunction, and CSF abnormalities are early and frequent findings (257).

Hamstring Muscle Scarring Entraps the Sciatic Nerve

Hamstring muscle tearing at the ischial tuberosity can result in scarring that will encase the sciatic nerve causing motor and sensory changes in the lower extremity (258).

Rheumatoid Arthritis

Clinical Laboratory Testing

Complete blood count, erythrocyte sedimentation rate and rheumatoid factor (RF) assay, and antinuclear antibody (ANA) assay are laboratory tests often used to evaluate patients with signs and symptoms compatible with rheumatoid arthritis (RA). Approximately 70% of patients with RA have positive test results for serum RF, a group of proteins that represent autoantibodies of immunoglobulins IgG, IgA, or IgM isotope and react with autologous IgG. A strongly positive test result for RF (at a dilution of 1:320 or above) helps to strengthen the initial suggestion of RA. Thirty to forty percent of older persons may have a weakly or moderately positive RF test result without manifesting any obvious clinical disorder.

An ANA and chemistry profile are not essential for diagnosing RA, but they are useful for monitoring the patient's subsequent progress and possible adverse reaction to various therapeutic agents. Many patients with established RA have positive test results for the presence of ANA.

Perinuclear antibodies have been found in about 78% of patients with classic (IgM RF-positive, subcutaneous nodules) RA and in 40% of patients with IgM RF-negative RA (259).

Methotrexate-induced Lymphoma When Treating Rheumatoid Arthritis

Two patients with longstanding seropositive RA treated with oral methotrexate (MTX) developed large cell lymphoma of B cell phenotype. Epstein-Barr virus (EBV) was found within the malignant lymphoid cells. In both cases, the lymphoma was undetectable several weeks after diagnostic biopsy followed by discontinuation of MTX. These observations suggest that, in patients with RA who develop an EBV-associated lymphoproliferative disorder, a trial discontinuation of immunosuppressive agents may be warranted before chemotherapy is considered. In addition, a need is seen for a heightened awareness of the development of lymphoma in this patient population (260).

Abdominal Aneurysm

A 58-year-old man presented with low back pain, and radiographs revealed an abdominal aneurysm. Note the calcific expansion of the atherosclerotic abdominal aorta, measuring 4.5 cm in diameter (normal is 1.75 to 3.0 cm) (Figs. 10.229 and 10.230). Treatment consisted of surgical care.

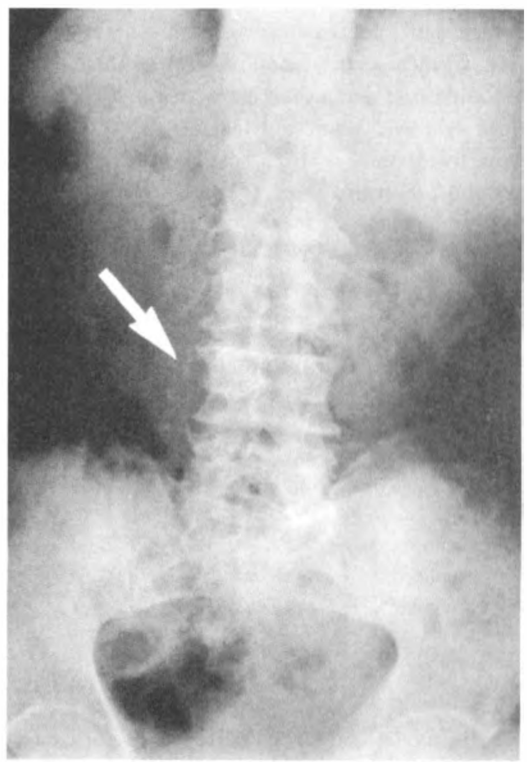


Figure 10.229. Left aortic expansion on anteroposterior view (*arrow*).

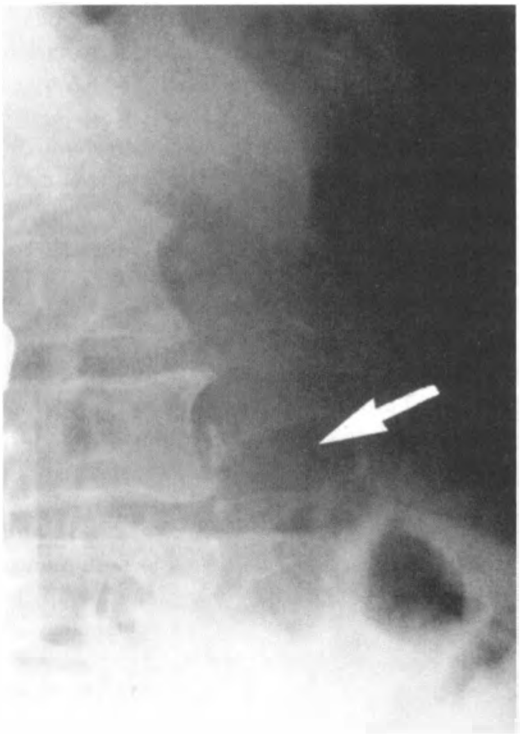


Figure 10.230. Arteriosclerotic expansion on oblique projection. (*Arrow* shows aneurysm.)

All physicians must be aware of the study (261) in England finding an abdominal aneurysm in 3% of those over 50 years of age, which caused death in 1.5% of cases. In patients with other manifestations of arteriosclerosis, 9.5% have an abdominal aneurysm. Clinical examination may miss a third of them. Statistics on untreated aneurysms show that half of these patients were dead within 2 years and that 60 to 80% of those with symptoms lived only 1 year. Small aneurysms rupture and grow about 4 to 5 mm a year.

Acute Aneurysm May Present as Femoral Neuropathy

A leaking aneurysm may present as an acute femoral neuropathy from retroperitoneal compression of the femoral nerve roots (262).

Surgery, as opposed to watchful waiting, is recommended for abdominal aneurysms less than 5 cm in diameter. Watchful waiting is generally favored for patients with a low risk of aneurysm rupture, including those with a an aneurysm less than 4 cm in diameter. More accurate data concerning the rupture risk of abdominal aneurysms less than 5 cm are needed, which would improve clinical decision-making (263).

Osteomyelitis of the L3–L4 Disc

A 41-year-old man complained of generalized lower back pain, especially on the left side from L3 to the sacroiliac region, radiating down the anterolateral left thigh and leg. Movement aggravated the pain, and rest relieved it. The patient presented with loss of lumbar lordosis and a mild left list of the thoracolumbar spine.

History revealed that the back pain first occurred when the patient was getting out of his car 18 days prior to seeking care. He had seen an osteopathic physician, who used manipulation, with no relief. A medical doctor prescribed Motrin and muscle relaxants, with no relief.

Findings on chiropractic workup were left spinal tilt; loss of lumbar lordosis; positive Minor's, Bechterew's, and Valsalva signs; pain on palpation over the L3–L4 left lumbar area; Kemp's sign positive bilaterally; toe and heel weak normal; SLR positive at 45° for low back pain; Patrick's sign positive for hip pain; and Gaenslen's sign positive for low back pain. Deep tendon reflexes were +2 bilaterally, motor findings were normal, and sensory examination was normal.

The impression at the time was an L3–L4 disc protrusion with L4 dermatome paresthesia. Treatment with flexion-distraction manipulation and therapy gave relief.

The patient then returned to weightlifting, and the pain worsened. A surgeon examined the patient and agreed with the diagnosis of a midline and left L3–L4 disc rupture. A myelogram was done which was indeterminate because of the subdural injection of the contrast media. A CT scan was done and interpreted as normal. Plain x-rays films were read as only showing minimal hypertrophic changes of the lower lumbar spine. A bone scan (Fig. 10.231) showed moderate uptake at the L3–L4 level. The patient was released from the hospital.

Two weeks later, as the pain grew worse, the patient readmitted himself to the hospital. His blood tests revealed a sedimentation rate of 116, and gram-positive cocci (*Staphylococcus aureus*) were cultured. A CT scan (Fig. 10.232) now showed destruction of the L3 and L4 vertebral body plates and cancel-

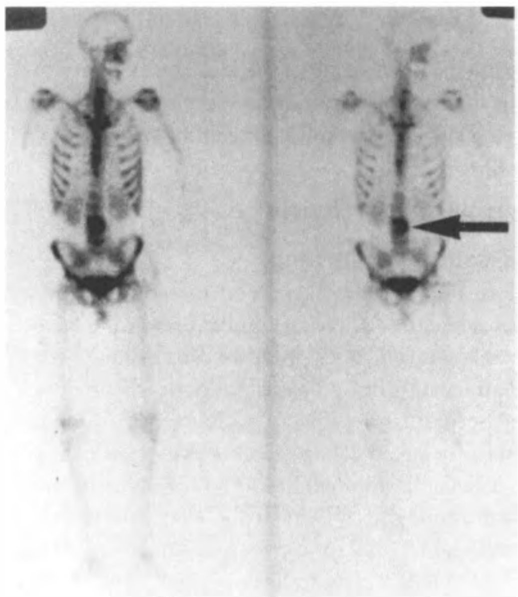


Figure 10.231. Bone scan reveals increased uptake of the left L3–L4 vertebral level (*arrow*).

lous bone with the loss of disc space. Figure 10.233 is the lateral view of the lumbar spine showing the L3–L4 disc space narrowing and the reactive periostitis of the opposing vertebral body plates indicative of infectious spondylitis.

The final diagnosis was osteomyelitis of the L3 and L4 vertebrae and intervertebral disc. The patient responded well to antibiotic therapy and, after healing, underwent chiropractic flexion-distraction manipulation because of persistent stiffness and pain.

This case is a good example of how the symptoms and signs of an organic illness mimicked a disc lesion and misled several clinicians until the disease revealed itself.

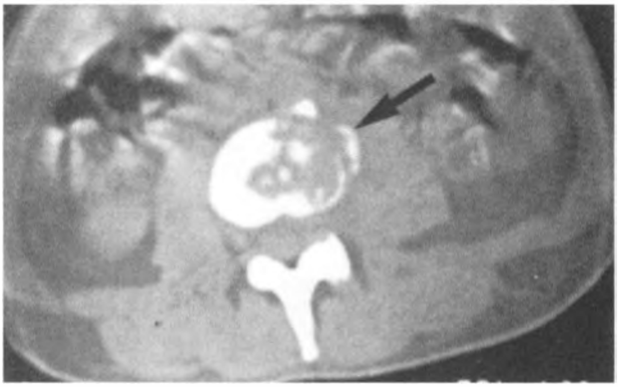


Figure 10.232. Computed tomography scan shows destruction at the left L3 vertebral body with soft tissue swelling and bone density paravertebrally into the soft tissues (*arrow*). (Case courtesy of Walter P. Kittle, DC.)

Figure 10.233. Lateral projection reveals the same finding as in Figure 10.232 (*arrow*).

Congenital Hip Dislocation

This 11-year-old girl was seen because her gym teacher noted a strange gait pattern. Indeed, she had a "duck-waddle" gait. The pelvis appeared widened, and the lumbar spine appeared markedly lordotic. The abdomen protruded somewhat. The patient denied any problem in locomotion.

Figures 10.234 and 10.235 are the anteroposterior and lateral hip projections revealing bilateral dislocation of the hips. The femoral heads rest against the lateral wall of the ilii.

The cause of this condition is unknown, but it is known to involve several members of the same family. Females are affected approximately 9:1 more than males, and the condition is especially prevalent in the Mediterranean countries, notably Italy.

This is an unfortunate case of bilateral hip dislocations which was allowed to go undiagnosed until seen by a chiropractor.

Spondylitis

A 36-year-old woman complained of weakness of the left lower extremity. Figure 10.236 reveals a destructive bone and intervertebral disc lesion at the right T4–T5 level. Figure 10.237 is the CT scan, which reveals marked destruction of the T4 vertebral body and a large soft tissue abscess that proved to be tubercular spondylitis.

This is a good case to alert one to organic causes of leg pain and weakness.

Figure 10.234. Anteroposterior pelvic x-ray study shows bilateral hip dislocations. (Case courtesy of David Galken, DC.)

Figure 10.235. Frog-leg x-ray study of the pelvis shows bilateral hip dislocations.

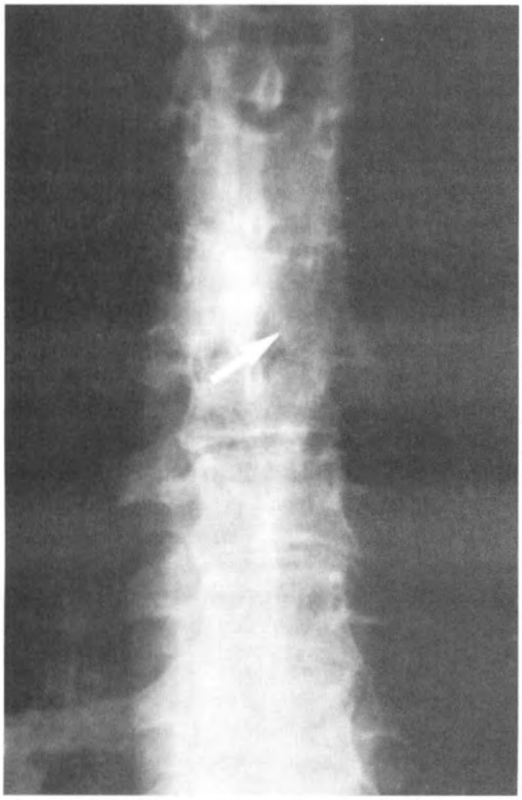


Figure 10.236. A destructive bone and intervertebral disc lesion is noted at the left (*arrow*) T4–T5 level (tubercular spondylitis). (Case courtesy of Gary Guebert, DC, DACBR.)

Avascular Necrosis of the Hips

The following is a case of avascular necrosis of both femoral heads. Figure 10.238 reveals increased radiopacity at the superolateral weightbearing portions of both femoral heads, appearing as a wedge-shaped area. The joint space appears well maintained. Figure 10.239 is an MRI study which, unlike the plain film, does show some loss of the sharp cortical definition of the femoral head at its articulation with the acetabulum, and a decreased signal intensity in the superior aspect of both femoral heads. Some joint space narrowing may be seen on the MRI study and an irregularity of the cortical outline superior to the area of avascular necrosis.

This condition is seen predominantly in men, usually in the fourth and fifth decade of life. Pain is the chief symptom, which begins around the hip or radiates into the thigh of knee joint. A limp may be associated with it, and a history of slight trauma or no trauma at all may be elicited.

Mitchell et al. in 39 consecutive patients with avascular necrosis of the femoral head, representing 56 total hips, the condition to be caused by steroid administration in 31 of the patients, ethanol abuse in 6, fracture dislocation of the hip in 1, therapeutic radiation for lymphoma in 1, and idiopathic in 17 (264).

The radiographic stages of avascular necrosis are defined by Steinberg et al. (265):

1. Normal radiographic findings.
2. Cystic and/or sclerotic changes without subcortical lucency (crescent sign).
3. Development of subchondral lucency and subchondral fracture, as evidenced by the crescent sign.
4. Subchondral collapse, depicted as flattening of the femoral heads.
5. Narrowing of the hip joint.

Magnetic resonance imaging appears to be more sensitive than bone scans for allowing diagnosis of early avascular necrosis. Pomeranz (266) would classify this case as a stage 2 avascular necrosis of the left hip joint.

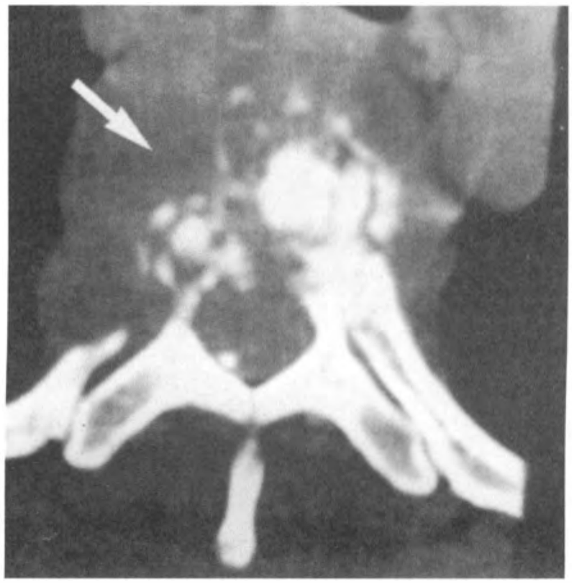


Figure 10.237. Computed tomography scan of the patient seen in Figure 10.236 shows extensive vertebral body destruction and a large soft tissue abscess extending into the chest (*arrow*).

Treatment

Avascular necrosis of the hip can be treated thorough debridement and cancellous bone grafting in young patients with stage 2 or stage 3 disease that will delay, if not prevent, the progression of osteoarthrosis and subsequent total hip arthroplasty (267). Total hip replacement, regardless of intermittent treatment, seems to be the eventual outcome of this condition.

Core decompression may be effective in symptomatic relief, but is of no greater value than conservative management in preventing collapse in early osteonecrosis of the femoral head (268).

Disease progression was studied in the asymptomatic hip of 19 patients with nontraumatic osteonecrosis and pain in the other hip who were followed for 5 years. Five were still asymptomatic and 14 had become painful. Less than half of the asymptomatic hips with radiographic evidence of osteonecrosis developed pain. This suggests a slow progression of the disease in nontraumatic osteonecrosis. Approximately 1.3 of asymptomatic hips that show initial radiographic involvement will have a total hip arthroplasty. A clinical question remains to whether the contralateral hip is truly free of disease or whether it escapes radiographic detection. A favorable outcome can be expected for most asymptomatic hips with normal findings on radiographic examination, which suggests routine use of diagnostic tests (e.g., intraosseous manometry) and the need for operative treatment is not necessary. Whether early detection of MRI signal change in asymptomatic hips with normal radiographs will lead to improved outcomes remains to be determined (269).

Figure 10.238. Both femoral heads show increased radiopacity and cystic changes of the superolateral weight-bearing portions as a wedge-shaped area (*arrows*). The joint space is maintained. (Case courtesy of David Taylor, DC.)

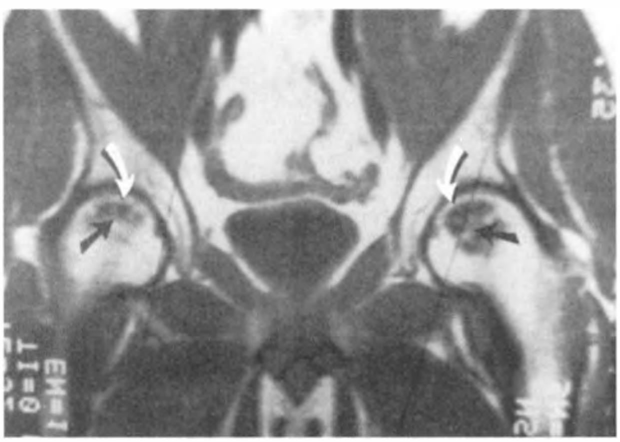


Figure 10.239. Magnetic resonance image shows marked signal intensity loss of both femoral heads (*straight arrows*), with irregular cortical outline at the articular surface (*curved arrows*).

Necrotic Material Percentage Determines Chance of Collapse

The hypothesis that the extent of necrosis at the initial MRI predicts the subsequent risk of collapse of the femoral head in avascular necrosis was tested. The arc of the necrotic portion in the midcoronal image and that in the midsagittal image were used to quantify the extent of necrosis by the formula:

$$(A/180) \times (B/180) \times 100$$

A strong correlation was found between this index and the risk of collapse before and after adjustment for age, gender, stage, and treatment group.

The index of necrotic extent was classified into three categories according to the values calculated based on the formula given above: grade A, small necrosis, ≤ 33; grade B, medium necrosis, 34 to 66; and grade C, large necrosis, 67 to 100. Hips with necrosis of less than 30% fall into a low-risk group, and those with 30 to 40% in a moderate risk-group, and those with more than 40% in a high-risk group (270).

The principal clinical problem with osteonecrosis is the segmental collapse of the femoral head. Spontaneous regression of the necrotic lesion in 14 (45%) of 31 hips with bandlike zones of necrosis showed incomplete regressive changes or returned to normal (271).

Stress Fracture of Metatarsal Bone

Figure 10.240 reveals a stress fracture of the second metatarsal bone. Note the osteodegenerative arthrosis of the first metacarpal phalangeal joint, which is the result of past hallux valgus bunion surgery. Following the surgery, this patient had a limp that probably resulted in stress on the second metatarsal bone, leading to the eventual stress fracture and the callus formation that is now seen. This case is presented to alert us again to the possibility of a pathologic cause of low back, leg, or foot pain.

Osteomyelitis

Figure 10.241 shows a pelvic radiograph of a 6-year-old boy who had been hospitalized for the treatment of staphylococcal

Figure 10.240. The distal second metatarsal bone reveals callous formation of a stress fracture (*straight arrow*). Note the arthrotic degeneration of the first metatarsophalangeal joint following surgery for hallux valgus (*curved arrow*).

Figure 10.241. A small radiolucent nidus in the femoral neck of a 6-year-old boy (*arrow*).

pneumonia for 2 weeks prior to this study being taken. Noted is a radiolucent nidus somewhat surrounded by an area of radiopacity within the right femoral neck.

Figure 10.242 reveals osteomyelitis; this study was taken 3 weeks following that shown in Figure 10.241. Seen is a hematogenous spread of the staphylococcal bacteria into the right femur, which demonstrates how rapidly osteomyelitis can fulminate.

Harrington Rod Fracture

Figures 10.243 and 10.244 reveal a fracture of the Harrington rod at the junction of the ratchet and the remaining rod.

This female patient had this rod placed in her spine approximately 8 years prior to this fracture. The fracture was identified only on a routine chest x-ray study for an upper respiratory infection. The patient had no spinal symptoms caused by the fractured Harrington rod.

Note that these rods typically fracture at an area of pseudoarthrosis, meaning that the fusion of the scoliotic curve did not take place firmly at that level, placing more stress on the rod, with its eventual fracture. It is also again noted that this fracture usually occurs at the level of the junction of the ratchet section with the rest of the rod.

Metastatic Carcinoma

A 61-year-old woman was seen complaining of low back pain. Radiographs of the lumbar spine (Figs. 10.245 and 10.246) reveal the right L1 pedicle to be absent, with loss of the vertebral body height and increase in the sagittal diameter of the vertebra. Also seen is some laceration in bone architecture, with areas of radiolucency mixed with areas of increased opacity of bone, which probably represents compaction caused by compression change. Figure 10.247 is a spot film of the first lumbar

Figure 10.242. Full-blown osteomyelitis of the right femur shown in a radiograph taken 3 weeks following that in Figure 10.241. (Case courtesy of Gary Guebert, DC, DACBR)

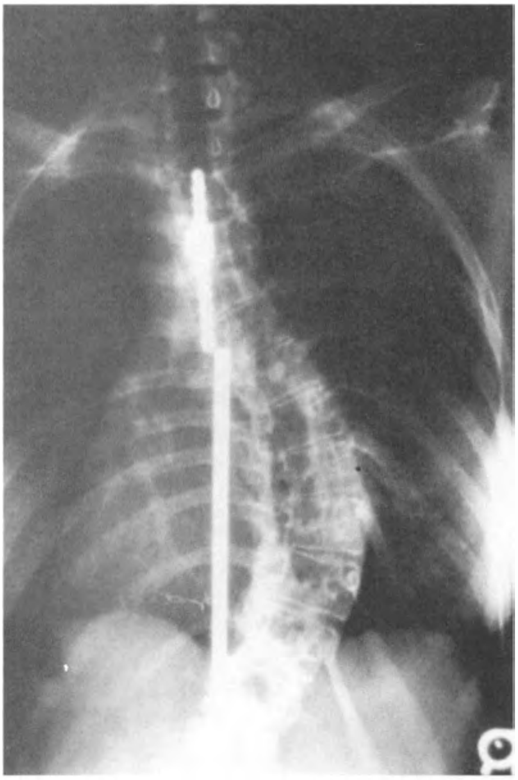


Figure 10.243. Fracture of a Harrington rod at the area of pseudoarthrosis in a scoliotic fusion.

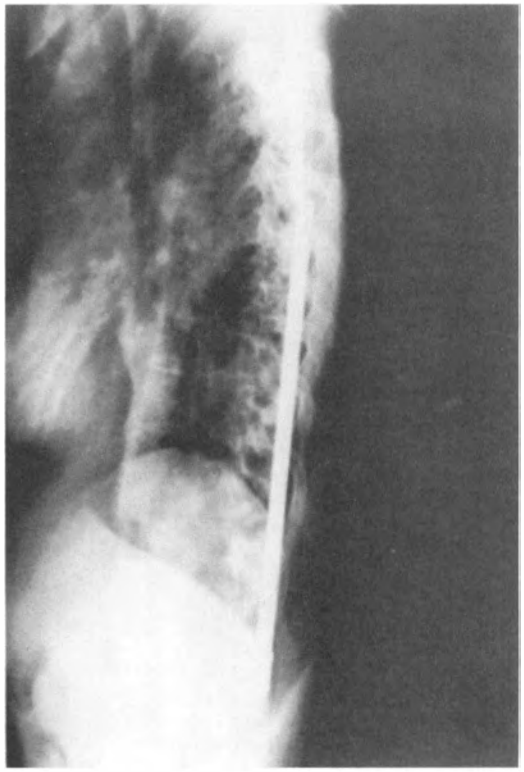


Figure 10.244. Lateral view of patient shown in Figure 10.243.

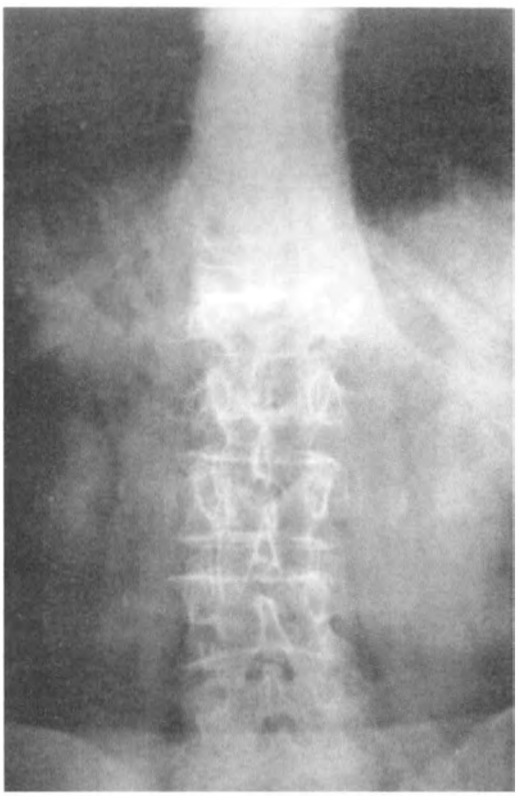


Figure 10.245. Posteroanterior lumbar spine radiograph shows absence of the right L1 pedicle ("one-eyed jack" sign) with loss of height of the lumbar vertebral body on the right (*arrow*).

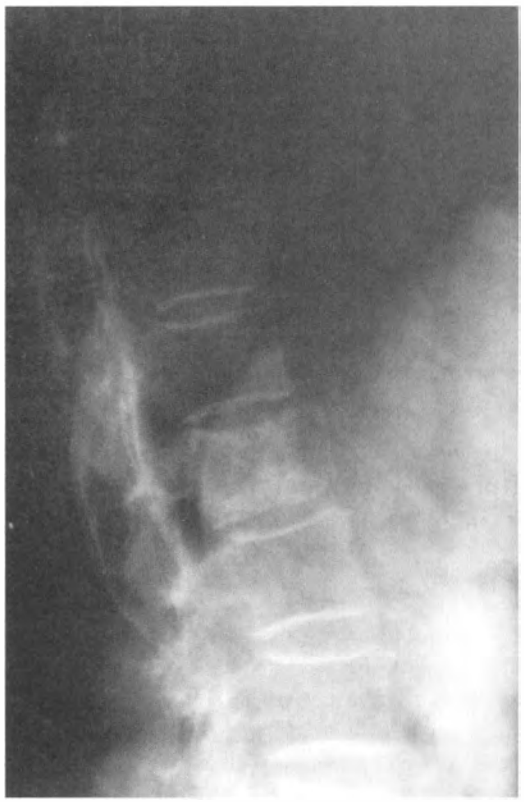


Figure 10.246. Lateral projection reveals loss of bone architecture, irregular bone outline, and radiolucency of bone of the first lumbar vertebral body.

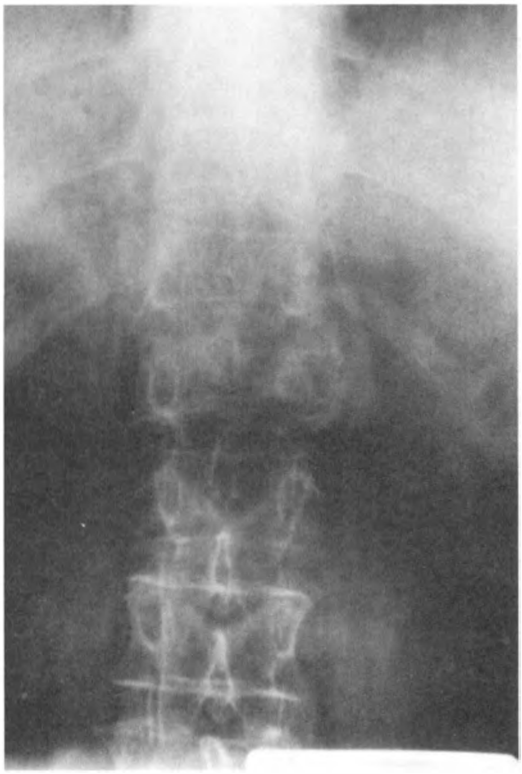


Figure 10.247. Spot film of patient shown in Figure 10.245.

vertebra in posteroanterior position that reveals the change in the right first lumbar vertebral body and pedicle.

History revealed that, 2 years prior to this onset of low back pain, the patient had a breast removed for carcinoma.

Figure 10.248 is a CT scan through the first lumbar vertebral body, which again reveals the alteration of bone architecture, with radiolucency throughout the vertebral body extending into the right pedicle. Figure 10.249 is an MRI study that reveals not only the altered bone architecture but also the extension of the posterior L1 vertebral body into the vertebral canal, which is creating a stenotic change at that level.

Treatment in this case consisted of radiation, and at last history this patient had a remission of the malignancy.

Normal Plain X-Ray Study of L2 Vertebral Body with Abnormal MRI of L2

Figure 10.250 shows degenerative L3–L4 disc changes. Figure 10.251 shows the same L3–L4 disc degeneration, and the inferior L2 vertebral plate reveals some nuclear invagination of its inferior body plate. Figure 10.252 shows a bone scan that was ordered since this patient continued to have night pain and unremitting low back pain. Here is seen that the L2 vertebral body has increased uptake, as well as two sites on the left paralumbar area that are felt to be within rib tissue. Figure 10.253 is an MRI that shows the L2 vertebral body to have low T1-weighted signal intensity in comparison to the adjacent vertebrae. The superior plate of L4 has a superior compression defect, a probable Schmorl's node. Also seen is an abdominal aneurysm with a large hematoma within it, anterior to the L3–L4 vertebral bodies.

At the time of this writing, the diagnosis of this case was not final, but a malignant disease was the primary impression.

This case again demonstrates the lack of diagnostic detail from plain x-ray film and supports the need for further detailed imaging in cases having unremitting pain under conservative care, especially when clinical findings are present.

Meralgia Paresthetica

In this condition the lateral femoral cutaneous nerve produces uncomfortable paresthesias and sensory impairment in its cutaneous distribution because of a benign entrapment (272). The point of entrapment is usually at the inguinal area where the nerve pierces

Figure 10.248. Computed tomography scan shows mixed radiolucent and radiopaque changes of the first lumbar vertebral body.

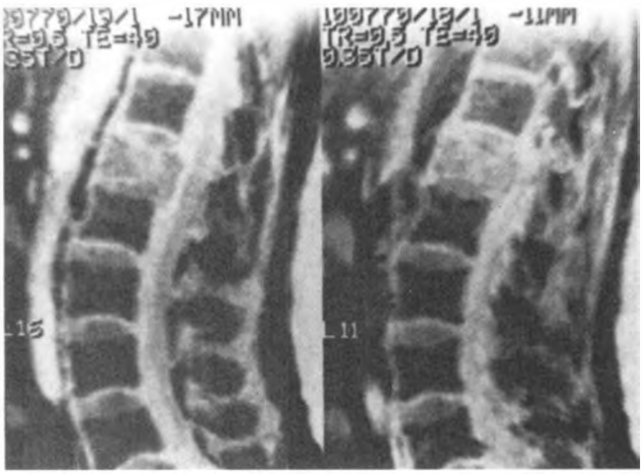


Figure 10.249. Magnetic resonance image reveals loss of signal intensity and vertebral height and extension of the L1 vertebral body posteriorly into the vertebral canal to create stenosis and possible compression of the conus medullaris area of the spinal cord.

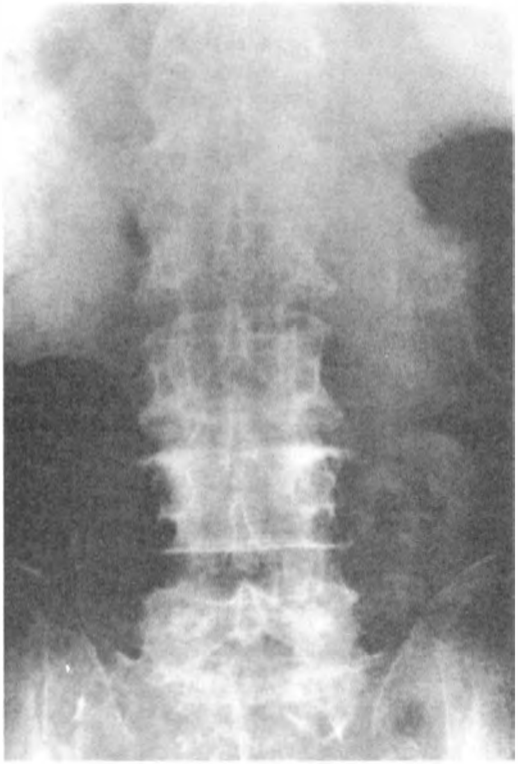


Figure 10.250. This study shows L3–L4 intervertebral disc degeneration. Sclerosis of the L4 superior vertebral body plate is seen.

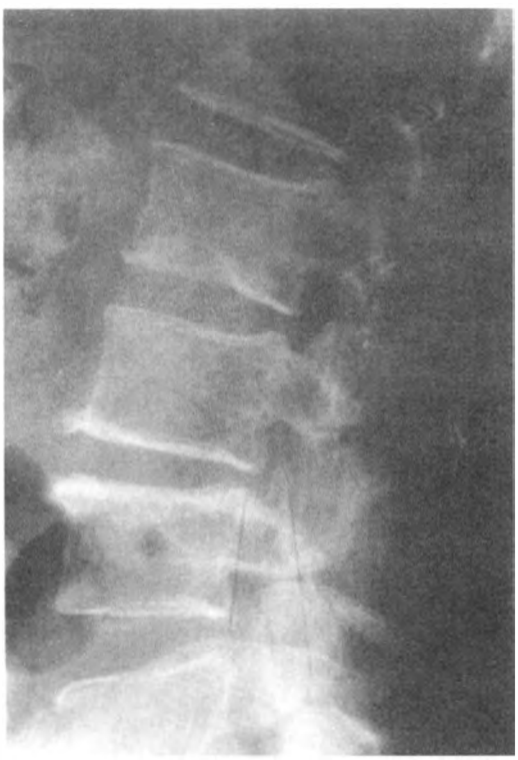


Figure 10.251. Lateral view of the spine seen in Figure 10.250 again shows L3–L4 discal degeneration. The inferior plate of L2 reveals nuclear disc invagination.

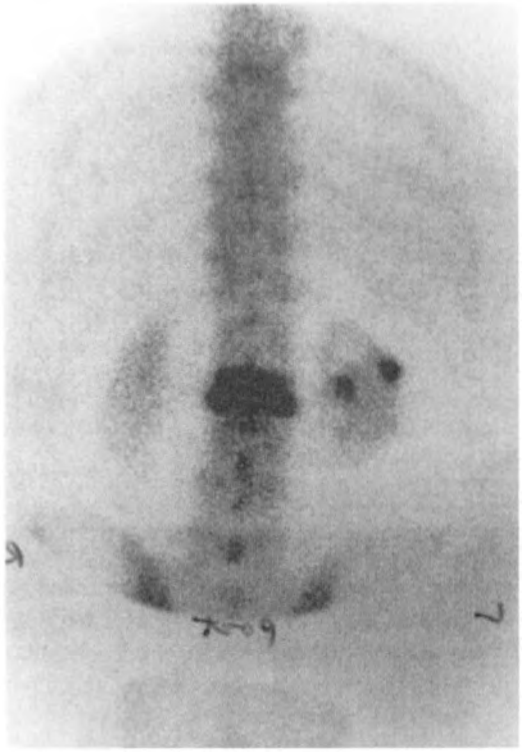


Figure 10.252. Bone scan shows increased uptake of radionuclide at the L2 vertebral body and the left lower two ribs.

Figure 10.253. Magnetic resonance imaging shows that the L2 vertebral body has lost signal intensity compared with the other lumbar vertebrae. The superior plate of L4 also shows a compression defect not appreciated on other plain films. Note also the large aortic aneurysm with a blood clot within it lying anterior to the L3–L4 vertebral bodies.

the ligament to enter the thigh at or near the anterior superior iliac spine. Trauma to the pelvic bones, scarring of the inguinal ligament, diabetes mellitus, obesity, toxic neuropathy (e.g., alcohol or drug), pregnancy, or tight clothing have been implicated in the etiology. To diagnose meralgia paresthetica, somatosensory evoked potentials (SSEP) have shown great benefit (272–277).

Headache with Chronic Low Back Pain

In a study of patients with chronic low back pain 75.2% reported that headache co-occurred with low back pain or emerged as a sequela of it. Patients with chronic low back pain should be screened routinely for the presence of clinically significant headache, including migraine headache, so that adequate treatment can be provided (278).

Lumbar Synovial Cyst

Most lumbar intraspinal facet cysts are associated with significantly degenerated facet joints. Patients with intraspinal facet cysts may respond to conservative treatments if no significant neurologic deficit is present. Surgical decompression and removal of large facet cysts usually is successful in relieving symptoms (279).

Low back pain and symptoms from unilateral nerve root involvement in lumbar synovial cyst formation are the most frequent signs. The L4–L5 facet joints are most frequently involved, and most prevalently in females. Treatment is usually, medical, surgical, or with corticosteroid intra-articular injection (280). An unusual case of hemorrhage into a right L3–L4 synovial cyst causing an acute cauda equina syndrome has been reported (281).

Synovial cyst, also termed a "ganglion cyst" in the past, is now termed "pigmented villonodular synovitis," the correct term for hypertrophic synovitis of the facet joint (282).

A 76-year-old woman was seen complaining of left buttock and posterior thigh pain extending to the knee. Night pain was present. MRI axial view (Fig. 10.254) and sagittal view (Fig. 10.255) showed the degenerative L4–L5 facet disease with protrusion of the synovial cyst into the left posterolateral vertebral canal space to contact the thecal sac (*arrows*).

Sequestered disc mimics synovial cyst. This case was treated with positive galvanic current into the cyst followed by distraction manipulation of the L4–L5 facet joints with complete relief of the left buttock and thigh pain.

A 70-year-old patient with a rare, misleading presentation of lumbar disc prolapse, which on CT mimicked a synovial cyst, later showed surgically that the whole nucleus pulposus had herniated, become sequestrated, and migrated behind the theca adjacent to the L4–L5 facet joint. No continuity was seen of the disc material with the intervertebral space. The patient had complete postsurgical relief from his pain (283).

Compression Fracture Caused by Osteoporosis

One condition commonly seen in elderly patients is osteoporosis of the spine, which carries with it the risk of compres-

Figure 10.254. Magnetic resonance image axial view shows the synovial cyst of the left facet capsule (*arrow*).

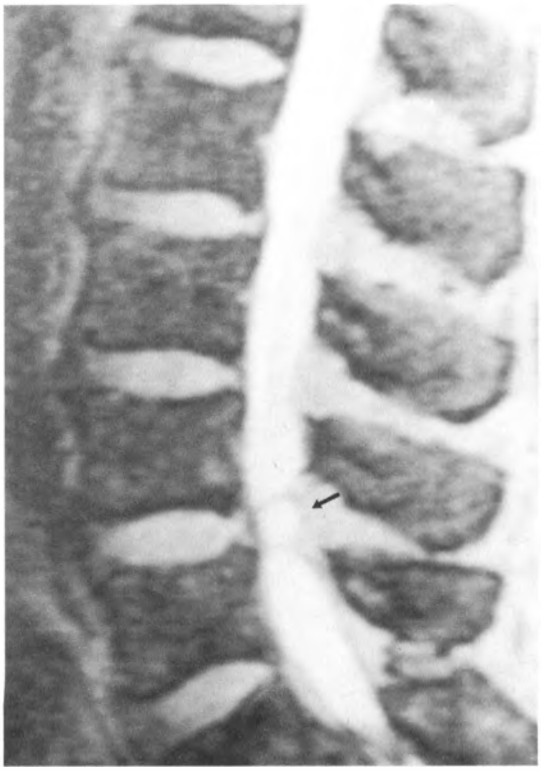


Figure 10.255. Magnetic resonance image sagittal view shows the synovial cyst contacting the thecal sac (*arrow*).

sion fractures. Four cases in which patients were noted to have compression fractures following chiropractic adjustments raised serious questions concerning the relationship between the adjustment and the occurrence of fracture. What is clear is that failure to diagnose a compression fracture, together with the application of adjustment into the area of fracture, can increase symptoms and prolong disability. It is recommended that patients with osteoporosis who have suffered a fall or injury be examined radiographically before treatment is given. In addition, special care should be exercised in elderly patients with osteoporosis (284).

Sacral Insufficiency Fractures

Sacral insufficiency fractures are an often unsuspected cause of low back pain in elderly women with osteopenia who have sustained, unknown, or only minimal trauma. Differential clinical and radiographic diagnosis of these fractures is often difficult. Recognition of the characteristic scintigraphic patterns in sacral fractures, which are frequent in osteopenic patients, could avoid mistaken diagnoses and unnecessary tests or treatment. One of the striking features of these sacral fractures is their invariable location. *The fractures extend vertically in the sacral alae, parallel to the sacroiliac joints.* They are located just lateral to the margins of the lumbar spine. This distribution suggests that such fractures could be partially caused by weightbearing transmitted through the spine (285).

Associated with dull buttock pain and, frequently, other fractures of the pelvic girdle and spine, may be a history of radiation therapy, long-term corticosteroid therapy, or minimal trauma. No neurologic deficit is associated. CT is required for the diagnosis. In uncertain cases, bone nuclear scintigraphy would appear to be the best diagnostic screen. Frequency of

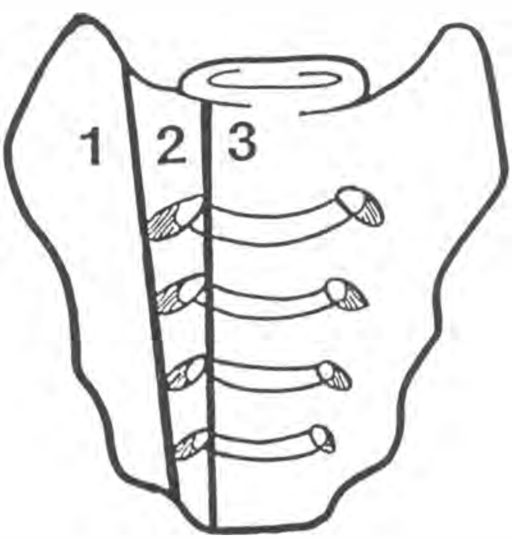


Figure 10.256. Classification of sacral fractures after Denis (18); insufficiency fractures occur in zone 1. (Reprinted with permission from Weber M, Hasler P, Gerber H. Insufficiency fractures of the sacrum: twenty cases and review of the literature. Spine 1993;18(16):2507–2512.)

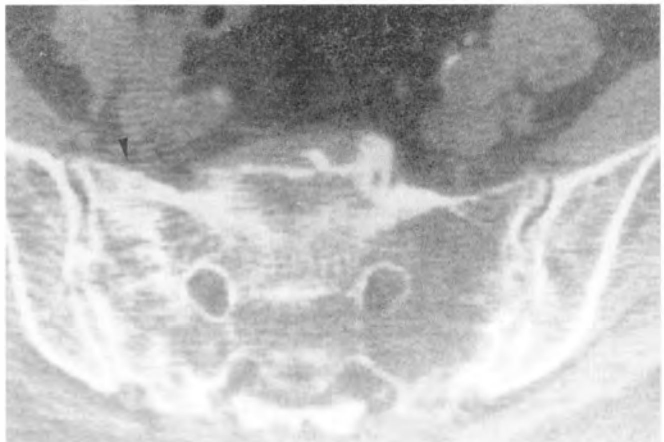


Figure 10.257. Computed tomography scan shows a vertical area of sclerosis (*arrow*) in the right sacral ala at the window settings appropriate for soft tissues. (Reprinted with permission from Weber M, Hasler P, Gerber H. Insufficiency fractures of the sacrum: twenty cases and review of the literature. Spine 1993;18(16):2507–2512.)

Figure 10.258. Computed tomography with displacement of both lateral portions of the upper sacral border (*arrows*). (Reprinted with permission from Weber M, Hasler P, Gerber H. Insufficiency fractures of the sacrum: twenty cases and review of the literature. Spine 1993; 18(16):2507–2512.).

sacral insufficiency fracture was found to be 1.8% in female patients older than age 55 (286). Figure 10.256 is the classification of sacral fractures and Figures 10.257 and 10.258 are examples of sacral fracture.

Ulcerative Colitis Causes Arthritis

Arthritis has long been associated with ulcerative colitis, but not at the 62% rate reported among 79 patients in Naples (287). The highest prevalence in previous studies was approximately 35%.

Among the 49 patients with evidence of arthritis, the diagnoses were ankylosing spondylitis (20 patients); peripheral

arthritis (15 patients), and unclassifiable (because it was observed in patients with colitis) spondyloarthritis (14 patients). None of the patients tested positive for rheumatoid factor.

Scoliosis with Syrinx

An 8-year-old girl was seen complaining of midthoracic spinal pain. She had complained of a flexible round back deformity for several years. She stands with the head and right knee flexed to relieve the pain in the midthoracic spine.

A 7° degree levoscoliosis of the thoracic spine is seen on plain x-ray film and an MRI is ordered. T1-weighted sagittal images of the thoracic spine (Fig. 10.259) revealed a vertically oriented tubular abnormality demonstrated within the central aspect of the spinal cord from the T6 through the T9 levels with an internal signal paralleling that of CSF. Mild associated fusiform expansion of the caliber of the spinal cord is seen at the T8 through T10 level. No abnormal signal intensity is demonstrated in the surrounding parenchyma of the spinal cord. The conus medullaris is normally situated at the T12-L1 level. Vertebral marrow signal is within normal limits. The intervertebral disc signal is normal. No evidence of compression on the spinal cord is seen and the neural foramen are patent.

The diagnosis was syringomyelia. Treatment discussion included syrinx drainage to decompress and maintain a decompressed position so that the nonstructural scoliosis might resolve. The final decision, because of a nonprogressive scoliotic curve or pain, was to watch the syrinx and curve, closely observing the young girl's symptoms. At publication of this book, this syrinx is gradually resolving without any treatment.

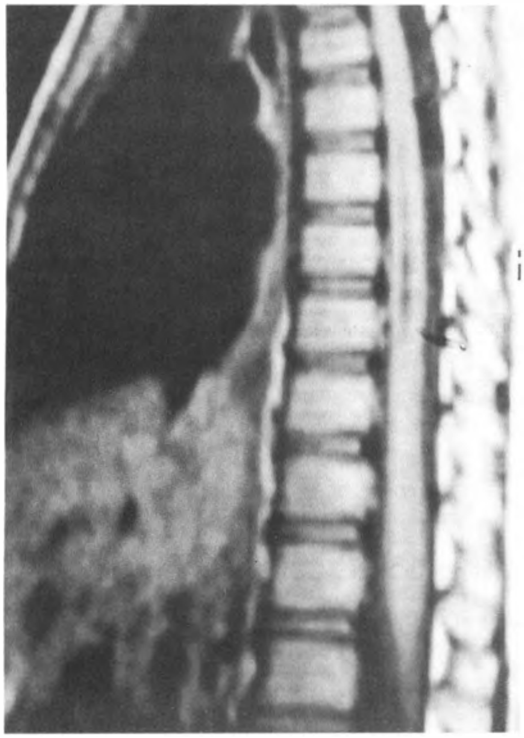


Figure 10.259. Syringohydromyelia.

The clinical pearl here is that scoliosis with neurologic deficit requires MRI to rule out spinal syrinx or tumor. Children with scoliosis and syringomyelia have an equal incidence of left and right-sided curves with a normal sagittal alignment. Most are seen at Risser 0 classification with significant curves, and curve progression occurs in half of the patients. Bracing is not effective in preventing curve progression. Neurologic signs, present in most children, stabilize after syrinx drainage. Neither the child's sex or age, nor type of curve or drainage of the syrinx has been found predictive of curve progression. In syringomyelia, the relationship of the syrinx and the scoliosis is not well understood (288).

An MRI evaluation of the entire spine is needed in all juvenile scoliosis patients or those with left-sided curves and a normal sagittal alignment, especially those with asymmetric abdominal reflexes. Neurosurgical drainage of the syrinx should be undertaken to stabilize the neurologic deficit (288). Evans et al. (289) conclude that MRI of all patients with juvenile scoliosis should be obligatory because in a consecutive group of 31 children with idiopathic juvenile scoliosis 26 were found to have abnormalities of the hind brain or cord.

Bracing of juvenile curves has a questionable role (288). Noonan et al. (290) reported that 92% of 111 immature patients in whom idiopathic scoliosis had been treated with a Milwaukee brace were followed to determine the effectiveness of the brace in preventing progression of the curve. They questioned that the brace did indeed alter the progression of the curve, a finding they admit did not agree with previously reported favorable results.

Postsurgical Scoliosis Strut Graft for Degenerative Lower Lumbar Disc Disease and Stenosis

A 46-year-old woman had fibular strut placement extending to the L4 level for scoliosis correction. Bilateral leg fatigue and pain and low back pain caused her to seek care. Figures 10.260–10.263 reveal the imaging in this case.

Treatment given was distraction manipulation of the two lower lumbar levels with the clinical goal of giving sufficient relief to allow the patient to have a quality of life compatible with her wishes. Six weeks of treatment, given two to three times weekly, resulted in tolerance of low back pain and lower extremity pain to the point of being able to perform those things she needed to do in her life. This was felt to be a good clinical result in a spine with arachnoiditis, osteoarthritis of the two lower lumbar facet levels, extensive disc degeneration at the two lower levels, and L5–S1 disc protrusion, all coupled with the continual stress of having all ranges of motion of the thoracolumbar spine placed at the two lower disc levels where such instability and degenerative changes exists.

Scoliosis with Aging

Scoliosis with progressive deformity can develop late in life. Two hundred patients older than age 50 years with back pain and recent onset of scoliosis were studied. Seventy-one percent of patients were women, and no patient had undergone spinal surgery. The curves involved the area from T12 to L5

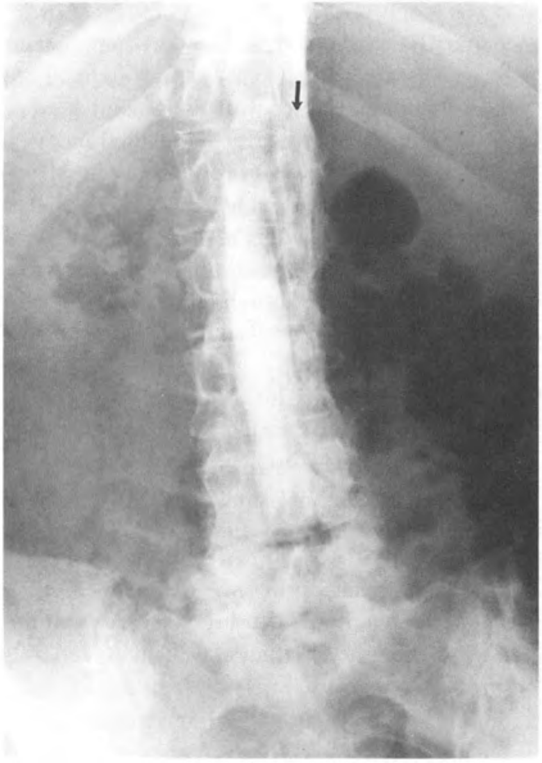


Figure 10.260. A fibular strut (*arrow*) is in place for scoliosis correction fusion extending to the L4 level.

Figure 10.261. Lateral projection of Figure 10.260 shows the extensive L5–S1 degenerative disc disease and less degeneration at the L4–L5 level. All motion occurs at the two lower disc levels because of the fusion to the L4 level by the fibular strut (*arrow*).

Figure 10.262. Axial computed tomography scan shows vacuum change within the nucleus pulposus at L5–S1 (*arrow*) and disc protrusion and bone hypertrophy at the left L5–S1 level, which effaces the thecal sac slightly (*arrowhead*). Also note the facet hypertrophy bilaterally with narrowing of both osseoligamentous canals.

Figure 10.263. Myelographically enhanced computed tomography scan shows the posterior spinal strut fusion. Note the nerve root filaments within the cauda equina are clumped in the midcoronal plane of the thecal sac and lobular indentation is seen dorsally (*arrow*). These findings suggest arachnoiditis scarring.

with the apex at L2 or L3, and they did not exceed 60° Cobb angle. Degenerative facet joint and disc disease were always present, and the curves were associated with a loss of lumbar lordosis. Forty-five patients with severe pain and neurologic deficits were studied using myelography. Indention of the column of contrast medium was seen at several levels. It was most severe at the apex of the curve and least severe at the lumbosacral joint. The curves progressed an average of 3° per year over a 5-year period in 73% of patients. Grade 3 apical rotation, a Cobb angle of 30° or more, lateral vertebral translation of 6 mm or more, and the prominence of L5 in relation to the intercrestal line were important factors in predicting curve progression (291).

Low Back Pain of Pregnancy

Back pain is a common complaint of three of four women during pregnancy. The pain intensity increases over time until delivery. Young women report more intensive pain than older women. The cause of low back pain starting during pregnancy is still not known (292).

To determine the prevalence of back pain and its development over the first postpartum period, 817 women who had been followed through pregnancy were studied a minimum of 12 months after delivery. More than 67% of the women experienced back pain directly after delivery, whereas 37% said they had back pain at the follow-up examination. Factors that correlated to persistent postpartum back pain were the presence of back pain before pregnancy, physically heavy work, and multiple pregnancies. Of these four factors, physically heavy work was found to have the strongest association with persistent back pain at 12 months.

Back pain occurring during pregnancy and delivery does seem to improve in most women during the first 6 months after delivery, and particularly in the first month. In particular, women who do heavy manual work may need help to recover more quickly (293).

Chiropractic Care During Pregnancy

No justification is reported for or against chiropractic care during pregnancy for the reduction of obstetric interventions during labor and delivery. Chiropractic care and craniosacral therapy do not necessitate increased obstetric procedures during labor and delivery and, therefore, should not be a concern in the treatment of pregnancy-related disorders, such as low back pain. Indeed, other evidence suggests that manual manipulation may prevent back labor in those patients with low back pain during pregnancy (294).

Nerve Damage During Delivery

Injuries to the lumbosacral plexus during labor and delivery in two cases localized the site of obstetric paralysis to the lumbosacral trunk (L4--L5) and S1 root where they join and pass over the pelvic rim. Paralysis can be mild or severe. Small maternal size, a large fetus, midforceps rotation, and fetal malposition can place the mother at risk for this nerve injury (295).

The nerve lesion probably results from direct pressure by the descending fetal head compressing the lumbosacral trunk and the S1 root as it joins the trunk against the rim of the pelvis during the rotation and descent of the second state of labor (296). The foot drop is almost always unilateral and, generally, on the same side as the infant's brow during the descent. As many as 1 of 2000 deliveries can be complicated by this palsy. It is important to distinguish this obstetric paralytic syndrome from compression of the peroneal nerve where it crosses the fibular head, which also causes numbness along the lateral calf and a foot drop, and it can be seen during labor as a result of compression by legholders.

The increased propensity for disc herniations during pregnancy stresses the need to consider this cause of foot drop in the differential diagnosis. Another possible cause of obstetric paralysis is damage to the lumbosacral roots from an epidural anesthetic catheter (295).

Between 50 and 90% of women develop symptoms of low back pain at some point during pregnancy. In 10 to 36% of these women, the symptoms are of such severity that they have a dramatic impact on the activities of daily living and frequently require prolonged bed rest (297).

MRI in Pregnancy Evaluation

A herniated disc during pregnancy occurs with a reported incidence of 1 in 10,000 cases. MRI, without ionizing radiation, is the imaging modality used to study the lumbar spine. Traditionally, caesarean section has been the preferred route of delivery with the anticipation that during labor increasing epidural venous pressures could precipitate progressive neurologic dysfunction.

However, during uterine contractions, increases in the CSF pressure have been reported to be directly proportional to the intensity of the perceived pain that subsequently influences the amount of concomitant skeletal muscle activity. The elevations in both CSF and epidural pressure are therefore not directly related to contraction of the uterine musculature itself but rather are a product of the reflex responses of skeletal muscles to pain (298).

Iliocostal Pain

Normally, the distance between the lower ribs and iliac crest is sufficient that no contact occurs. Iliocostal contact can be caused by severe osteoporosis; severe dorsal kyphosis because of dorsal, wedge-shaped compression fracture; lumbar compression fractures, multiple disc narrowings, or lumbar vertebral collapse from infection or metastases; lumbar or lumbodorsal scoliosis; and a combination of any of these factors.

The major symptom of iliocostal fracture is low back pain. Pain can also radiate to the groin, buttock, thigh, chest, and lower rib cage.

Treatment

Treatment can involve the following. (*a*) Surgical removal of the 12th and sometimes also the 11th rib has given permanent relief of pain. (*b*) Lower rib compression, which is done by using a strong elastic belt that compresses the lower ribs and re-

moves them from contact with the iliac crest. (*c*) Sclerosing injections—small amounts of hypertonic dextrose (12.5 to 25% with lidocaine) at the osseotendinous junction to relieve tenderness in this area (299).

Coccygodynia

Common coccygeal pain could come from the coccygeal disc in approximately 70% of cases. Idiopathic coccygodynia is poorly understood (300).

Breast Reduction Can Relieve Back and Neck Pain

Breast reduction surgery can relieve back and neck pain in large-breasted women. Reduction surgery significantly improves the pain and discomfort complex in this group of patients (301).

Camptocormia

Progressive lumbar kyphosis or camptocormia, a rare disease of the elderly, is characterized by an inability to immobilize the lumbar spine in relation to the pelvis. It appears to be a result of weakness of the paraspinal muscles.

Patients with camptocormia show spinal muscles with areas of low density on CT scans and MRI, similar to the features described in primary muscular dystrophies (302).

Camptocormia, disappearing in the recumbent position, is thus probably linked to muscle involvement. That often a family history of such disorder indicates that this is a genetically transmitted condition (303).

Transient Osteoporosis of the Hip

Transient regional osteoporosis of the hip (TROH) is a self-limiting and usually idiopathic condition that typically resolves symptomatically and radiographically over a period of 2 to 6 months from presentation. Occasional cases complicating pregnancy have been reported.

Although radiographs, radionuclide bone scan, and MRI are useful in making the diagnosis of transient regional osteoporosis of the hip, bone densitometry is ideally suited to monitor its rate of resolution. Symptoms alone are not a sufficiently accurate indicator. Bone densitometry may be useful in diagnosis and monitoring TROH (304).

Classically, TROH is characterized by disabling pain in the hip without antecedent trauma and by striking radiographic evidence of osteopenia that is isolated to the hip (305). Transient osteoporosis affects middle-aged men, and it affects women almost exclusively during the third trimester of pregnancy.

The presenting symptoms of transient osteoporosis is a dull ache in the inguinal area, buttocks, or anterior aspect of the thigh that is usually acute in onset but without antecedent trauma. It is frequently accompanied by a limp and an antalgic gait. The pain is exacerbated by weightbearing and relieved by rest.

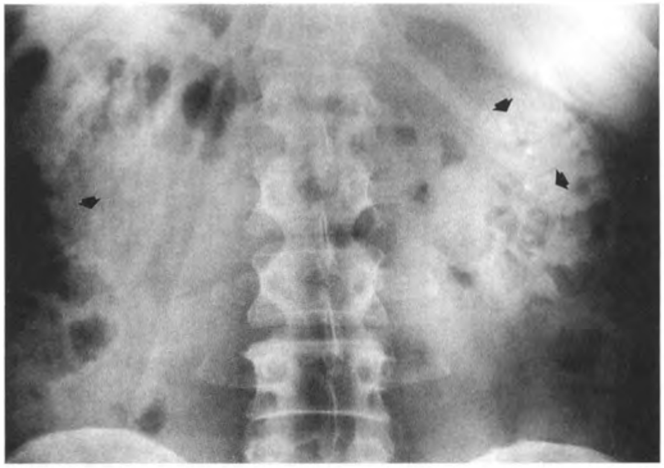


Figure 10.264. Nephrocalcinosis. See *arrows* for calculi.

Three distinct temporal phases of transient osteoporosis have been described. The initial phase, characterized by a rapid aggravation of the pain and functional disability, usually lasts for approximately 1 month. The next phase, in which the symptoms reach a plateau in intensity, typically lasts for 1 to 2 months. During this time, osteopenia is noted on radiographs. A final phase is characterized by regression of the symptoms and reconstitution of the radiographically visible bone density; this period is usually as long as 4 months (305).

Nephrocalcinosis

Figure 10.264 shows nephrocalcinosis of the kidneys, which caused low back pain in a patient with hyperparathyroidism—a case needing other than a spinal adjustment.

Testicular Torsion Causes Low Back Pain

Testicular torsion was found to be the cause of pain in a 7-year-old child with a brief history of low back pain radiating to the groin bilaterally. Testicular torsion does occur with some frequency in the pediatric population. Acute low back pain without history of trauma or injury is or should be continually suspect in the pediatric patient (306).

Aneurysmal Bone Cyst

Figures 10.265 and 10.266 are radiographs of a 20-year-old woman complaining of low back pain showing a 4-cm expansile bone lesion of the L3 transverse process and lamina-pedicle, which had smooth margins with no evidence of periosteal spiculation. Radiolucent areas were noted throughout the substance of the lesion. It does have a blown-out appearance and suggests invasion of the osseoligamentous canal. This lesion was not present on lumbar radiographs taken for low back pain 2 years previously.

Levoscoliosis of the lumbar spine perhaps occurs because of painful muscle splinting.

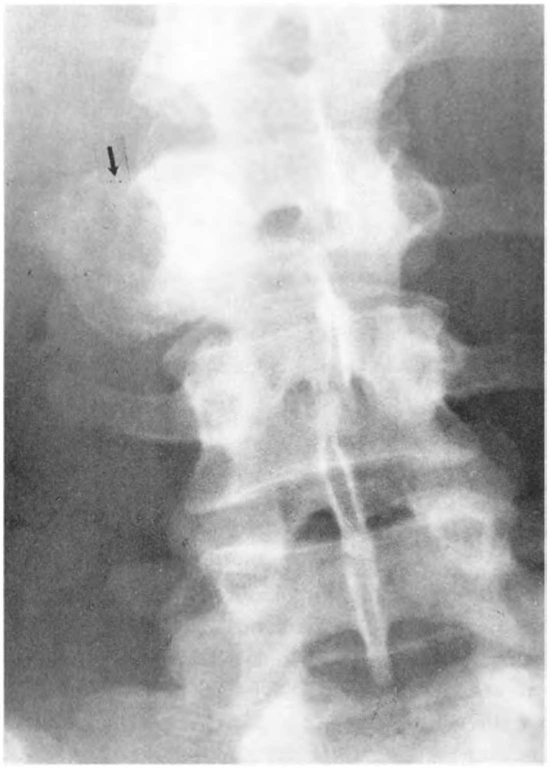


Figure 10.265. Aneurysmal bone cyst (*arrow*) on anteroposterior radiograph.

Differential diagnosis included osteoblastoma but this lesion is more radiopaque than the typical osteoblastoma which is more radiolucent in appearance.

Slipped Femoral Capital Epiphysis (SFCE, Epiphyseal Coxa Vara)

A 16-year-old white male had 6 months of left hip pain and limp. Figures 10.267 and 10.268 are the anteroposterior and lateral views of the left hip joint showing the medial and downward epiphyseal displacement on the femoral neck.

This patient was referred for surgical consultation. Regardless of treatment, degenerative arthritis is common with this condition.

Compression Fracture of the Second Lumbar Vertebral Body

A 51-year-old white woman fell from a horse and felt lumbar spine pain. MRI was performed because of a question of acute versus longstanding compression fracture at L2. The T1 sagittal image (Figure 10.269) revealed loss of signal intensity of the L2 mid and upper vertebral body (*arrow*) and hyperintensity on T2-weighted image (Fig. 10.270) (*arrow*). These findings would be indicative of acute inflammatory change suggesting fresh fracture. Also noted is posterior displacement of the second vertebral body on L3 with protrusion of the L2–L3 disc.

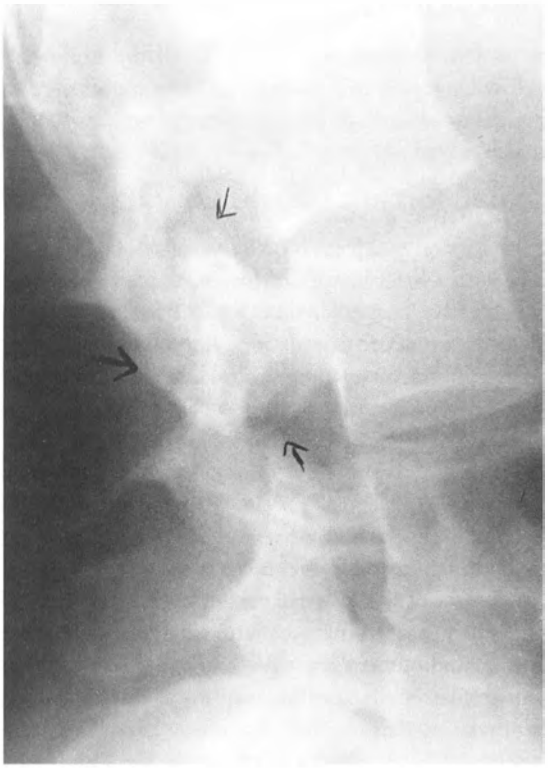


Figure 10.266. Lateral view of Figure 10.265 showing an aneurysmal bone cyst (*arrows*). (Case courtesy of Drs. Jon, Steven, and Michael Alter.)

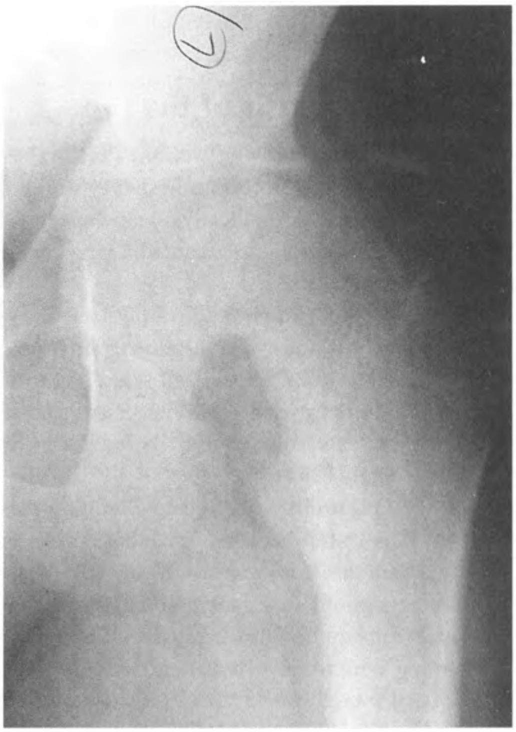


Figure 10.267. Slipped capital femoral epiphysis shown in this anteroposterior hip radiograph.

Figure 10.268. Lateral frog-leg view of Figure 10.267. (Case courtesy of Drs. Jon, Steven, and Michael Alter.)

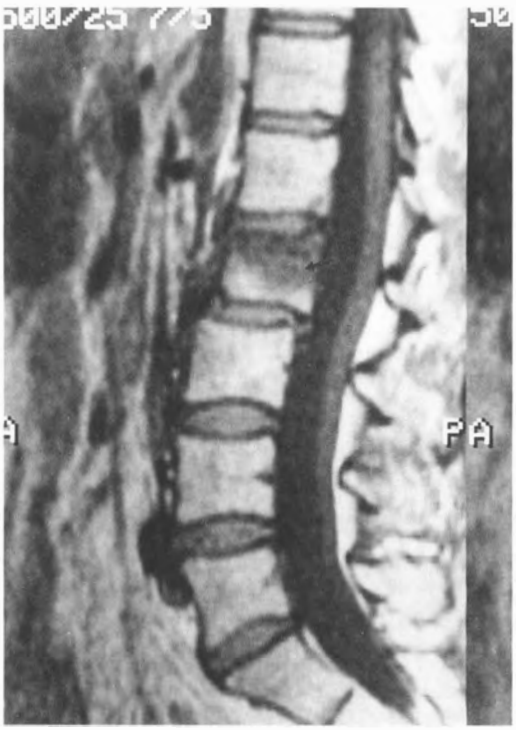


Figure 10.269. T1-weighted sagittal magnetic resonance image shows hypointensity of the L2 vertebral body (*arrow*).

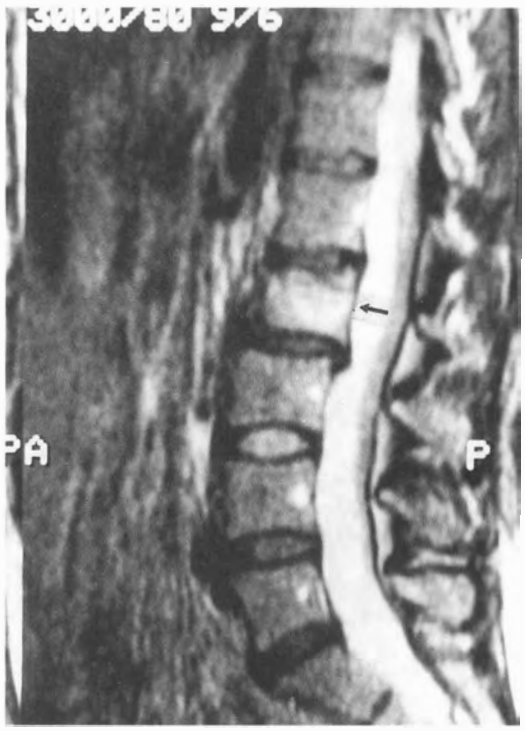


Figure 10.270. T2-weighted sagittal magnetic resonance image shows hyperintensity of the L2 vertebral body (*arrow*).

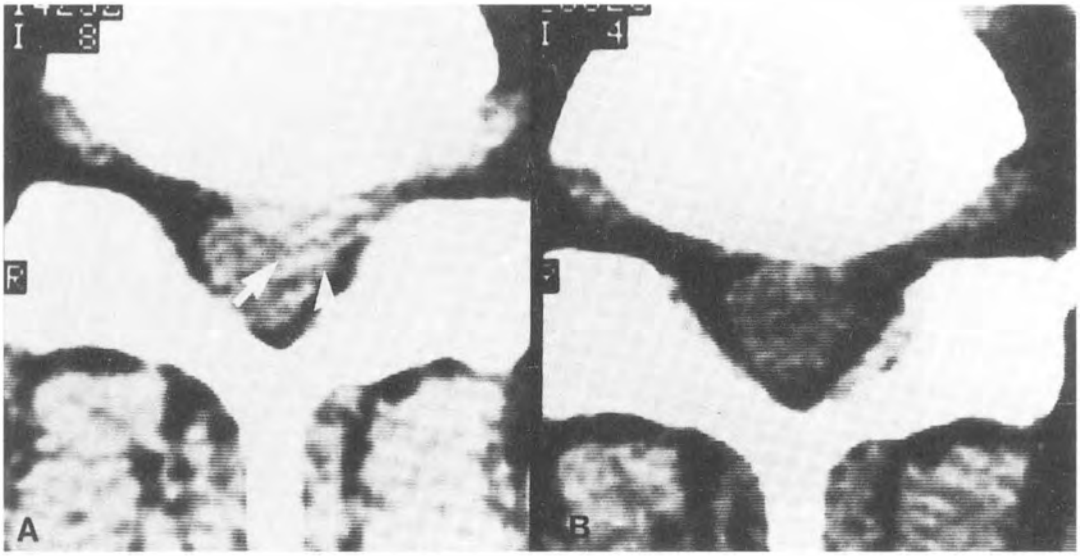


Figure 10.271. Computed tomography (CT) scan shows complete disappearance of herniation at L5–S1. **A.** CT scan at L5–S1 reveals a large central and left herniation (*white arrows*) in a 28-year-old man with severe left radiculopathy. The left L5 root appears slightly enlarged and denser than the right root. **B.** The pain and radiculopathy gradually disappeared; CT scan made 16 months later showed no evidence of the previous herniation. The root now appears normal. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:99, 146.)

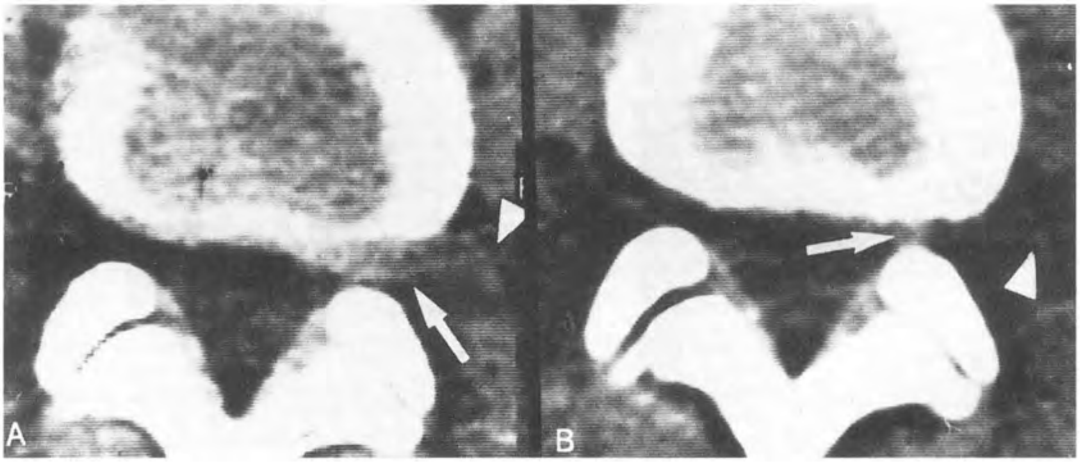


Figure 10.272. Computed tomography (CT) scan shows regression of foraminal herniation at L4–L5. **A.** CT scan of L4–L5 shows a left foraminal herniation (*arrow*) contiguous to a slightly swollen L4 root (*arrowhead*). The left radiculopathy improved greatly with conservative treatment. **B.** A corresponding slice of a CT scan made 2 years ago shows residual herniation (*arrow*); the left L4 root now appears completely normal. (Reprinted with permission from Teplick GJ. Lumbar Spine CT and MRI. Philadelphia: Lippincott-Raven, 1992:94, 146.)

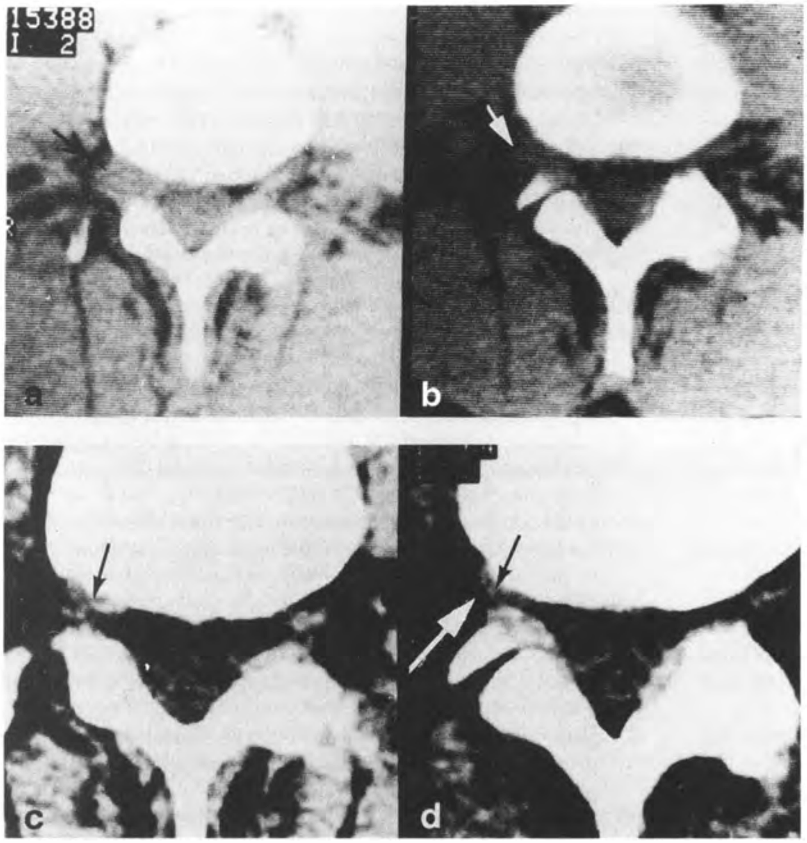


Figure 10.273. Computed tomography (CT) scan shows foraminal herniation with swollen root resolution in 4 months. **A** and **B.** A soft tissue mass in the right foramen (*arrows*) at L3–L4 is a conglomerate of a right foraminal herniation and a swollen nerve root. The myelogram was entirely negative. The patient's symptoms were consistent with a right L3 radiculopathy. Marked clinical improvement occurred with conservative therapy. **C** and **D.** CT scan 4 months later, when the patient was virtually symptom free, shows a tiny right foraminal herniation (*black arrows*) with a normal appearing right L3 root (*white arrow*).

Spontaneous Regression of Lumbar Herniated Discs

This chapter on diagnosis concludes with a discussion of disc herniation diagnosis and the challenge that disc herniation may or may not be symptom producing. Teplick (307) defines spontaneous regression as a diminution or total disappearance of a herniation that has not had surgical discectomy, chemonucleolysis, or percutaneous discectomy. The fact that disc herniation can reduce without surgical intervention, and allow a patient to become asymptomatic, is shown in Chapter 3, *Neurophysiology and Pathology of the Nerve Root and Dorsal Root Ganglion,* and is presented in three cases for your awareness and contemplation.

Figures 10.271–10.273 are three excellent examples of partial or total regression of disc herniations with conservative care. My opinion is that herniated disc regression, regardless of size, is well documented in MRI or CT literature, and even by myelography. So well known is it today, that I do not believe it even warrants great discussion anymore. We know that discs diminish in size by 100 to 0% with total regression of symptomatology. At this time, visualizing these cases should be accompanied by the thought that the significance of the reduction is little known, and even of less importance.

As I conclude this chapter it is exciting to end it with a discussion of the insignificance of the size of disc herniation in the production of pain, whereas a mere 10 years ago, when writing the fifth edition, the question of observing the reduction of disc herniation after nonsurgical care was screaming to be addressed. We are always presented with new questions!

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Laboratory Evaluation

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chapter 11

The average person puts only 25% of his energy and ability into his work. The world takes off its hat to those who put in more than 50% of their capacity and stand on its head for those few and far between souls who devote 100%.

—Andrew Carnegie

A thorough diagnostic evaluation lays the foundation for a logical treatment plan. However, the phrase "laboratory diagnosis" is a misnomer. In actuality, the evaluation of blood, urine, and other specimens is but one of the five major means of evaluating patients with low back pain, the others being the history, physical examination, routine radiographs, and special studies (electromyography [EMG], computed tomography [CT], magnetic resonance imaging [MRI], and so on). Laboratory tests, in and of themselves, should never be considered as the primary or only investigatory means, but rather as tools to assist the physician in analyzing and correlating other clinical findings.

Although many different causes are found for low back pain, the clinical laboratory is most useful in evaluating infectious, inflammatory, metabolic, and neoplastic disorders. Most simple traumatic, mechanical, and degenerative conditions are not associated with significant laboratory abnormalities. Indeed, those conditions seen most frequently in the office (e.g., strain or sprain syndromes, disc disorders, degenerative joint disease, and myofascial pain syndromes) are characterized by normal laboratory test results.

Because the prevalence of these common conditions is so much greater than that of other disorders, few laboratory tests are sufficiently cost-effective to be used as routine procedures. As the prevalence of a condition diminishes, the possibility of encountering a false-positive test result becomes greater, and may even exceed the incidence of a true-positive test. Because of the differences in sensitivity, specificity, and predictive value of laboratory tests, it is reasonable to use laboratory tests in pursuing a statistically reasonable diagnosis rather than haphazard screening. In other words, the selection of laboratory tests should be guided by the working diagnosis generated by the history and physical examination, rather than simply performed as indiscriminate screening. As will be seen, the "rheumatic" or "arthritic" profile, which commonly consists of tests for the rheumatoid factor, antinuclear antibodies (ANA), uric acid, and antistreptococcal antibodies (e.g., antistreptolysin-O), is almost never indicated in the patient with isolated low back pain because the conditions that are associated with abnormalities of those tests almost never produce symptoms in the low back without considerable concomitant peripheral involvement.

If the initial history and physical examination raise the possibility of a nonmechanical, nondegenerative disorder resulting in low back discomfort, then appropriate follow-up procedures are selected. The most common laboratory tests used to evaluate patients with low back pain are discussed in the following section.

Tests can be broadly considered as either "nonspecific" or "specific." In the former category, which includes such tests as the erythrocyte sedimentation rate and the C-reactive protein assay, the tests frequently yield abnormal results in many different disorders without identifying any one particular disease. In contrast, "specific" tests are aimed at detecting a specific condition or pathophysiologic state. Unfortunately, such tests seldom meet the ideal goal of being 100% specific (i.e., abnormal only in patients with the disease in question), but they do help narrow down the possibilities when used appropriately. A better classification term than "specific" is "focused," implying that a test is being used to evaluate for a narrow range of possible disease states.

NONSPECIFIC LABORATORY INDICATORS OF DISEASE

Erythrocyte Sedimentation Rate

The erythrocyte sedimentation rate (ESR) is a widely used nonspecific test. The basis of the test is that red blood cells settle with gravity in a vertical tube of blood at a rate dependent on such variables as the number of cells, the size and shape of the cells, and the type and amount of plasma proteins. Abnormalities result in an elevation (increase) in the rate of sedimentation. Anemias may result in an increased ESR, as do many diseases resulting in an antibody response. With low back pain patients, the ESR is of most use in suspected cases of vertebral osteomyelitis, lumbar disc infections, and systemic inflammatory conditions. The ESR is elevated in most cases of vertebral osteomyelitis, with sensitivity ranging from 88 to 98% (1–3). Tuberculosis of the spine does not produce as dramatic a change in the ESR as do suppurative forms of osteomyelitis, with the ESR being significantly elevated in only 70% of cases and seldom elevated more than 50 mm/h.

Infection of the intervertebral disc following lumbar discectomy can be a difficult diagnosis to make. In the typical scenario, the patient has undergone a lumbar discectomy and is seen in the office 1 or more weeks after discharge complaining of progressively increasing discomfort in the lumbar spine. The ESR can be used to determine if the symptoms are probably the result of a postoperative discitis. Elevation of the sedimentation rate above 50 mm/h at 2 or more weeks postoperatively appears to be a reliable indication of a secondary discitis, and this precedes diagnostic radiographic changes (4, 5). As will be discussed in the next section, C-reactive protein is an earlier and more sensitive marker of osteomyelitis and postoperative disc infections.

Malignancies, including plasma cell dyscrasias, primary bone tumors, and metastatic disease to the lumbar spine, can also cause elevations of the ESR; however, the sensitivity is not sufficiently great to comfortably rule out a tumor on the basis of a normal result or to support the use of ESR as a screening procedure for cancer.

The ESR has been shown to be of considerable value in the diagnosis of polymyalgia rheumatica and temporal arteritis, with most cases having rates in excess of 40 mm/h.

Table 11.1 summarizes the results of the ESR in orthopaedic conditions affecting the low back and pelvis.

Table 11.1

Erythrocyte Sedimentation Rate (ESR) in Low Back and Pelvic Orthopedic Disorders

ESR Usually Normal	ESR Often Elevated[a]
Degenerative joint disease	Postsurgery
Sacroiliac syndromes	Suppurative osteomyelitis
Spondylolisthesis	Tuberculous osteomyelitis
Fibromyalgia	Intervertebral discitis
Intervertebral disc syndromes	Multiple myeloma
Osteoporosis	Ankylosing spondylitis
Facet syndromes	Reiter's syndrome
Common compression fractures	Metastatic disease
	Psoriatic arthritis
	Polymyalgia rheumatica
	Polymyositis
	Osteosarcoma

[a]Frequency of elevation varies considerably in these disorders.

C-Reactive Protein

C-reactive protein (CRP) is a protein synthesized in the liver in response to tissue damage. It is considered, along with haptoglobin, fibrinogen, ceruloplasmin, complement, and several other proteins, as an "acute phase reactant" because its levels rise rapidly in response to inflammatory states and tissue destruction. Measurement of CRP by sensitive quantitative methodologies (e.g., nephelometry and immunoassay) has made slide agglutination techniques obsolete and has increased the usefulness of the test. Because the ESR is affected by changes in acute phase proteins, especially fibrinogen, it is understandable that many of the conditions that cause elevated sedimentation rates also cause increased serum levels of CRP. CRP is of particular use, being more sensitive than the ESR, in monitoring disease activity in patients with low back pain caused by ankylosing spondylitis and Reiter's disease (6). In general, CRP tends to become abnormal sooner than does the ESR, and it falls to normal values sooner during the recovery period.

Urinalysis

Urinalysis is a low-cost procedure that is an important part of the evaluation of patients with low back pain and lower extremity radicular pain. It should be performed whenever no obvious direct cause is seen for the patient's discomfort. A complete discussion of urinalysis is beyond the scope of this chapter; instead, the focus will be on those components directly relating to low back pain. These consist of the chemical evaluation for protein, blood, and glucose, and the determination of the presence of bacteria and white blood cells. In most cases, a simple dipstick assessment will suffice.

Routine determination of protein in urine actually evaluates only for the presence of albumin. Dipsticks are not sensitive to globulins or to immunoglobulin free light chains (Bence Jones protein). Albuminuria in trace amounts is often seen in normal persons; however, greater amounts should be evaluated by means of 24-hour urine protein quantification. Significant albuminuria usually indicates a disorder of the renal glomerulus or tubules. This might be caused by an organic disorder (e.g., glomerulonephritis or secondary damage to the nephrons in multiple myeloma) or occur as a physiologic variant. Relating

to the latter, heavy exercise can induce transient proteinuria, and some persons spill protein into the urine in the erect posture (orthostatic proteinuria).

Hematuria should always be taken seriously. Blood can get into the urine from any part of the urinary tract, so the range of conditions producing hematuria is quite extensive. Hematuria may be the only finding early in the course of renal cell carcinoma, a condition to be considered in patients over the age of 20. Other conditions associated with hematuria and which can produce back pain include renal and ureteral stones, pyelonephritis, glomerulonephritis, cystitis, and prostatic diseases.

Glucosuria, even in trace amounts, should be evaluated further by means of a fasting plasma glucose level. Glucosuria is most often seen in diabetes mellitus, and these patients will have either a fasting plasma glucose level in excess of 140 mg/dL or an abnormal glucose tolerance test. Patients with glucosuria in the absence of abnormal glucose tolerance testing have renal glucosuria, a benign condition. Diabetic neuropathy can produce an anterior femoral neuralgia, and urinalysis should always be considered in patients presenting with that pain pattern. Because not all diabetic patients have glucosuria, if diabetes is strongly suggested serum glucose testing, including functional studies, should be considered.

Infections of the kidney, prostate, and bladder can refer pain to the low back or pelvis, and they usually are associated with bacteriuria and pyuria. Current dipstick technology allows for screening for bacteria through the detection of nitrites that were converted by bacteria from normal urinary nitrates. Leukocyte esterase determination is useful in the chemical (dipstick) detection of white blood cells. If both the nitrite and leukocyte esterase tests are negative, then urinary tract infection as a cause of low back pain can initially be ruled out. If either is positive, then microscopic evaluation and possibly culture should follow.

Alkaline Phosphatase

Alkaline phosphatase actually represents several isoenzymes sharing similar activity, but with slight differences in physical structure. Isoenzymes are produced in a variety of tissues, the most clinically significant of which are bone, liver, placenta, and small intestine. Elevations of the serum enzyme level result from increased metabolic activity or cellular damage. High alkaline phosphatase levels in patients with low back pain are most likely caused by physiologic variation, response of osteoblasts to osseous injury or malignancy, metabolic bone disease, Paget's disease of bone, unrelated hepatobiliary disease, or medication-induced cholestasis. Physiologic variations from normal adult values occur in pregnancy (placental origin), childhood (osseous origin), the postprandial state (intestinal origin), and in some healthy elderly patients (7). Age-adjusted reference values must be used when evaluating the alkaline phosphatase levels of a pediatric patient. Alkaline phosphatase levels are typically increased in the healing stage of fractures because of the increased activity of osteoblasts. In all age groups, fracture of long bones are more likely than vertebral or small bone fractures to be associated with increased alkaline phosphatase activity. An elevated alkaline phosphatase level in an older patient with an apparent osteoporotic compression fracture should prompt the physician to consider other possible causes of the enzyme elevation. Alkaline phosphatase levels gradually rise in pregnancy, peaking at 32 to 34 weeks of gestation and remaining constant until a few days after delivery (8). As with all tests, the possibility exists of pharmacologic and physiologic causes of abnormal results.

Metabolic and malignant diseases of bone that are unaccompanied by a significant osteoblast response have normal serum alkaline phosphatase values. For this reason, a purely lytic bone disease can have normal serum alkaline phosphatase levels. Although most patients with Paget's disease (osteitis deformans) have elevated alkaline phosphatase levels, serum levels of the enzyme are occasionally normal in patients in phases of that disease characterized by minimal osteoblast activity. In addition to those elevations seen with pregnancy and healing fractures, serum alkaline phosphatase can rise with drugs that can induce cholestasis, in some adults after a fatty meal, and in the elderly patient.

Because of the multiorgan origin of the enzyme, it is understandable that many different diseases can result in elevation of the serum level. Table 11.2 lists the more common disorders associated with elevated alkaline phosphatase levels.

Further evaluation of an elevated alkaline phosphatase can be done in two ways. As shown in Figure 11.1, determination of the tissue of origin of alkaline phosphatase can be done by searching for elevations in other serum enzymes that parallel those of alkaline phosphatase in certain diseases, or by separation and quantification of the various isoenzymes. G-glutamyl transferase (GTP, G-GTP, G-glutamyl transpeptidase [GGT]) is elevated in many hepatic disorders but is not affected by osseous diseases. GGT is sensitive to alcohol intake and elevations in the low back pain patient may represent a response to heavy alcohol consumption (9). Many routine chemical profiles include both alkaline phosphatase and GTP. Serum 5′-nucleotidase and leucine aminopeptidase can also be measured, and changes tend to parallel those in GTP, although neither is as sensitive. Measurement of alkaline phosphatase isoenzyme can be performed; however, the accuracy of the analysis varies with the method used and the experience of the laboratory.

Of particular concern to the practitioner is the patient with a history of cancer who presents with low back pain. Osseous primary and secondary osteoblastic malignancies are often associated with elevations of serum alkaline phosphatase, and the finding of such in a patient with a history of cancer should prompt further evaluation, such as radionuclide bone scanning. In breast cancer patients, serial measurement of alkaline phosphatase isoenzymes and GTP has been shown to be useful in detecting the occurrence of liver and bone metastases, with abnormal levels found in slightly more than 40% of all patients with these metastases, and in 75% of those patients who are symptomatic because of the metastases (10). In general, biochemical tests and tumor markers have a lower sensitivity to metastatic bone disease than imaging procedures such as bone scanning.

Table 11.2

Pathologies Associated with Elevated Serum Alkaline Phosphatase Levels

Musculoskeletal	
	Primary and metastatic osteoblastic tumors
	Paget's disease (osteitis deformans)
	Fractures
	Rickets
	Osteomalacia
	Hyperparathyroidism
	Rheumatoid arthritis[a]
	Gaucher's disease
Hepatobiliary	
	Drug-induced cholestasis
	Primary and metastatic liver tumors
	Liver abscess
	Hepatic cysts
	Biliary cirrhosis
	Cholangitis
	Choledocholithiasis
	Carcinoma of head of pancreas
	Carcinoma of ampulla of Vater
	Acute hepatitis (mild elevation)
	Infectious mononucleosis (mild elevation)
	Hepatic cirrhosis (mild elevation)
Gastrointestinal	
	Extensive gastric or bowel ulceration
	Intestinal infarction
Miscellaneous	
	Hyperthyroidism
	Renal infarction
	Severe diabetes mellitus

[a]Elevation in rheumatoid arthritis is primarily due to hepatobiliary involvement.

Uric Acid

Serum uric acid is a common part of the laboratory rheumatic profile, but it has little use in the evaluation of the patient with low back pain. Gout is the primary rheumatic disease associated with hyperuricemia, and it is characterized by an acute inflammatory response triggered by uric acid crystal precipitation in synovial fluid. Gout preferentially affects distal joints, most notably those of the foot, ankle, knee, and wrist. Seldom are joints of the spine affected, most likely because the higher temperature in those joints helps keep the uric crystals in solution. It would be extremely unusual for gout to affect the lumbar spine or sacroiliac joints without previously involving the peripheral joints. A more likely situation would be the incidental finding of hyperuricemia in a patient being evaluated for other reasons. Elevation of the serum uric acid level can result from several mechanisms, including decreased renal excretion, increased formation, and metabolic changes (11). Table 11.3 lists the more common causes of hyperuricemia.

Calcium

The blood calcium level is normally closely regulated by the complex interactions of parathyroid hormone, vitamin D, bone, plasma proteins, and calcitonin. Disturbances of those factors can result in alterations in the calcium balance, as reflected by increased or decreased serum levels. Calcium is transported in the blood by binding to albumin and some globulins. As calcium is needed for metabolic functions, it is freed from the plasma proteins and becomes physiologically active in this ionized form. The routine serum calcium assay is actually a measurement of the combined amount of calcium bound onto plasma proteins and ionized, or "free," calcium. A wide variety of disease can result in abnormal serum calcium levels. Table 11.4 lists the most common causes of hypocalcemia and hypercalcemia. It should be noted that the serum calcium level is typically normal in osteoporosis and in degenerative joint disease. Primary hyperparathyroidism and metastatic carcinoma account for most of the cases of hypercalcemia. An elevated serum calcium level should be followed by measurement of the serum parathyroid hormone (PTH). An elevated PTH level in a hypercalcemic patient is indicative of primary hyperparathyroidism, whereas suppressed PTH levels suggest lytic bone disease as the cause of the hypercalcemia.

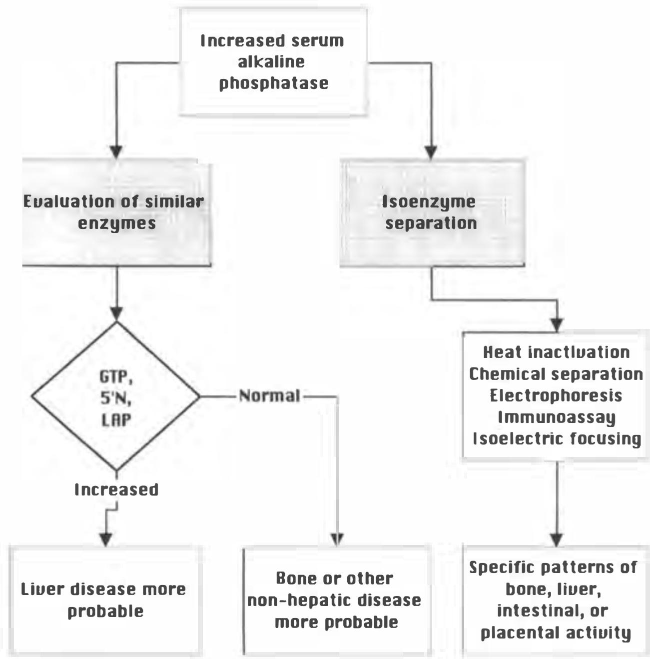

Figure 11.1. Methods to determine origin of increased serum alkaline phosphatase. Differentiation can be made by measurement of other enzymes with similar activity or by various methods of isoenzyme determination. *GTP*, gamma glutamyl transpeptidase; *LAP*, leucine aminopeptidase; *5'N*, 5'-nucleotidase.

Table 11.3

Common Causes of Hyperuricemia

Increased production of uric acid	
High-purine diet	
Increased turnover of nucleic acids	
	Psoriasis
	Multiple myeloma
	Pernicious anemia
	Polycythemia vera
	Leukemia
Primary gout (some cases)	
Decreased excretion of uric acid	
Renal failure	
Alcohol	
Aspirin	
Primary gout (most cases)	
Diuretics	
Miscellaneous (multifactorial) causes	
Obesity	
Primary hypertension	
Hypertriglyceridemia	
Idiopathic hyperuricemia	

Table 11.4

Causes of Serum Calcium Abnormalities

Hypercalcemia	
Increased release of calcium from bone	
	Metastatic carcinoma to bone
	Primary hyperparathyroidism
	Multiple myeloma
	Sarcoidosis
	Tumorous release of PTH-like substance
	Hyperthyroidism
	Prolonged immobilization
Decreased urinary excretion of calcium	
	Renal failure (secondary hyperparathyroidism)
	Thiazide diuretics
Increased gastrointestinal absorption	
	Excess vitamin D intake
	Sarcoidosis
	Hyperparathyroidism
Hypocalcemia	
Nutritional disorders	
	Osteomalacia
	Rickets
	Malabsorption
Hypoalbuminemia	
Hypoparathyroidism	
Pseudohypoparathyroidism	

Phosphorus

Serum phosphorus (phosphate) levels are affected by many of the same conditions that alter serum calcium levels. In hyperparathyroidism, serum phosphorus levels are usually decreased, an inverse relationship to calcium. Vitamin D-resistant rickets may also have a low serum phosphorus level. Hyperphosphatemia can result from chronic renal failure, vitamin D excess, hypoparathyroidism, and some healing fractures. Children tend to have higher phosphorus levels than do adults.

FOCUSED LABORATORY TESTS

Acid phosphatase

Measurement of serum acid phosphatase, an enzyme produced predominantly by prostatic epithelial cells, but also by platelets, red blood cells, bone, and other tissues, has only limited diagnostic usefulness. Elevation of the serum acid phosphatase level is found in many cases of advanced prostatic cancer with either local extension of the tumor or metastasis. Although it was hoped that techniques such as radioimmunoassays and monoclonal antibody-based immunoassay would improve the detection of prostatic cancer while the disease was still confined to the prostate, studies have yielded varying results, and serum prostatic acid phosphatase testing cannot be considered a reliable screening procedure for prostatic cancer (12, 13).

Prostate-specific Antigen

Prostate-specific antigen (PSA), a glycoprotein produced solely by prostatic epithelial cells, has emerged as the biochemical test of choice in detecting and staging prostate cancer (14). Measurement of the ratio of free to total PSA further increases the sensitivity of the test by detecting a significant number of tumors with total serum PSA values below the normal cutoff used to recommend biopsies in patients with normal digital rectal examinations (4.0 ng/mL) (15). PSA testing is best done in conjunction with the digital rectal examination, and it significantly increases the detection rate of prostate tumors compared to the physical examination alone (16). As with prostatic acid phosphatase, serum levels of PSA can elevate in benign conditions and following diagnostic procedures. Benign prostatic hyperplasia and acute and chronic prostatitis can result in increased PSA levels (17). Digital rectal examination does not consistently elevate PSA levels; however, it is advisable to wait at least 24 hours following examination prior to collecting blood samples (18). Similarly, sexual activity can elevate PSA serum levels for approximately a day (19).

Immunologic Studies

Rheumatoid Factors

Rheumatoid factors (RF) are a family of immunoglobulins reactive with autologous immunoglobulin G (IgG). Although most of these anti-IgG autoantibodies are of the immunoglobulin M

(IgM) class, RF belonging to most of the other classes have also been discovered. Traditional tests for RF search for IgM RF; they are based on agglutination of either sensitized sheep erythrocytes or antibody-coated latex particles. The sheep erythrocyte procedure appears to be a more specific test for rheumatoid arthritis than the latex method, but it is less sensitive. It has been shown that the combination of positive results for RF by both methods is highly specific for rheumatoid arthritis (20). Many laboratories now measure rheumatoid factor directly using enzyme-linked immunosorbent assays (ELISA) and report the results as units rather than titers. These methods may be more sensitive than the standard latex fixation method, although universal agreement is not found on this (21, 22).

Because the RF in a patient can be of one or more antibody types, because it is polyclonal in origin, and because considerable laboratory variation is seen in testing methods, it is not surprising that standard RF tests often fail to detect the presence of the antibody in patients with rheumatoid arthritis. Rheumatoid arthritis patients who have negative RF tests are said to be "seronegative." Some seronegative patients will convert to positive: however, this usually occurs during the first year of the disease. As more sensitive tests for rheumatoid factors are developed, the number of seronegative cases of rheumatoid arthritis will diminish. Another source of confusion is that RF is not specific for rheumatoid arthritis. Table 11.5 summarizes the more common disorders associated with the presence of RF. It should be noted that levels of RF tend to be higher in the rheumatic diseases than in the nonrheumatic disorders.

Rheumatoid arthritis seldom causes low back pain and almost never produces low back pain without concurrent symptomatic involvement of the peripheral joints and cervical spine. Therefore, no justification is found for ordering a rheumatoid factor test in a patient with isolated low back pain. It must also be realized that a positive rheumatoid factor test is neither the only, nor a mandatory, criterion for the diagnosis of rheumatoid arthritis (23).

Table 11.5

Frequency of Rheumatoid Factor (RF)[a] in Various Disorders

Condition	Percent Seropositive
Rheumatoid arthritis	75–80
Sjögren's syndrome	80–90
Systemic lupus erythematosus	30–50
Progressive systemic sclerosis (scleroderma)	20–30
Mixed connective tissue disease	20–30
Hepatic cirrhosis	20–30
Polymyositis/dermatomyositis	15–20
Juvenile rheumatoid arthritis	10–15
Normal subjects	3–15[b]

[a]Measured by latex agglutination method; sensitivity is lower with sheep hemagglutination method.
[b]The higher values are seen in the elderly and are usually associated with low titers of RF.

Antinuclear Antibodies

Antinuclear antibodies (ANA) are autoantibodies directed against antigenic components of cell nuclei, including nucleic acids and nucleoprotein complexes. These antibodies occur in many connective tissue diseases as well as a variety of other disorders. Although antinuclear antibody testing has traditionally been done by an immunofluorescent technique (IF-ANA, F-ANA), enzyme immunoassays (EIA) have been developed that appear to perform as well or better than these IF assays. Many laboratories now screen samples with EIA and confirm positive results with IF assays using human epithelial cells (HEp-2) (24, 25).

As shown in Figure 11.2, dozens of specific ANAs reactive with isolated cellular antigenic components have been described. Many of these autoantibodies are of research interest only at this time, whereas less than a dozen are of practical value for the physician.

Although the connective tissue disorders seldom cause low back pain, the vague arthralgias accompanying the conditions

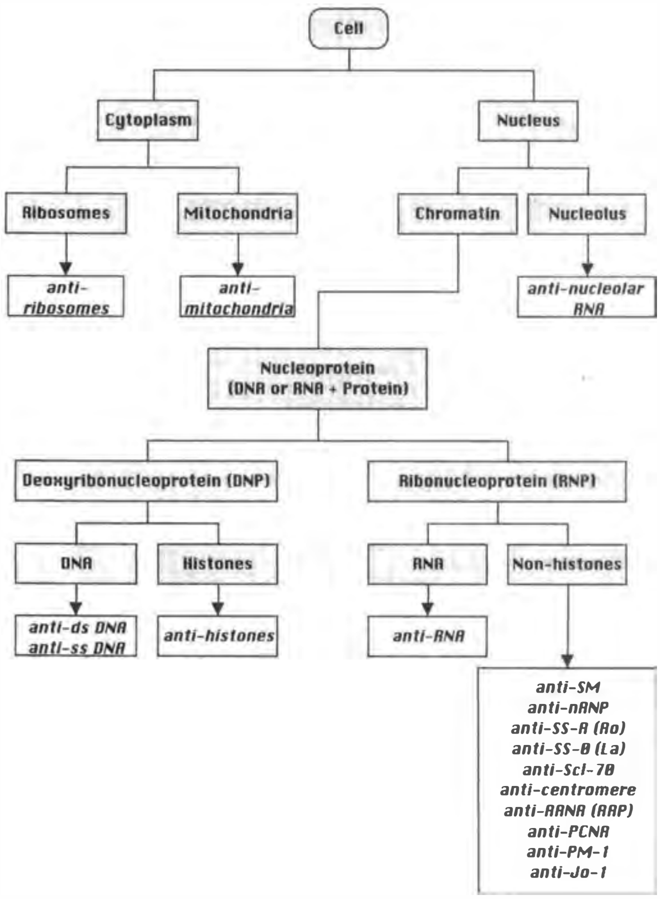

Figure 11.2. Autoantibodies in rheumatic diseases. *nRNP*, nuclear ribonucleoprotein; *PCNA*, proliferating cell nuclear antigen; *RANA*, rheumatoid arthritis nuclear antigen; *RAP*, rheumatoid arthritis precipitin; *ds*, double stranded (native); *ss*, single stranded.

Table 11.6

Frequency of Antinuclear Antibodies (ANA) in Various Disorders

Condition	Percent Positive
Systemic lupus erythematosus	90–98[a]
Mixed connective tissue disease	>95
Progressive systemic sclerosis (scleroderma)	40–95
Rheumatoid arthritis	30–60
Polymyositis/dermatomyositis	20–50
Sjögren's syndrome	40–80
Hepatic cirrhosis	20–30
Elderly patients	10–20[b]

[a]The higher values are obtained with HEp-2 or enzyme-linked immunosorbent assay (ELISA) methods.
[b]Usually low titers.

may often prompt the ordering of a laboratory arthritic profile, which usually includes an ANA test. Isolated low back pain is not an indication for ANA testing. The approximate incidence of ANA in various disorders is shown in Table 11.6.

Laboratories typically report the results of the ANA assay as both an antibody titer and the pattern of fluorescence. The latter is determined by the specific autoantibody interaction with the nuclear antigens. It can be helpful, along with the clinical picture, in deciding which specific ANA assays should be ordered (Fig. 11.3). It must be emphasized that antinuclear antibody test titers and pattern identification provide only presumptive evidence and must be interpreted in the overall clinical context. Although certain ANA types correlate well with disease states (e.g., the high specificity of double stranded anti-DNA with systemic lupus erythematosus), in many other cases relatively low test sensitivity and specificity are found.

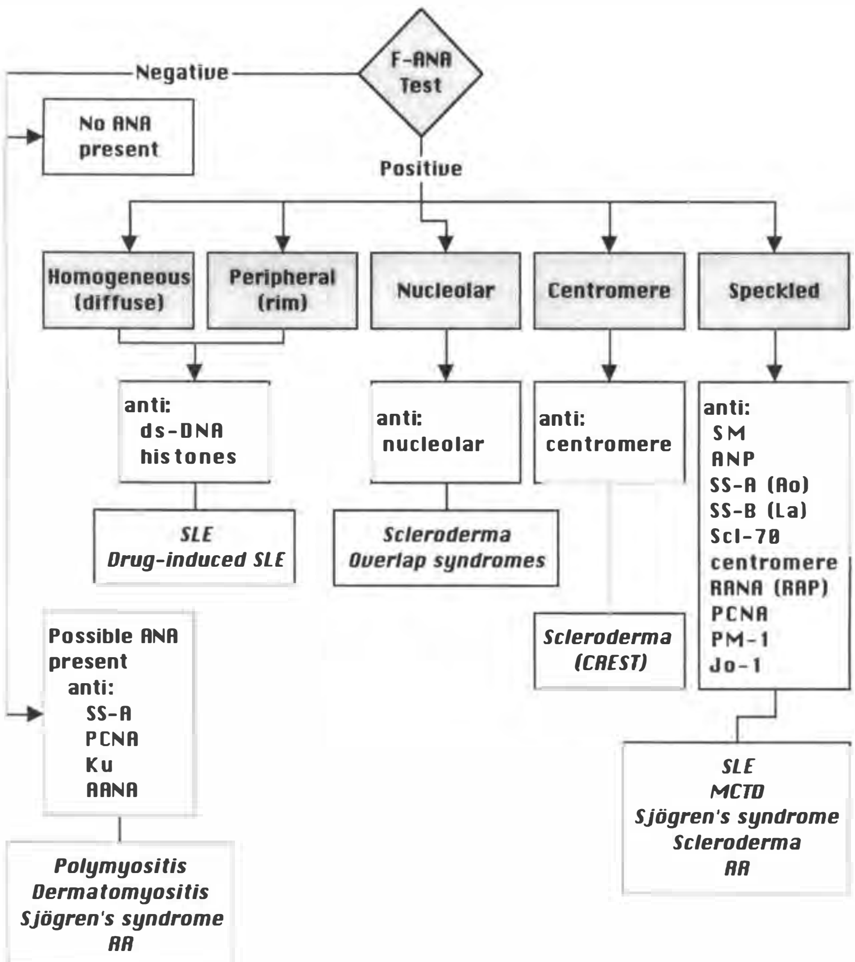

Figure 11.3. Patterns of ANA immunofluorescence. *ANA*, antinuclear antibodies; *RNP*, ribonucleoprotein; *PCNA*, proliferating cell nuclear antigen; *RANA*, rheumatoid arthritis nuclear antigen; *RAP*, rheumatoid arthritis precipitin; *ds*, double stranded (native); *SLE*, systemic lupus erythematosus; *RA*, rheumatoid arthritis; *MCTD*, mixed connective tissue disease.

Human Leukocyte Antigen (HLA) System

The HLA system, also referred to as the "major histocompatibility complex" (MHC), consists of a series of genes on chromosome 6 and regulates the production of proteins serving as antigenic markers on cell membranes and participating in important immune reactions. As can be seen in Figure 11.4, the MHC has several major categories, each of which has a series of numbered subcategories (alleles). Each parent contributes a haplotype, resulting in the offspring having up to two HLA antigens from each major category. Testing of peripheral blood lymphocytes establishes the HLA typing of an individual.

The HLA-A, -B, and -C antigens are located on most nucleated cells in the body. HLA-D antigens are found primarily on lymphocytes and macrophages. The HLA system functions to regulate the immune response of the body, including the killing of viral-infected target cells by cytotoxic T lymphocytes, recognizing foreign antigens, and controlling synthesis of complement factors (26). In addition to the use of HLA typing in organ transplantation, it has become increasingly recognized that certain HLA types are associated with an increased frequency of disease states. Table 11.7 lists several rheumatic diseases that show a greater frequency of specific HLA types than is found in the normal population.

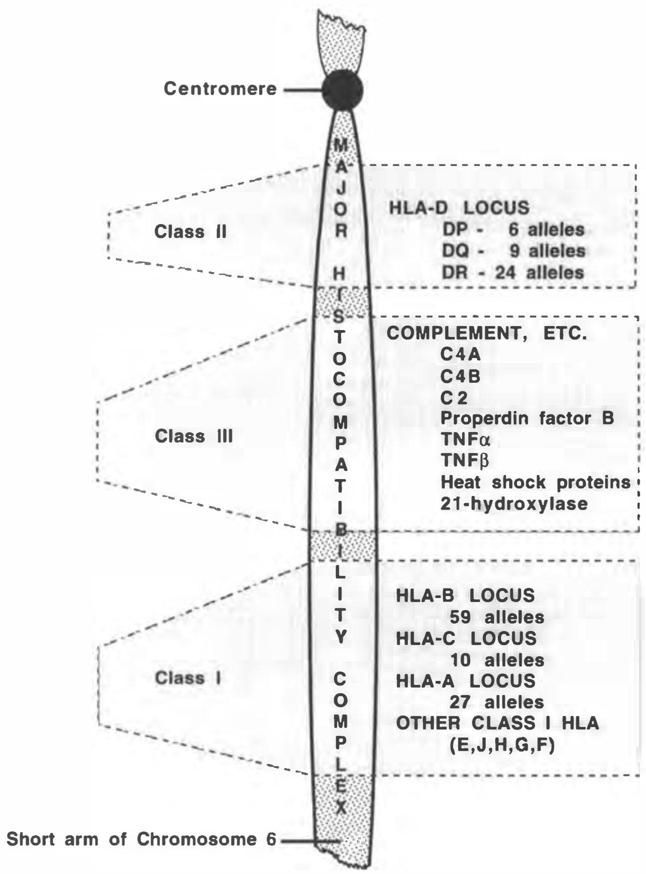

Figure 11.4. The major histocompatibility complex (HLA system) on the short arm of chromosome 6. Because of reclassification, numbering of foci is no longer consecutive and number of foci assigned to each region is subject to change. Class III proteins other than complement and factor B are only loosely linked to HLA.

Table 11.7

HLA-B27 Association with Various Disorders

Condition	Percent Positive
Ankylosing spondylitis	85–95
Reiter's syndrome	70–90
Yersinia reactive arthritis	40–95
Enteropathic (inflammatory bowel disease) arthritis	30–60
Psoriatic arthritis	20–50
Normal (healthy) population	3–8

The association of HLA-B27 with ankylosing spondylitis initially led to its use as a screening test in patients with low back pain. Subsequently it was recognized that several factors prevent HLA-B27 testing from being an effective diagnostic test except in certain unusual circumstances. These factors include the occurrence of the B27 antigen in up to 10% of the normal population, variation in the distribution of the B27 type among various ethnic groups, the association of B27 with other seronegative types of sacroiliitis and spondylitis, and the increased frequency of B27 in asymptomatic relatives of patients with ankylosing spondylitis. Because of its low predictive value (i.e., the ability to identify a specific illness in an unselected patient population) HLA-B27 testing is of no value as a screening test in low back pain patients. Similarly, typing would contribute little information in the presence of obvious radiographic and clinical evidence of ankylosing spondylitis. At best, the test may prove helpful in cases with equivocal radiographic findings; a negative result in these cases would argue strongly against ankylosing spondylitis (27).

Although several theories exist regarding the association of ankylosing spondylitis with HLA-B27, perhaps the most widely accepted is the concept of "molecular mimicry" in which the specific class I proteins produced by HLA-B27 so closely resemble antigens of certain bacteria (perhaps *Klebsiella* or *Proteus* organisms) that the immune system becomes confused and produces antibodies that attack both the bacteria and the HLA-B27 proteins, triggering an inflammatory cascade (28).

LABORATORY EVALUATION OF SPECIFIC DISORDERS

Osteoporosis

Osteoporosis, the diminution of bone density and mass, is a common disorder of the skeletal system producing considerable morbidity in the elderly population and a significant economic burden on the health care system. Although it is possible that uncomplicated osteoporosis can produce some dis-

comfort, most low back pain in osteoporotic patients is either caused by varying degrees of compression fracture or to concurrent degenerative conditions. Routine laboratory tests, including measurements of serum calcium, phosphate, and alkaline phosphatase levels are most often normal in osteoporosis. Little reparative process follows an osteoporotic compression fracture and accordingly little change is seen in alkaline phosphatase in that complication.

Specialized testing can be useful in identifying those patients with increased bone turnover and the development of osteoporosis. The most reliable indicators of loss of bone density are imaging procedures, including dual-energy x-ray absorptiometry (DXA) and quantitative computed tomography. Several potential biochemical markers of bone turnover exist, including serum alkaline phosphatase, urinary hydroxyproline, and serum Gla protein (osteocalcin). These laboratory tests either lack sensitivity or are impractical for widespread use. More recently developed and available in many laboratories is the measurement of urinary cross-linked N-telopeptides (NTx). As type I collagen fibers in osseous tissue degrade in osteoporosis and other disorders with increased bone turnover, the cross-linked N-telopeptides are released into the bloodstream and are subsequently filtered through the kidney. Elevated urine levels of cross-linked N-telopeptides are seen in osteoporosis, Paget's disease, hyperthyroidism, and other diseases with degradation of collagen. Urinary N-telopeptide levels have been demonstrated to be sensitive markers of early osteoporosis and may be useful indicators of response to therapy (29–31).

Lumbar Spine and Sacroiliac Infections

Infections of the lumbar spine can involve either the intervertebral disc or the vertebral body. Discitis is primarily a concern in children because the vascular supply to the disc diminishes in the adult. Discitis in adults is usually a complication of surgical intervention or is secondary to vertebral body osteomyelitis.

Discitis in children is characterized by low back pain, difficulty in walking, local tenderness, and loss of spinal motion (32). Many, but not all, cases have constitutional symptoms, such as nausea, irritability, and fever. Radiographs may not show diagnostic changes until several weeks into the disease process, so the more sensitive procedure of radionuclide bone scanning should be considered early. The white blood cell total and differential counts are often normal; however, the ESR is almost always elevated. Adult cases of discitis are often more difficult to diagnose because the condition typically follows lumbar disc surgery and, therefore, already some local discomfort often exists. The most reliable and earliest indicators of a postoperative discitis are the CRP and ESR. As discussed previously, elevations of CRP precede elevations of the ESR. The ESR is elevated in most postoperative patients during the first week; however, its elevation 2 or more weeks after surgery should prompt further investigation (4, 33). Bone scans in this type of patient are not reliable because the procedure is not sufficiently sensitive early in the case and because discectomy itself may cause an abnormal scan. In contrast to the ESR, the CRP offers an earlier and more sensitive marker of postsurgical discitis because of greater variation in the normalization period of the ESR in postoperative patients (Fig 11.5) (34). Magnetic resonance imaging appears to be sensitive to the vertebral end plate abnormalities in these patients, and it is indicated if the CRP or ESR fail to normalize within the expected recovery period following surgery (35, 36).

Vertebral osteomyelitis is most common in the thoracic and lumbar regions and is most often seen in patients with preexisting infections, especially involving the urinary tract. The

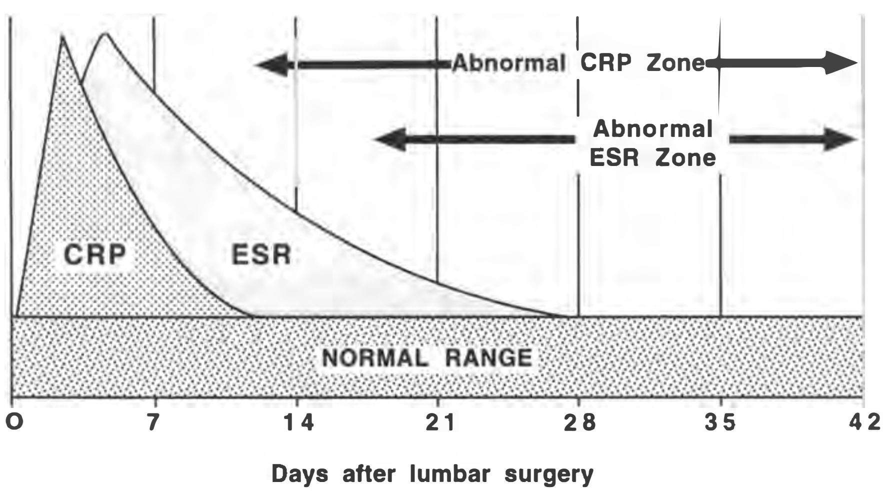

Figure 11.5. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) after lumbar surgery. The CRP peaks sooner and returns to normal sooner than the ESR. *Arrows* mark the zones during which a second peak or a persistently elevated level of CRP or ESR may signify the occurrence of a postoperative discitis. ESR values more than 40 mm/h can be significant. (Adapted from Larsson S, Thelander U, Friberg S. C-reactive protein (CRP) after elective orthopedic surgery. Clin Orthop 1992;275:237–242.)

onset of the disease is often subtle, and the diagnosis may not be made for several months. The patient typically complains of back pain, often with sciatica, and psoas muscle irritability is frequently found (1). With acute infections, the patient may be febrile and may have localized tenderness, redness, and warmth, whereas with chronic infections, fever and local findings other than tenderness are uncommon (37). Chronic vertebral osteomyelitis can occur as a sequela to an inadequately treated acute osteomyelitis or it may occur as a result of an insidious infection with an organism of lower virulence. As with discitis, the white cell count is not a reliable indicator of infection, but the ESR is elevated in most of the cases. Further evaluation of suspected cases of osteomyelitis includes plain film radiography and radionuclide bone scanning. In acute osteomyelitis, a lag of a week or more may occur between the onset of symptoms and the development of plain film findings of vertebral destruction. During this radiographic latent period, technetium-99 bone imaging has greater sensitivity. Indium-111-labeled leukocyte scans and gallium-67 scans have the additional ability to detect extraosseous infection sites, including paraspinal abscesses (38, 39). In chronic osteomyelitis, the plain film frequently shows abnormalities. Gallium-67 and indium-111 scans are more sensitive in chronic osteomyelitis than the standard technetium bone scan. MRI has high sensitivity and specificity for vertebral infections (40, 41).

Sacroiliac joint infections can mimic mechanical lesions of the low back, pelvis, and hip. As with vertebral osteomyelitis, sacroiliac infections may be the result of pyogenic organisms or a more insidious process, such as tuberculosis. Infection should be ruled out in all cases of unilateral sacroiliitis. Pain may be present in the low back, pelvis, and hip, and radicular symptoms are common, along with difficulty in weightbearing and pain on joint compression (42). Children with sacroiliac joint infection complain of hip, thigh, and buttock pain, and often have a positive Patrick's test as well as a painful limp (43). Almost all cases of sacroiliac infection, whether in adults or children, have an elevated ESR. As with vertebral infections, the white blood cell count is unreliable. CT scanning is of particular value in the diagnosis of sacroiliac joint infection.

Multiple Myeloma

Multiple myeloma is a hematologic malignancy in the lymphoma family, characterized by the monoclonal proliferation of plasma cells and the resultant hypersecretion of immunoglobulins and their subunits. The disease occurs after age 30, with most cases found in the sixth and later decades. The replacement of normal bone marrow with neoplastic cells, the alteration of normal ratios of immunoglobulin synthesis by plasma cells, the secretion of osteoclast activation factors, and the damaging effects of immunoglobulin fragments on renal cells result in anemia, abnormal serum and urine protein levels, increased susceptibility to infections, osteolytic lesions, and impairment of renal function (44).

The most common presenting symptom of myeloma is bone pain. Any marrow-containing bone is susceptible, with vertebral involvement being common, especially in the thoracic and lumbar areas. Plain film radiographs of the area may reveal classic multiple osteolytic lesions, but they are also likely to simply

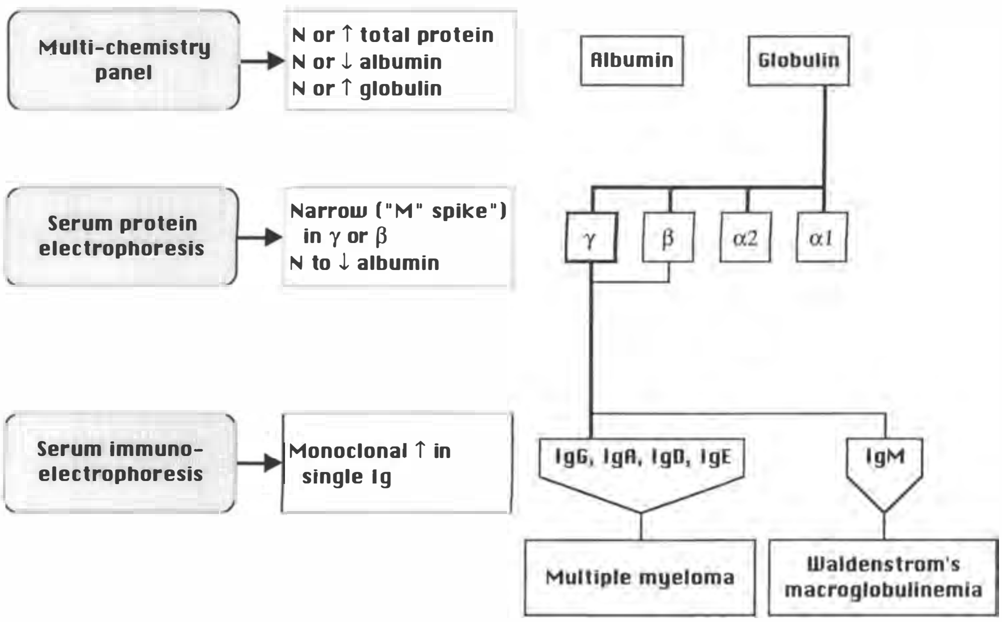

Figure 11.6. Serum protein evaluation in plasma cell dyscrasias. Sensitivity to protein abnormalities is greatest with immunoelectrophoresis and least with routine chemistry panels. Immunoglobulin abnormalities are typically within the γ-globulin region; however, some migration into the beta region may occur. N, normal; ↓ decreased; ↑, increased.

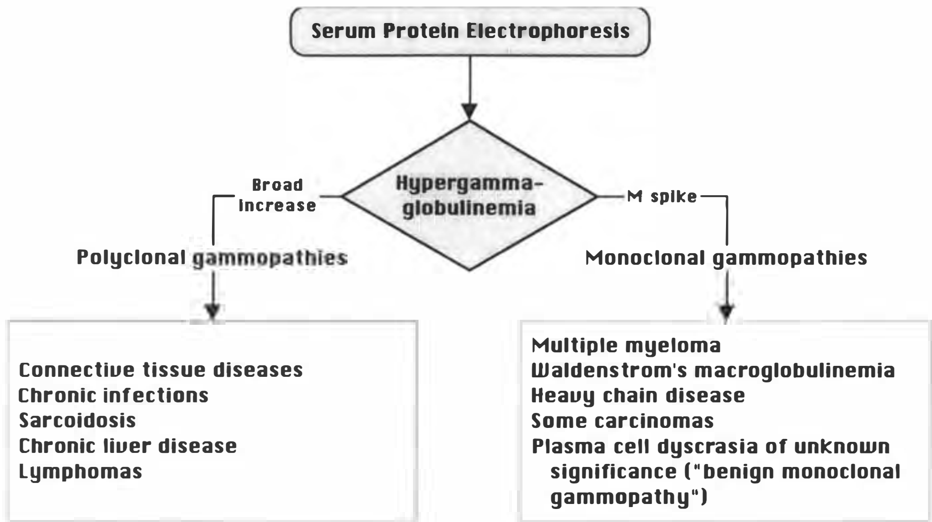

Figure 11.7. Serum γ-globulin electrophoretic patterns. Stimulation of multiple plasma cell types results in polyclonal increases of γ-globulin, whereas proliferation of a single plasma cell series results in a monoclonal increase.

show osteopenia and are frequently entirely normal in early cases. These patients with negative radiographs frequently have abnormalities evident on MRI (45, 46).

Clinical laboratory abnormalities occur before the osseous lesions become evident on imaging. Thus, unexplained bone pain in the middle-aged and older patient should prompt the ordering of appropriate blood and urine studies. The increased synthesis of immunoglobulins by the proliferating plasma cells produces quantitative and qualitative abnormalities in the plasma proteins. As shown in Figure 11.6 these changes can be detected by several methods.

A routine chemistry panel might show elevations in the total protein and globulin levels; however, hypergammaglobulinemia is nonspecific. It is best to pursue any case of hypergammaglobulinemia with serum protein electrophoresis, which allows for a determination of which type of globulin is responsible for the elevation. The pattern of electrophoresis is extremely helpful in evaluating globulin elevations. Conditions that cause stimulation of multiple clones of plasma cells produce a "polyclonal gammopathy" in which a diffuse elevation of several antibody types occurs. Such elevations are seen in chronic infections, connective tissue disease, sarcoidosis, chronic liver disease, and some lymphomas (Fig. 11.7). Multiple myeloma, in contrast, produces a monoclonal gammopathy that is revealed on protein electrophoresis as a narrow, homogeneous peak in the gamma or beta region. This finding would then be followed by serum immunoelectrophoresis, which will identify the specific type of immunoglobulin present and verify the monoclonal nature of the gammopathy. Light chains can also be assessed at this time. Most cases of multiple myeloma are IgG, with a lesser number being IgA. Less than 5% are IgD, and only a few cases of IgE myeloma have been reported. An IgM monoclonal gammopathy is characteristic of Waldenstrom's macroglobulinemia.

Excess production of free light chain portions of the immunoglobulins is common in multiple myeloma. These light chains (Bence Jones protein) are rapidly cleared by the kidney and can be demonstrated by immunoelectrophoresis to be present in the urine (Fig. 11.8). Some types of Bence Jones protein can cause renal tubular damage. Because 15 to 20% of the cases of multiple myeloma produce only light chains rather than complete immunoglobulins, if the clinical suggestion of myeloma is raised, urine immunoelectrophoresis should be ordered along with the serum protein electrophoresis to ensure detection of almost all cases of myeloma.

Secretion of osteoclast activation factors by the malignant plasma cells results in the lytic changes in bone and is accompanied by hypercalcemia in many cases. Alkaline phosphatase levels usually remain normal because of the lack of osteoblast activity. Bone scans are often normal for this same reason.

Suppression of erythropoiesis results in a normocytic anemia in most patients. Production of abnormal immunoglobulins causes the red blood cells to tend to clump together (rouleau), and this causes the ESR to increase (Table 11.8).

Figure 11.9 summarizes the diagnostic evaluation of multiple myeloma. The diagnosis of multiple myeloma is fairly straightforward in the middle-aged or older patient with bone pain, bony lesions on radiograph, anemia, or increased ESR, along with a monoclonal gammopathy; however, a need is still seen for confirmation by bone marrow evaluation. The bone marrow study will show at least 10% abnormal plasma cells, a finding which, when accompanied by clinical symptoms and either marked monoclonal globulin elevations (usually exceeding 3 g/dL), monoclonal light chains in the urine, or osteolytic lesions, becomes diagnostic (47). As shown in Figure 11.7, other causes of a monoclonal gammopathy exist, and consultation with a hematologist is recommended.

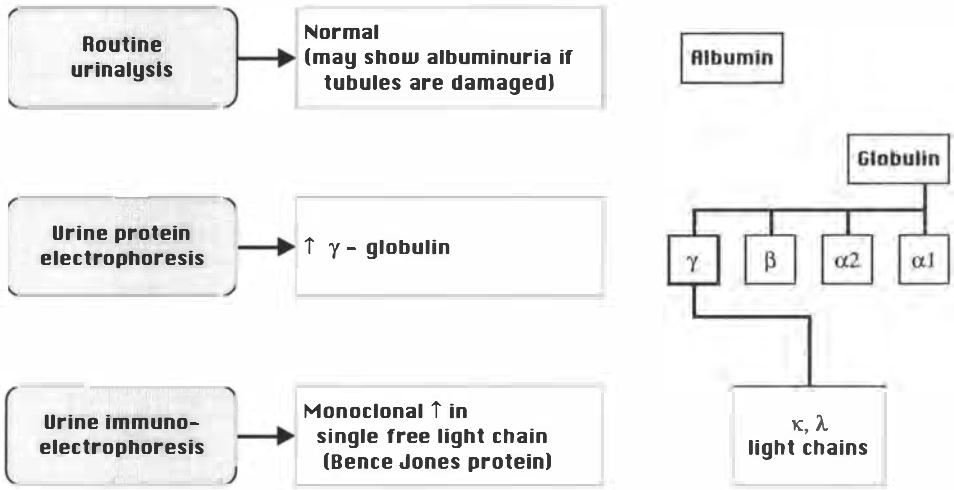

Figure 11.8. Urine protein evaluation in multiple myeloma. Routine urinalysis does not detect increases in globulin. Monoclonal κ or λ light chains may be the only protein abnormality detected in up to 20% of patients with multiple myeloma. ↓, decreased; ↑, increased.

Table 11.8

Common Hematologic Findings in Multiple Myeloma

Normocytic, normochromic anemia
Normal or decreased reticulocyte count
Rouleaux formation
Elevated erythrocyte sedimentation rate
Normal or low total white blood cell count
Normal differential count or relative lymphocytosis

Metastatic Carcinoma

Because of the extensive vascular network of the spine, metastatic disease of the vertebra is a relatively common occurrence. Beside bone pain, clinical findings can include pathologic compression deformity and osteoblastic and osteolytic lesions. The alkaline phosphatase level can be elevated in osteolytic as well as osteoblastic lesions, although it is more consistently and markedly elevated in the latter. Lytic lesions can also cause the serum calcium level to rise. The ESR and CRP may be increased, but these tests are not sufficiently sensitive to be relied on in the decision process. Radionuclide bone scanning is sensitive to metastatic disease and is an important means of differentiating the bony involvement from that caused by osteomalacia, osteoporosis, multiple myeloma, and other osseous disorders.

Metabolic Disorders

A number of metabolic disorders can produce orthopaedic complaints. Osteomalacia is the adult version of rickets, in which is seen deficient bone mineralization caused by disturbances in the vitamin D pathway (e.g., vitamin D deficiency or malabsorption), hypophosphatemia (e.g., malnutrition or malabsorption), or calcium deficiency. Symptoms of osteomalacia include bone pain and pelvic girdle muscle weakness. Radiographs may show diffuse osteopenia, which must be clinically differentiated from other causes (Table 11.9). Compression deformities of the vertebra can occur. Pseudofractures in the ribs, pelvis, or femurs would indicate osteomalacia rather than osteoporosis. Serum studies may show the calcium and phosphorus levels to be low-normal, and the alkaline phosphatase is usually elevated. Specific assays of the various forms of vitamin D are available.

Hyperparathyroidism is an unusual cause of back pain because most cases are detected before compression deformities can occur. In hyperparathyroidism caused by a parathyroid tumor or hyperplasia (primary hyperparathyroidism), the serum calcium level becomes elevated, while the phosphorus level drops. Measurement of the serum parathyroid hormone (PTH) level is important. Sensitive immunometric assays for PTH are now available that are not confused by circulating degradation fragments of PTH and are highly sensitive and capable of differentiating between hypercalcemia of primary hyperparathyroidism and hypercalcemia of malignancy (48, 49).

Cushing's syndrome is hypercortisolism resulting from iatrogenic steroid excess, a pituitary lesion causing excessive corticotropin secretion (Cushing's disease), or an adrenal adenoma. The primary orthopaedic complication is the development of osteoporosis and vertebral compression fractures. Most of these patients will also be obese and hypertensive. If hypercortisolism is suspected, the initial test of choice is a 24-hour measurement of urine free cortisol, which will show elevated levels in most Cushing's patients.

Inflammatory Lumbar Sacroiliac Disorders

Several inflammatory diseases, collectively referred to as "spondyloarthropathies," affect the spine and pelvis. These con-

ditions include ankylosing spondylitis, reactive arthritis (Reiter's syndrome), enteropathic arthritis, and psoriatic arthritis. Many of these patients will be HLA-B27 positive, and it has been suggested that the HLA-B27 antigen somehow makes an individual susceptible to infection with an arthritigenic organism (50). It is apparent that the process is multifactorial because, as previously pointed out, ankylosing spondylitis and the other spondyloarthropathies can occur in the absence of the HLA-B27 antigen. Clinical indicators of an inflammatory disease process include chronic low back pain of insidious onset in a young patient, morning stiffness, and relief of symptoms with exercise. In the absence of symptoms of inflammatory bowel disease (enteropathic arthritis), a personal or family history of psoriasis (psoriatic arthritis), or a recent episode of urethritis, cervicitis, conjunctivitis, or oral mucosal lesions (Reiter's syndrome), ankylosing spondylitis is the most likely diagnosis. Diagnostic emphasis is placed on the plain film radiographs and, to a lesser extent, on bone scanning . As mentioned, HLA-B27 testing is of limited value in the diagnostic evaluation of sacroiliitis and low back pain.

An intriguing relationship is found between spondyloarthropathies and intestinal inflammation. Changes in bowel flora and increased intestinal mucosal permeability may allow abnormal entrance of microbes, including *Klebsiella* and *Proteus* species, linked to spinal inflammatory diseases to cause excessive antigen stimulation (51). Intestinal permeability can be evaluated through the determination of the absorption of different polysaccharides (e.g., mannitol and lactulose). Specialty laboratories performing permeability tests also typically provide comprehensive stool evaluations, including the identification of disturbances in the bacterial flora. It has been suggested that the management of patients with ankylosing spondylitis could include dietary measures to reduce the quantity of *Klebsiella* microbes in the gut (52). Serial stool analyses can be used to follow changes in bowel flora following dietary intervention.

Table 11.9

Causes of Vertebral Osteopenia

Osteomalacia
Osteoporosis
  Primary
  Endocrine related (e.g., Cushing's syndrome, hyperparathyroidism, hyperthyroidism, diabetes mellitus)
Multiple myeloma
Metastatic lytic carcinoma
Osteogenesis imperfecta

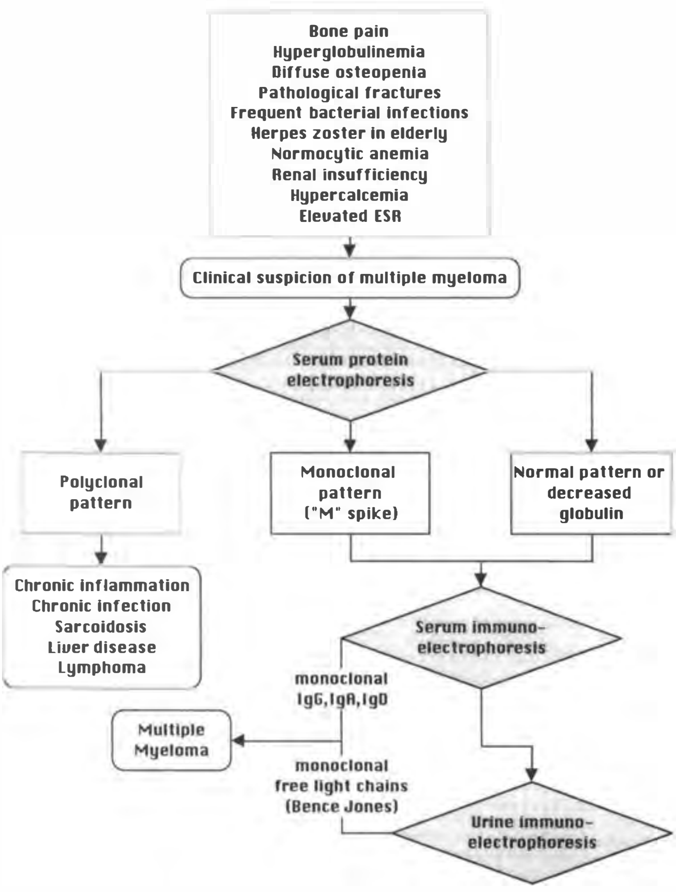

Figure 11.9. Sequence of laboratory evaluation in multiple myeloma. The combination of serum and urine protein studies enhances detection of the disease.

Polymyalgia Rheumatica

Polymyalgia rheumatica is a clinical syndrome of unknown cause that is characterized by muscle pain in the shoulder and pelvic girdles, constitutional symptoms, an elevated erythrocyte sedimentation rate, and a high incidence of temporal arteritis. It is primarily a disease of the elderly, with occurrence in patients less that 50 years of age being rare. Patients with polymyalgia rheumatica often have diffuse low back or pelvic pain and tenderness, typically accompanied by neck or shoulder pain. Although the onset is often sudden, in other cases it is gradual and the patient may initially be treated for a diagnosis of fibromyalgia for weeks to months. Patients may complain of vague constitutional symptoms such as fatigue, low-grade fever, and anorexia (53). Although the symptoms of polymyalgia rheumatica can cause disability, of greater concern is the temporal arteritis (giant cell arteritis) that so often accompanies the disease. The arteritis, in addition to causing headache and local tenderness, can result in loss of vision, cerebral ischemia, and jaw claudication. In almost all cases of polymyalgia rheumatica, the ESR and CRP are elevated (54). If the clinical suggestion of the condition is slight, the normal ESR and CRP studies rule out the diagnosis; however, if suspicion is strong, a normal ESR and CRP tests should not preclude further investigation (55, 56). The few patients with an ESR less than 40 mm/h tend to have a less severe clinical presentation (57).

Polymyositis

Polymyositis is an inflammatory muscle disease producing weakness in the shoulder and pelvic girdles. Clinical features include progressive proximal muscle weakness, difficulty in ambulation, and minimal pain or tenderness (unlike polymyalgia rheumatica). A skin rash may occur on the face and hands (dermatomyositis). Although low back pain is not present, the condition is considered in the differential diagnosis of lower extremity weakness. The diagnosis is based on the symmetric involvement of the proximal muscles, abnormal serum levels of the muscle enzymes (creatine kinase [CK], aspartate aminotransferase [AST, SGOT], and lactic dehydrogenase [LDH]), and a characteristic electromyogram (58). Specific antinuclear antibodies have been found, including PM-1 and Jo-1, but the sensitivity of these has varied, and they have not been widely adopted for clinical use.

Acquired Immunodeficiency Syndrome (AIDS)

A disease of the immune system, AIDS is caused by the human immunodeficiency virus (HIV). Infected persons are susceptible to opportunistic infection from several organisms and can manifest symptoms from both the primary HIV infection and the secondary infection. Of particular importance to the chiropractic physician is the high frequency of musculoskeletal complaints in HIV-infected persons, with some type of rheumatic manifestation in up to 75% of these patients. A frank arthritis affecting one or more joints occurs in approximately 10% of HIV-infected patients, and intermittent or acute arthralgias occurs in almost 45% (59). Involvement of the knees is most common, followed in order of frequency by the shoulders, elbows, ankles, neck, wrists, sacroiliac joints, hips, hands, feet, and lumbar spine. An increased incidence of Reiter's syndrome has been noted to occur, which may be the result of the immunosuppression allowing for infection with arthritigenic organisms. HIV patients, particularly intravenous drug abusers, are also at risk of secondary skeletal infections, including vertebral osteomyelitis (60). Testing for HIV infection should be considered in male homosexuals, hemophiliacs, intravenous drug users, or other high-risk individuals who complain of joint pain.

Lyme Disease

Lyme disease is a systemic disorder with a significant arthritic component. It is named after an outbreak of recurrent joint pain in many residents of Old Lyme, Connecticut, in 1975. Since that time, the disease has been reported across the United States, particularly in the upper Midwest and Northeast. The disease is caused by infection with a spirochete (*Borrelia burgdorferi*) transmitted through the bite of a tick. In most patients, the disease is heralded by a slowly enlarging anular skin lesion (erythema chronicum migrans) developing at the site of inoculation, accompanied by malaise, headache, arthralgias, and fever. Back pain occurs in approximately one fourth of the patients (61). The rash and constitutional symptoms last approximately 1 month if untreated, but can recur. Over the next several months (perhaps years), the patient may experience recurrent arthralgias, heart block, and neurologic abnormalities. In approximately 10% of the patients, chronic knee arthritis develops.

Early in the course of Lyme disease, at a point when backache occurs, the ESR may be elevated. The diagnosis is supported by an elevated antibody titer to the *B. burgdorferi;* however, commercial assays for the antibody may provide false-positive or false-negative results. When presumptive evidence of Lyme disease is found, based on the history, physical examination and nonspecific laboratory data, then a two-step antibody testing process is recommended to further support the diagnosis (Fig. 11.10) (62, 63).

Paget's Disease

Paget's disease (osteitis deformans) is characterized by an abnormal structural arrangement of bone in which there is disorganized activity of the osteoblasts and osteoclasts. This condition is of unknown cause and it primarily affects elderly patients. The disease progresses through stages of excessive bone resorption and excessive bone formation. Most patients are asymptomatic, with their disease discovered incidentally in the evaluation of another complaint. Although low back pain is classically described as a common feature of Paget's disease, it appears that the pain is most often caused by an accompanying osteoarthritis rather than to the pagetic process itself (64). Laboratory evaluation shows the serum alkaline phosphatase level to be markedly elevated. If a patient with known Paget's disease develops increased pain and a sudden rise in the already elevated alkaline phosphatase level, the development of a sarcoma should be suspected. In addition to serum alkaline phosphatase levels, urine levels of cross-linked N-telopeptides (NTx) and hydroxyproline are usually elevated as a result of the degradation of the involved type I collagen (65).

Infective Endocarditis

Subacute infective endocarditis is an infection, usually bacterial, of the endocardium in patients with an existing cardiac defect. The development of symptoms is often quite insidious, and the diagnosis may not be made for weeks or months. The classic features of subacute endocarditis, consisting of low-grade fever, anorexia, murmur, and embolic phenomena, are well known by physicians, but often unappreciated is the frequent occurrence of musculoskeletal complaints. As many as one fourth of patients with subacute endocarditis have low back pain, and in many this is the presenting complaint (66, 67). Moderate to severe low back pain, buttock pain, sacroiliac tenderness, and flank pain have all been noted in patients with en-

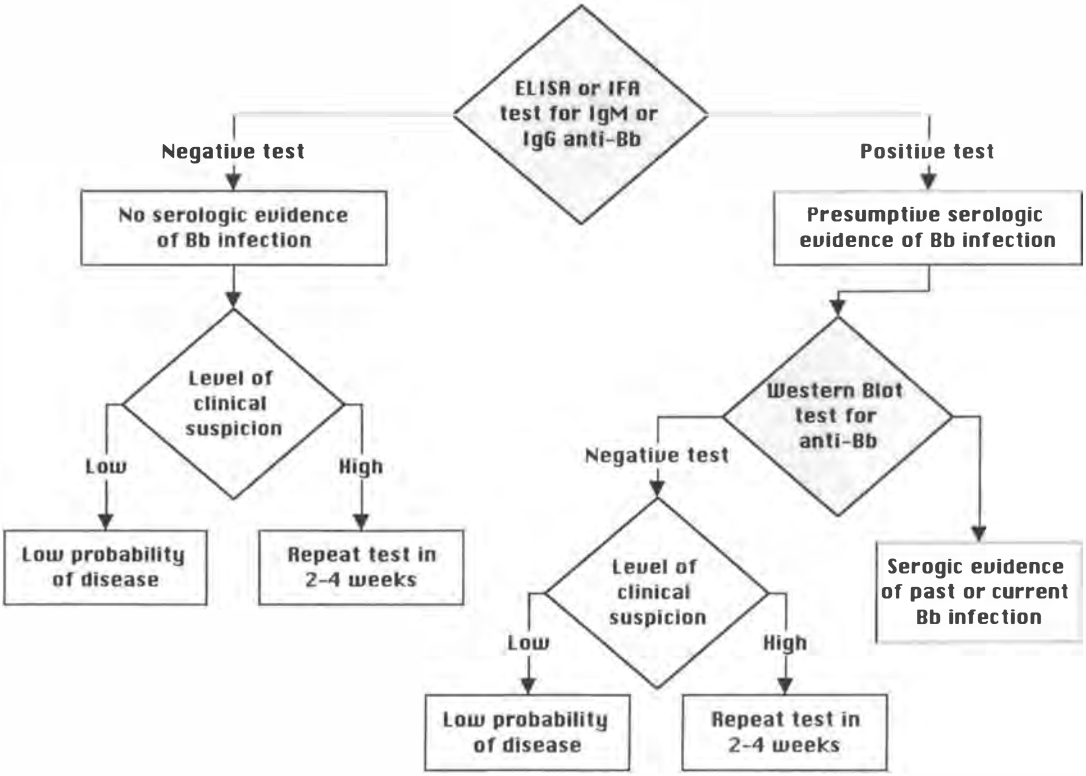

Figure 11.10. Two-step approach to serologic diagnosis of Lyme disease. *Bb, Borrelia burgdorferi; ELISA,* enzyme-linked immunosorbent assay; *IFA,* immunofluorescent assay.

docarditis (68). The white blood cell count is often normal and regional radiographs usually are negative, although signs of osteomyelitis or discitis may be seen. The sedimentation rate is elevated in approximately 75% of the cases, whereas the CRP is elevated in almost all cases (96%) (69). Echocardiography provides strong presumptive evidence, and the diagnosis of infectious disease is usually confirmed by blood cultures.

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Care of the Intervertebral Disc Patient

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chapter 12

There is one thing stronger than all the world, and that is an idea whose time has come.

—Victor Hugo

COST AND OUTCOME CONSIDERATION OF BACK CARE

Characteristics of Low Back Pain Patients

Persistent low back pain is most common among white, well-educated, affluent, employed people in their mid to late 30s and early to mid 40s, who have had low back pain intermittently for 10 years; report significant functional impairment at work, at play, and at home; and do not display significant psychological distress. Most patients have spondylitic abnormalities involving root compression or lumbar instability or both, with root compression as the primary cause of the complaint. Myofascial syndrome and lumbar instability are the next most common diagnoses. Three of five persistent low back pain patients are prescribed an additional course of conservative therapy, one in five is prescribed surgery, and the rest are prescribed no treatment. Chronic pain syndrome patients are characterized by significant behavioral and psychological comorbidities (1).

Prevalence and Cost of Low Back Pain Treatment

People who repetitively lift more than 40 pounds each day are three times more likely to have low back pain than those who lift less than 10 pounds. During a lifetime, 60 to 80% of persons will develop low back pain, 5% annually develop it, and 15 to 20% have symptoms at any given time (2).

There are 5.2 million low back disabled Americans with 2.6 million permanently disabled. Temporary or permanent disability costs 80% of the expense in treating low back pain patients. Surgical rates in the United States are approximately 100 per 100,000 population. Direct and indirect medical costs are between $50 and $100 billion annually, with 75% attributed to the 5% who become disabled (2). Approximately 95% of the cost of low back pain goes to 25% of the sufferers (3).

Ten percent of chronic low back pain patients account for 80% of the cost of care and 90% of low back pain patients are well within 6 weeks. The few who develop chronic pain and disability account for up to 70 to 90% of the costs of back pain in health care, work loss, social security, and compensation. General practitioners treat acute low back pain with rest and analgesia for 96% of their patients, physiotherapy for 73%, manipulation for 19%, and traction is requested for 8% (4).

Chronic Low Back Pain Costs Are High

Total costs for 157 workers with chronic back pain (average age 38 years), 131 men and 26 women; out of work for an average of 10 weeks, combining both medical and compensation expenses, totaled $6,188,867 for the 157 cases, which averages out to $39,419 for each worker (5). In 84% of the cases, the total cost exceeded $10,000. A patient who had leg pain cost $18,000 more than a patient with back pain.

An abnormal x-ray study or computed tomography (CT) scan boosted the cost of a typical case by nearly $50,000. Litigation was associated with an extra $26,000 per case (5).

Chronic low back pain disability is the most expensive benign condition in industrial countries. It is also the number one cause of disability in persons under age 45. After 45, it is the third leading cause of disability (4).

In 1990, the total annual cost of back care in the United States was $85 billion, with the direct costs being 20 to 33% and indirect costs 67 to 80% of the total expenditure. The mean cost per industrial back injury claim from 1986 to 1988 in 45 states was $6800, with a median cost of $400. Chronic back pain patients account for only about 10% of the total

number of back pain sufferers, but they account for 50 to 85% of the cost (6, 7).

Of the 20 to 25% of adult Americans who have at least one episode of back pain care every 3 to 5 years, most will be well within a month with few practitioner visits. Five percent of the patients will have four or more episodes of back pain care during this time frame, and about 8% of episodes last longer than 6 months and involve more than 20 practitioner visits (8).

After care, 66 to 75% of people continue to have at least mild back pain 1 month after seeking care and 33% have moderate pain. At 1 year, 33% report intermittent or persistent pain, with one of seven reporting severe pain (9). At 15-year follow-up, 175 of 513 patients reported recurrent episodes of pain and 47 reported continuous pain (10).

Low back pain cases at Liberty Mutual Insurance Company represented 16% of all claims but 33% of all claims costs in 1989; the mean cost per case was $8321 and the median cost per case was $396. Medical costs represented 32.4% of the total costs; indemnity costs (i.e., payment for lost time) represented 65% (11).

In patients seeking care, 55% saw an orthopaedic surgeon, 64% a primary care doctor, 25% a chiropractor, and 29% a physical therapist. Chiropractor mean number of visits was 15.7, whereas for a physical therapist it was 17.2 visits. Chiropractic patients were more likely to be users of all types of medical care; more likely to be employed, white, insured, in good health; have a higher income; and were more likely disabled and to have less severe current pain (12).

Cost of Intense Exercise Stabilization for Chronic Low Back Pain Patients

Intensive pelvic stabilization exercises to isolate and rehabilitate the lumbar spine musculature in 895 chronic low back pain patients showed 76% of patients completing the program had excellent or good results with maintenance of the improvement in 94% of patients 1 year later. The average cost of the entire program including all physician fees and home exercise equipment was $2250 (13).

Is intense exercise stabilization for chronic low back pain cost effective? Answer: Programs for chronic lumbar pain usually cost much more, sometimes over $10,000. For comparison, in the city, magnetic resonance imaging (MRI) costs $1000, a discogram $2000, and a single epidural injection $690. A program costing $10,000 to $15,000 would be very cost effective if the patient returned to gainful employment and stayed out of the health care system. Nelson et al.'s study (12) suggests that aggressive exercise is a valuable, cost-effective treatment for chronic low back pain.

Low Back Pain Better Treated Outpatient Than Hospitalized

Diagnostic testing accounts for half the hospitalizations in the United States for patients with nonspecific back pain and herniated discs and the other half for pain control. Forty percent of patients are admitted by family physicians or internists, and 40% by orthopaedic or neurologic surgeons. Most of the tests and treatments identified are known to be safe in the outpatient setting. Many hospitalizations for "medical back problems" are unnecessary because the tests and treatments are safe in the outpatient setting, suggesting a need for improved outpatient and home-based alternatives to hospitalization (14).

Chiropractic Physicians Are Positioned as Outpatient Clinicians

Chiropractic physicians are trained as outpatient clinicians, capable and accustomed to working within restricted parameters of diagnostic facilities while being forced to make competent clinical impressions on which to build treatment protocol. Perhaps no other member of the health profession has been so highly trained in the clinical practice arena without the sophistication of radiology and laboratory facilities for detailed workups. The chiropractic doctor is highly skilled in using faculties of observation, palpation, plain x-ray study, and clinical diagnosis to evaluate patients. It seems that such training is what is being called for in medicine today—a time of cost conservation with a demand for continued quality care. Chiropractic can thank its ancestors for their insight in preparing our profession for this time in conservative health care delivery.

Geography Determines Surgical Rates

A growing realization is found that a patient's chances of having surgery may be determined as much by longitude and latitude as by symptoms and signs. A ninefold difference in fusion surgery rates is seen among regions of the United States and a 12 times difference in back surgery rates between cities 200 miles apart in New England. Most forms of spine surgery have not been subjected to systematic scientific assessment. An apparent correlation exists between the per capita number of spine surgeons and the surgery rate (15).

Surgical rates for the treatment of low back pain increases if a Blue Cross Blue Shield of Iowa subscriber is a woman or older than 44 years of age (16). The surgery is more likely to take place in hospitals with an occupancy rate less than 62%, fewer than 774 staff members, fewer than 257 beds, or no residency programs (12). The total number of spine operations in the United States approaches half a million annually with 319,000 disc excisions performed in 1992 (17).

Only Cesarean Section and Tubal Ligation Instigate More Hospitalizations Than Back Surgery

The rate of back surgery in the United States is 40% higher than in any other country and five times greater than England and Scotland. Back surgery rates increased almost linearly with the per capita supply of orthopaedic and neurosurgeons in the country. Back surgery ranks only behind cesarean section and tubal ligation as a cause of surgical hospitalization (18).

Children with Disabling Back Pain

One third of all school children have back pain at some time, and about 15% of all children have disabling pain or have received medical care for back pain (19).

No Treatment May Be Best Treatment

Patients (316) treated by chiropractors and physical therapists reported that 47% treated by physical therapy thought that the treatment made the situation worse, 32% found little or no effect, and 21% a good effect. Of those treated by chiropractic 39% reported that their condition worsened, 31% little or no effect, and 30% a good effect. The data from the patients suggest that chiropractic treatments were somewhat better than physiotherapy. It is noted that the number of patients whose low back pain worsened was higher than the number that benefited from either physiotherapy or chiropractic treatments (20).

STAGING OF LOW BACK PAIN BY TIME

- Acute low back pain is 6 weeks or less
- Subacute low back pain is 6 to 12 weeks
- Chronic low back pain is 12 weeks or longer

Low back pain is the most common cause of disability in persons under 45 years old. Eighty to 90% of low back pain attacks resolve within 6 weeks (21).

FACTORS AFFECTING LOW BACK PAIN PROBABILITY AND SEVERITY

Pre-Employment Radiographs

Pre-employment radiographic studies have little effect in curbing the cost of back problems in industry; lumbosacral radiographs are not helpful in predicting who is more likely to make a back injury claim, or those few who make up the vast majority of the costs for industrial back pain by becoming disabled for more than 6 months. The radiation exposure is not justified by their predictive value as a pre-employment screening tool (22).

Spina bifida occulta, spondylolysis, spondylolisthesis, transitional vertebra, Scheuermanns' disease, disc space narrowing or osteophyte formation greater than 1 mm, retrolisthesis, and facet tropism were not found with greater frequency in pain patients than in nonpain patients (22).

Range of Motion Testing Is Invalid

The American Medical Association Guides to the Evaluation of Permanent Impairment were tested for validity on 81 healthy subjects. All of the normal subjects were noted to have some degree of impairment ranging from 2 to 38.5%, with a mean value of 10.8%, showing that impairment may be overestimated by up to 38%. The current method of impairment determination based on spinal motion may not accurately reflect impairment in many patients. Alternative methods of impairment evaluation should be developed that are more specific for individuals with true functional impairment and that account for age-related differences in spinal motion (23).

Job Satisfaction

In 1990, the Boeing Aircraft Company, with a population of 3000 volunteers followed up for 4 years, found the only significant predictive factors for the recurrence of low back pain were job dissatisfaction and distress as expressed on the Minnesota Multiphasic Personality Inventory (24).

Age

Most patients have a long history of recurrent back pain prior to the onset of sciatica, but when a frank disc herniation occurs, leg pain usually overshadows the back pain. The peak incidence of herniated lumbar discs is in adults between the ages of 30 and 55 years (25).

SURGERY CONTRASTED WITH CONSERVATIVE CARE OF SCIATICA PATIENTS

Disc surgery may be a luxury that society cannot afford (26). Except for the few cases wherein an emergency loss of neurologic function occurs, most cases of disc herniation recover on their own with conservative care. The 30,000 failed spinal operations per year consume an "extraordinary amount" of societal resources. "Can we afford," asks Hanley, "to expend such large sums of money on this small percentage of the population with a self-limited problem?" (27).

The number of available orthopaedic surgeons exceeds the HMO requirement of 5/100,000 by 150%. Even a 50% reduction in the number of residents would not bring the level of orthopaedic surgeons into line with HMO projections for more than a quarter century (28).

During the first 6 to 8 weeks of care for herniated disc cases, little reason is seen to order tests or plan invasive management because only a small percentage of patients with herniated discs should consider surgical intervention (26). Long-term results of surgery are only slightly better than both conservative measures and the natural history of a lumbar disc herniation (29).

Factors Predicting Outcome for Lumbar Disc Herniation

Absent crossed straight leg raise (SLR) sign, spinal motion in extension that does not reproduce the leg pain, large extrusion or sequestration, 50% relief of leg pain within the first 6 weeks of onset, a 12-year educational level, good fitness, and progressive return of neurologic deficits within the first 12 weeks predict good nonoperative outcome for lumbar disc herniation (30).

Surgical Numbers Increase

During the periods 1979 to 1981 and 1988 to 1990, in each sex, the rate of hospitalizations for cervical spine surgery increased more than 45%, with the rates for cervical fusion surgery increasing more than 70%. The rate of hospitalizations with lumbar spine surgery increased more than 33% in each sex, with the rate for lumbar fusion surgery increasing more than 60% in each sex, the rate for lumbar disc surgery increasing 40% among males and 21% among females, and the rate for lumbar exploration or decompression surgery increasing more than 65% in each sex.

Surgery rates are influenced by the ratio of surgeons to population. Between 1980 and 1990, the number of neurosurgeons and orthopaedic surgeons per capita increased by 24% (31).

Drop in Surgical Success

The results of surgery for disc herniation in 1950 showed 95% good results. Studies from Germany, Sweden, and other countries today find good results in only about 75% of patients.

"Are we operating on the wrong patients?" asks Nachemson. "Are we doing too much? Are the insurance benefits [for continuing disability] too great? Or is the pain perception of patients changing? I don't know" (32).

Some observers believe the overall success rate for disc surgery in the United States to be closer to 70% than it is to 90%, although precise data are not available. At 12 months, 51.5% of the surgery patients had a good outcome after disc surgery, 28.4% had a moderate outcome, and 18.6% had a bad outcome (33).

Only 31 of the 118 patients (26%) who underwent lumbar disc surgery in a city compensation setting returned to full duty and were considered satisfactory. The surgical treatment of lumbar disc disease in this group of patients resulted in a 74% rate of permanent disability (34).

Motor and Sensory Alterations

Approximately 50% of the neurologic changes associated with motor weakness or sensory deficit will be retained after conservative care. Weakness of the big toe or some sensory loss of the outer foot may, however, be an acceptable adverse side effect for successful conservative management, when one considers the unpredictable risks of surgery (35).

Absence of Presurgical Neurologic Deficits

Patients complaining of deterioration after operation constituted 16.9% of 36 patients in one study (36). In 10 of the 36 patients, the lumbar operation was repeated, in 6 of them once, in 3 twice, and in 1 three additional times. The repeat operations were mainly re-fusion after failed fusion, and laminectomy at a segment different from the first one.

Results of the ten patients who underwent at least one repeat operation were, four immediate failures of which three had been immediate failures and one a late failure after the first intervention; six late failures of which one had been immediate; and five late failures after the first operation.

In 20 of the 36 patients, lumbar surgery carried out in the absence of any preoperative sign of neurologic deficit raises the question of the indications for surgical intervention. No doubt, the driving factors in many of these cases were the patient's pain and functional disability without response to conservative therapy, patient and, possibly, physician frustration, chronicity of the low back pain, and radiologic evidence of disc damage (36).

Quality of Life After Disc Surgery

Disc surgery does not appear to return patients to work any faster or prevent long-term disability any more effectively than nonoperative treatments. It does, however, offer a significant benefit in terms of quality of life and symptom alleviation. It can afford 5 extra months of comfortable living over a 10-year period, compared with nonoperative treatments.

Disc surgery is reasonably cost-effective and well within the range of most other medical treatments regarded as standard and appropriate. Of employed patients, 87.8% of the surgical group reported an improvement in quality of life at 1 year versus 65.3% of the nonsurgical group (37).

Postsurgical Adhesions

Hyaluronic acid decreases the biomechanical strength of extradural adhesions following disc surgery when compared with use of fat graft or no interpositional membrane (38). Polyactive, an elastomeric segmental copolymer, consistently yielded less scar adhesions as compared with free fat graft (39). Placing Gelfoam or free-fat graft over the nerve root and dura after excision of a herniated lumbar disc had no effect on patient outcome regarding symptoms, functional status, or MRI findings. Placing an interposition membrane over the nerve root may have no beneficial effect on the outcome of lumbar disc surgery (40).

Muscle Weakness

Disc surgery patients seem to develop long-term strength and lifting deficits, particularly in extensor strength (41).

PEDIATRIC LUMBAR DISC HERNIATION

Herniated lumbar nucleus pulposus is rare in the pediatric population with the surgically proved incidence being between 0.8 and 3.2%. A 15-year-old boy with three level lumbar disc herniations refused surgery and underwent conservative care consisting of nonsteroid anti-inflammatory drugs, passive extension, lumbar traction, segmental mobilization, and a progressive program of dynamic lumbar stabilization exercises resulting in an asymptomatic state and return to limited sports.

The report on this patient also reiterated an important law of caring for low back disc herniation patients:

1. The absolute indication for surgical intervention of herniated nucleus pulposus is neurogenic bladder or bowel dysfunction (cauda equina syndrome) and progressive neurologic deficits.
2. Relative indications include intractable radicular pain and lateral spinal stenosis (42).

Another study of 48 adolescent patients, average age of 16 years, undergoing discectomy found excellent or good outcomes in 91% of the patients, and poor outcomes in 9% at follow-up. In the patients treated nonoperatively, the results were rated as excellent or good in 25% and poor in 75%. Six-year follow-up study suggests that discectomy yields excellent to good long-term results in children and adolescents (43). Good results following discectomy do not justify prolongation of conservative care beyond that recommended for adults (44).

OUTCOME MEASURES OF PRIMARY VERSUS REPEAT LUMBAR SPINE SURGERY

Nerve root compression caused by recurrent disc herniation or bony compression respond well to repeat decompression. Sciatica caused by nerve-root scarring is seldom improved by a repeat operation (45).

The success rate after primary lumbar surgery ranges from 80 to 95%. Good results after revision lumbar surgery, which range from 28 to 81%, are rarely comparable to primary surgery. Factors predicting a favorable outcome of second lumbar surgery are a noncompensable injury, absence of litigation, achieving a solid fusion, and the patient not disabled from work. Age, number of previous operations, and poor psychological profile were not predictive of an unsuccessful outcome from additional surgery (46).

Successful reoperation occurred in young patients working outside the home who had an initial period of improvement after the first surgery, and who had fewer surgical levels on primary surgery and a revision procedure incorporating an anterior interbody arthrodesis (47).

Sixty-nine percent of reoperated patients were on a disability pension compared with 40% of the patients who underwent a single surgery (48).

Workers' Compensation patients presenting within 1 year with recurrent complaints after discectomy and whose radiologic findings indicated a same-level, same-side recurrence represent extremely poor outcome risks for repeat discectomy (49).

IMAGING AND SURGERY OFTEN ARE INAPPROPRIATE CARE FOR LUMBAR DISC HERNIATION

Premature or unnecessary CT or MRI studies ordered in the evaluation of patients with low back pain may have a significant effect on overall health care costs. Moreover, the isolated finding of a herniated disc, without corresponding clinical signs, can lead to inappropriate surgical referral (50).

Magnetic resonance imaging and physician charges are the major cost in care of low back pain patients, accounting for 19% of the cost in patients not meeting appropriate criteria for testing (32).

Patients pressure doctors to order high technologic tests such as MRI and doctors must resist the temptation to order them. Between weeks 4 and 6 the patient passes from the acute into the chronic phase, and prognosis begins to change. At 6 weeks all patients still complaining of low back pain are studied radiographically. Patients showing no improvement should be referred to a specialist. Patients who are improving should continue their current program without further testing. At 12 weeks, any patient who is still symptomatic requires referral to a specialist (51).

WHEN DOES DIAGNOSTIC IMAGING BECOME NECESSARY IN A PATIENT WITH RADICULOPATHY?

The status of the patient determines the necessity of diagnostic imaging in patients with radiculopathy, which can be determined by the following (52):

Order Imaging Studies:	If the Patient Has:
Immediate, emergency basis	Loss of bladder or bowel function or rapid neurologic deterioration in neurologic function
As soon as possible, to avoid future permanent neurologic deficit	Slow, progressive neurologic loss of motor or sensory or reflex function
After a 4- to 6-week delay while conservative treatment is attempted to resolve the pain; sooner if the patient is severely incapacitated and bedridden	No neurologic deficits but severe pain
After a 6- to 10-week delay and depending on results of the clinical examination	Mobility with some leg pain, unresponsive to conservative treatment
Earlier rather than later	More leg pain than back pain
Perhaps never, because x-ray study results are unlikely to change treatment protocol significantly	Back pain only

Radiographic Study at 7 Weeks

For the patient with a first episode of low back pain, present for less than 7 weeks, who has not been treated or who is improving with treatment, no radiographs of the lumbar spine are indicated unless one or more of the following exceptions are obtained (53):

- Age over 65
- History suggesting high risk for osteoporosis

- Symptoms of urinary tract dysfunction
- Symptoms of persisting sensory deficit
- Pain worsening despite adequate treatment
- Intense pain at rest
- Pain worse at night
- Fever, chills
- Unexplained weight loss
- History of injury of sufficient violence to cause fracture
- History of repetitive stress of sufficient severity to cause stress fracture
- Recurrent back pain with no radiographs in the past 2 years
- Previous lumbar surgery or fracture
- History of radiographic abnormality elsewhere reported to patient but with no films or reliable report reasonably available
- History of finding from other study (e.g., bone scan or gastrointestinal series) that requires spine radiograph for correlation
- Anticipation of need for another study or treatment that would be facilitated by preliminary radiograph (e.g., epidural injection)
- Patient unable to give a reliable history

Atypical physical findings including:

- Significant motor deficit
- Unexplained deformity

Special psychological or social circumstances including:

- Crippling cancer phobia focused on back pain
- Inability to secure another evaluation within 7 weeks from the onset of pain
- Need for immediate decision about career or athletic future
- High risk for violent injury
- Need for legal evaluation

What views of the lumbar spine should be taken? In general, anteroposterior and lateral views only should be done initially (53).

WHEN DOES A PATIENT WITH BACK AND/OR LEG PAIN BECOME A SURGICAL CANDIDATE?

Patients with a definite diagnosis of ruptured lumbar intervertebral disc (IVD) and sciatic or other radicular pain with neurologic signs and symptoms should be carefully observed and treated by nonsurgical means for 4 to 8 weeks, unless the patient presents with progressive loss of motor, bladder, or bowel function or has excruciating pain that cannot be relieved by nonoperative treatment (54).

Recommendations for Intervention for Disc Herniation

The American Academy of Orthopaedic Surgeons (55) recommends the following interventions for disc herniation:

1. Functionally incapacitating pain in the leg, extending below the knee with a nerve root distribution.
2. Nerve root tension signs (positive SLR) with or without neurologic abnormalities, fitting the radiculopathy.
3. Failure of clinical improvement after 4 to 8 weeks of conservative therapy.
4. Confirming imaging study; abnormal myelogram, CT, or MRI correlated to the physical signs and distribution of the pain.

Studies on the results of disc hernia surgery all emphasize inappropriate patient selection as the leading cause of surgical failure (55).

Microsurgery

McCulloch (56) outlines the following indications for microsurgery:

- Bladder and bowel involvement
- Increasing neurologic deficit
- Significant neurologic deficit with significant and persisting SLR reduction
- Failure of conservative treatment—**the most common reason for surgical intervention**
- Recurrent episodes of sciatica

NATURAL COURSE OF DISC HERNIATION

Large Percentage of Sciatica Patients Have Prior Low Back Pain

One or more earlier attacks of acute lumbago were reported by more than 90% of 280 verified herniated lumbar disc patients with radiculopathy. An average of 10 years passed before the first attack of sciatica, which often developed insidiously. No factors were found that could differentiate between a transitory attack of low back pain and a pain that was the forerunner of sciatica.

Approximately 25% of the patients improved during a 2-week hospitalization. Another 25% with serious symptoms and signs underwent surgery. The remainder (126 patients) with uncertain indication for surgery were randomized for either conservative treatment or surgical intervention.

Examination after 1 year of observation showed a satisfactory result in 90% of the surgically treated patients and 60% in the conservatively treated group (57, 58).

Conservative Care for Disc Herniation Patients Before Surgical Consideration

Patients with radicular symptoms and signs caused by a herniated lumbar disc but without definite indications for immediate surgery should be observed for 2 to 3 months before a final therapeutic decision regarding operation is taken (57, 58).

Provided warning signals of a critical condition can be excluded, indications are for an initial 4-week conservative approach to acute sciatica. Lack of satisfactory improvement after 4 to 6 weeks indicates radiologic examinations are needed. If a clear, visible herniation is demonstrated, the choice between continued conservative therapy and surgical interference must be considered. Many factors are involved in this evaluation, including the natural course of the disease (58–60).

Most Sciatica Patients Well With 4 Months of Conservative Care

Energetic nonoperative care results in successful recovery in approximately 90% of verified herniated disc patients with radiculopathy treated with traction therapy, which is perhaps the treatment most commonly recommended in cases of radicular sciatica.

The real challenge to the physician's knowledge, experience, art, and psychological insight arises when conservative effort fails and surgery needs to be considered. Definite indications for surgery are cauda equina syndrome, intolerable pain, and progressive muscle weakness. The decision to continue with the conservative regimen or recommend surgical intervention should be made with the patient. Extended conservative care is the patient's option (59).

Schvartzman et al. (60) found that surgical care was not more cost effective than nonsurgical care, and it had no better outcome than continued conservative management in a comparison study of 55 white male truck drivers who presented with acute sciatica. Findings were 91% confirmed L4-L5 herniated discs. After 12 weeks of bed rest, physical therapy, and drug treatments proved ineffective, 25 patients opted to undergo lumbar discectomy.

No significant difference was found between the two groups in outcome or cost of treatment. Results were good or satisfactory in 80% of both and the average total medical and compensation cost during 1985 to 1989 was $56,054 for surgical treatment and $55,638 for conservative treatment. The conservatively treated patients lost significantly more time from work over the 5 years than patients who underwent surgery—a total of 97.4 weeks and 78.9 weeks lost, respectively.

Schvartzman et al. recommend an initial 3 months of physical therapy, and if the patient's condition does not deteriorate during that time, conservative measures are continued. The patient should ultimately make the decision whether to proceed to surgical intervention. *A patient not responding to the initial trials of conservative therapy has the option to undergo continued conservative treatment* (60).

Surgery Seldom Necessary

The life-time incidence of surgery for back pain and sciatica ranges from 1 to 3%; 50% of patients with disc hernia-induced sciatica will recover spontaneously in 4 to 6 weeks. Although surgery hastens the recovery from disc hernia-induced sciatica, it seems to have little influence on risk of recurrence (19).

Only 5 to 10% of symptomatic lumbar disc patients require surgery, and the best overall plan is to help patients avoid back disease by encouraging them to modify risk factors, provide them with preventive exercises, and teach them the proper and improper methods of lifting (50).

Most cases of back pain and even most clinical manifestations of symptomatic disc herniation (pain, reflex loss, imaging changes, and muscle weakness) resolve with bed rest and analgesia. The proportion of all persons with low back pain who undergo surgery for disc herniation is only about 2% (50). Long-term outcome of surgical care is only slightly better than conservative care for lumbar disc herniation (61).

Does Delaying Disc Surgery Cause Permanent Nerve Damage?

If a person undergoes surgery, regardless whether early or late, within a 12-week period, it does not really influence outcome in terms of future motor function, according to Frymoyer (62). In fact, a slightly increased risk is seen of sensory loss if surgery is performed too early. There is the same long-term relief of pain. No evidence currently exists and no medicolegal reason is seen, to intervene early with surgery—even when the patient has a dropped foot. If a person has cauda equina syndrome, however, that is an acute, surgical situation (62).

The cauda equina syndrome occurs in only 1 to 2% of all lumbar disc herniations that come to surgery, so its prevalence among all patients with low back pain is about 0.0004 (four cases per 10,000 patients) (63).

What Does a Surgeon Say About Spinal Surgery?

A surgeon's response to the question of spinal surgery was reported as follows: "Do you need to have surgery? I make my living doing surgery, but the answer I give my patients is you do not need surgery. In fact, if you look at the literature on herniated discs, patients who don't have surgery and patients who do have surgery feel about the same after one year. There is no significant difference between the two groups.

"The reason to recommend surgery is that you might have continued decreasing function in the leg such as loss of sensation or loss of motor power in any muscle group. A particularly

worrisome, but rare, symptom is the inability to control your bowel or bladder movements.

"If you do not have the problems mentioned above, the decision to have surgery must be made by the patient, and it's usually recommended if there's no improvement after an adequate course of conservative therapy" (64).

VALIDITY OF SUBJECTIVE INSTRUMENT MEASUREMENTS OF PATIENT RELIEF

Oswestry Disability Scale

In assessing the outcome of surgery in the lumbar spine, the percentage change in the Oswestry Disability scale is reliable and independent of surgeon bias, and it correlates well with the patients' subjective assessments of improvement (65).

Quebec Back Pain Disability Scale

The Quebec Back Pain Disability scale is a 20-item self-administered instrument designed to assess the level of functional disability in individuals with back pain. The following factors are used in evaluating patients (66):

1. Get out of bed.
2. Sleep through the night.
3. Turn over in bed.
4. Ride in a car.
5. Stand up for 20 to 30 minutes.
6. Sit in a chair for several hours.
7. Climb one flight of stairs.
8. Walk a few blocks (300 to 400 m).
9. Walk several miles.
10. Reach up to high shelves.
11. Throw a ball.
12. Run one block (about 100 m).
13. Take food out of the refrigerator.
14. Make your bed.
15. Put on socks (pantyhose).
16. Bend over to clean the bathtub.
17. Move a chair.
18. Pull or push heavy doors.
19. Carry two bags of groceries.
20. Lift and carry a heavy suitcase.

Response options: (0–5): 0, not difficult at all; 1, minimally difficult; 2, somewhat difficult; 3, fairly difficult; 4, very difficult; 5, unable to do.

Comparisons with the Roland Morris and Oswestry scales suggest that the Quebec scale may be more reliable and is at least as sensitive to change as the best available measures. The scale can be recommended as an outcome in clinical trials, to monitor the progress of patients participating in treatment or rehabilitation programs and to compare different groups of back pain patients (65).

COMPARISON OF SURGICAL APPROACHES

Which to Choose?

The results of both chemonucleolysis and automated percutaneous lumbar discectomy in a prospective randomized study were generally disappointing because 48% of the overall population entering the study considered treatment a failure and 20% submitted to open laminectomy within 6 months (67).

Percutaneous Discectomy

Percutaneous discectomy is barely better than placebo treatment. Discectomy continues to be the "gold standard" for disc surgery (68).

Gill and Blumenthal (69) feel that percutaneous discectomy, because of its safety and efficacy, should play a valuable and additional role in the treatment of the herniated nucleus pulposus in the years to come. It has the lowest morbidity of all invasive treatment options in the care of patients with herniated lumbar discs.

Little change in the appearance of the disc lesion with successful percutaneous discectomy outcome may intimate that the mechanism by which the procedure relieves pain remains to be elucidated (70). No association was demonstrated between change in size of the herniation or disc space and clinical outcome or amount of nuclear material that was removed at nucleotomy (71).

Broad-based disc protrusion showed best relief (80% success) with automated percutaneous lumbar discectomy. Patients with a disc protrusion with a narrow dye base had an overall success rate of only 53%. The outcome depends, however, on the shape of the protruded nuclear material as shown by CT discography, which makes this examination a *conditio sine qua non* before treating patients with a disc protrusion with automated percutaneous discectomy (72).

Use of automated percutaneous discectomy can be a wise decision. In selected patients it can reduce sciatica, but it only completely eliminated sciatica in 5% of patients with a follow-up period of 2.5 years (73).

Chemonucleolysis: Differing Opinions

Chemonucleolysis produces inferior short-term results and offers no advantage over conventional discectomy and costs more than conventional laminotomy (74). Chymopapain injection was found superior to placebo at 10-year follow-up in a study of 60 patients (75). With adherence to strict criteria for selection and performance of chemonucleolysis, this procedure is as effective as laminectomy, and it is safer, without the risk of arachnoiditis and its associated charges and disability that can occur after laminectomy. In addition, chemonucleolysis has proved to be substantially less expensive than laminectomy in short and long-term periods, with the potential to significantly reduce the financial burden of health care in the United States. Having been time-tested, chemonucleolysis is an attractive alternative to laminectomy (76).

Laser Discectomy

LASER is an acronym for **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation. This kind of electromagnetic radiation is created by external stimulation of a laser medium that has been put into a condition termed "population inversion." This active laser medium can be solid material (ruby crystal), gas (helium neon), dyes (rhodamine), or semiconductor material such as gallium arsenide. Laser light is characterized by three qualities: it is monochromatic, coherent, and collimated (77).

Percutaneous endoscopic laser-assisted discectomy (PELD) is intended to decompress the lumbar spinal nerve by selective endoscopic removal of the herniated parts of the nucleus pulposus.

Nonendoscopic percutaneous laser disc decompression (PLDD) is intended for internal decompression of the disc. This is achieved with coagulation and shrinking of central parts of the nucleus pulposus by the applied laser energy. The tip of an 18-gauge cannula is placed into the center of the lumbar disc space under fluoroscopic control via a posterolateral approach. Laser-light is then applied to the nucleus through a 400 to 600-u quartz fiber introduced through the cannula (77).

Percutaneous insertion of the laser fiber into a lumbar IVD permits the ablation of the nucleus pulposus and central anular fiber by creating a 1.5 cm^3 defect. The disc is decompressed, with minimal thermal change in the neural elements or adjacent end plates. In experimental animals, the disc is replaced by dense fibrous tissue that eventually undergoes ossification (78).

No question is found that these techniques reduce disc volume and can lead to improvement in some patients' symptoms. However, these patients often have minimal disc displacement, and almost every one of them improves with a graduated exercise program. With such a vigorous approach to conservative management, it is difficult to find 5% of patients who are candidates for the procedure (79).

Laser discectomy, however, seems to be an unpredictable procedure, and a research study of it was abandoned because of adverse changes in the discs of the subjects. The success rate of laser discectomy in relieving sciatica is "a flip of a coin." High rates of complications and reoperations and low success rates show laser discectomy clearly worse than the natural history of contained disc herniations (80). Acute foot drop following laser-assisted discectomy showed it may produce reversible and irreversible nerve root injuries (81).

Comparison of Surgical Procedures

Success with open discectomy is 90%, 53% with chemonucleolysis, and 31% with laser discectomy (82).

No differences were found between those having microdiscectomy and those having laminectomy so far as perioperative bleeding, complications, inpatient stay, time off work, or end results short-term or at 1 year were concerned (83). Higher herniated nucleus pulposus recurrence rates have not been found in using a limited discectomy technique. Advocates of the microdiscectomy technique have reported a faster return to work, a quicker functional recovery, and a shorter hospital stay in their patient groups, secondary to the less extensive exposure (84). Laminotomy is more effective in relieving radicular pain, and it must be considered the standard of surgical treatment for lumbar herniated disc disease (85).

Extreme Lateral Lumbar Disc Herniation Excision

Extreme lateral lumbar disc herniation represents 10 to 12% of all disc herniation, more common in the upper lumbar spine in patients between 50 and 60 years of age. Because extreme lateral lumbar disc herniation affects nerve roots at the level of herniation, it often mimics classic disc herniations at the level above. It often presents with anterior thigh and groin pain, quadriceps weakness, and it may be accompanied by a positive femoral stretch test. Often, little back pain is present and the Lasegue's sign is usually negative. Rapid localization and safe excision is done of extreme lateral lumbar disc herniations without the need for bone resection (86).

COMPLICATIONS OF LUMBAR DISC SURGERY

It is important for the chiropractor to be aware of surgical complications because of the increasing number of failed back surgical syndromes seen in our offices. Chiropractic treatment protocol is affected by knowing this information.

Neurologic Complications of Lumbar Laminectomy

Among the causes of complications of lumbar laminectomy are (87):

1. Dural and nerve root injuries: "Battered Root"—burned, lacerated, or torn in surgery.
2. Cauda equina syndrome: Artery of Adamkiewicz, which supplies most blood to lower spinal cord, is damaged.
3. Formation of scar tissue: Arachnoiditis (extradural scar tissue formation), or perineural fibrosus (intradural scar tissue formation).

Reflex Sympathetic Dystrophy

Reflex sympathetic dystrophy (RSD) developed in 11 patients after surgery for lumbar spondylolisthesis or lumbar instability that was associated with degenerative disc disease or osteoarthrosis of a facet joint. After the operation, all patients had burning pain, vasomotor dysfunction, and dystrophic changes in the lower limb and foot. The symptoms began 4 days to 20 weeks after the operation.

Prerequisites for RSD are (*a*) a painful lesion; (*b*) an abnormal autonomic reflex rather than a normal sympathetic reflex; and (*c*) a diathesis, or unusual susceptibility. Irritating stimuli for lumbar nerve pain syndromes are processed in the lumbar nerve root ganglia (88).

Chronic, intractable pain is treated successfully by electroconvulsive therapy, which raises the questions of possible cerebral contribution to the pathophysiology of RSD (89).

Venous Thrombosis

Thromboembolic disease and its treatment remain a leading cause of morbidity and death among patients undergoing orthopaedic procedures. Deep venous thrombosis rates after spinal surgery range from 0 to 20%, with pulmonary emboli occurring in up to 8% of patients (90).

Nucleus Pulposus Emboli

Following discography using 52% diatrizoate meglumine and 8% diatrizoate sodium, a fatal systemic reaction occurred. Postmortem examination showed nucleus pulposus pulmonary emboli on random lung sections. Speculation was that the spasmodic back extensions imposed compressive forces on the lumbar vertebrae, causing nucleus pulposus to be extruded into the vertebral marrow sinusoids (thus creating emboli) and possibly causing these emboli to flow anteriorly into the anterior external vertebral plexus, which resulted in pulmonary emboli exclusively with no spinal cord emboli (91).

Epidural Fibrosus—Not a Surgical Condition

The pathogenic role of epidural fibrosus as seen on MRI in pain generation is questioned as it is found to be similar in symptomatic and asymptomatic patients after lumbar discectomy. In patients with persistent or recurrent sciatica after lumbar discectomy, in whom epidural fibrosus is the only neuroradiologic finding, repeat decompression should be discouraged (92).

Epidural Fat Graft-Induced Nerve Root Compression

Six years following L5–S1 lumbar disc herniation surgery, a 36-year-old woman developed severe sciatic pain on the same side as before surgery. The left S1 nerve root was compressed with a piece of free fat autograft in the foramen, which was used in the first operation (93).

Scar Tissue or Dorsal Ramus Damage from Surgery Cause Failed Back Syndrome

Severe postoperative failed back syndrome patients have dorsal ramus lesions covered by scar and local paraspinal muscle atrophy at the corresponding segments. Disturbed back muscle innervation and loss of muscular support leads to the disability and increased biomechanical strain, which might be one important cause to the failed back syndrome. It may be possible to develop operating techniques that save back muscle innervation better than the ones in current use (94).

Paraplegia and Quadriplegia

Two cases of thoracic level paraplegia after lumbar spinal surgery showed cord edema and spinal cord infarct in the thoracic region, representing acute spinal cord infarcts in the "watershed" region of the thoracic cord (95). Extreme head rotation and neck rotation during lumbar disc surgery led to vertebrobasilar vascular thromboses and embolism (96).

Seronegative Arthritides May Have Poorer Surgical Outcomes

Patients who are HLA B-27 positive may be more likely to have a poor outcome from disc surgery (97).

DIFFERENTIAL DIAGNOSIS OF RECURRENT HERNIATED DISC MATERIAL FROM SCAR TISSUE

Gadolinium-DTPA, a paramagnetic agent, is injected prior to MRI of the lumbar spine. The differential between scar and disc material is that the gadolinium-DTPA localizes to vascular tissue. Because granulation scar tissue is highly vascular, it will enhance and appear as an area of hyperintensity enhancement on T1-weighted images, whereas the avascular herniated nuclear material will not show any enhancement and will remain hypointense on MRI (87).

Differential diagnosis of recurrent disc herniation from scar tissue involves the following clinical facts: (*a*) recurrent disc herniation usually occurs on the same side as the original lesion within the first 5 to 7 years after initial surgery, (*b*) if the patient complains of a gradual increase in symptoms during the first 6 months after surgery a gradual formation of epidural scar tissue is suggested, whereas a more abrupt onset after 6 months indicates recurrent disc herniation. Also, spinal instability and arachnoiditis must be considered (98).

MRI Postsurgically Is Error Filled

Magnetic resonance imaging is unreliable in distinguishing between scar and a retained or extruded disc fragment in the early postoperative period (less than 6 months) (99). In 257 lumbar spine surgical patients who showed symptoms suggesting persistent or new disc herniation 6 to 18 months after surgery underwent contrast-enhanced MRI, which found evidence of disc herniation. These patients were associated with a significantly greater frequency of repeat surgery with poor relief (100).

Postsurgical Multifidees Muscle Changes

Inactivity and axon injury contribute to atrophy of the multifidii muscles in disc patients. These pathologic structural changes correlated well with clinical outcome, and, most importantly, they can be reversed and diminished by adequate therapy (101).

SURGICAL APPROACHES: BENEFITS AND PROBLEMS

Severe Tissue Injury Patients Respond Best

The best outcome after surgery befalls those with the most severe tissue injury, that is, patients with ruptured anulus with complete perforation of the nuclear material respond better and faster than those with intact anulus protruding discs (102).

In 244 patients operated on for posterolateral first time disc herniation, 77% had absence of leg pain directly after the operation and 14% later on. In 8% the pain persisted. Ninety-four percent of the patients described themselves as excellent or good, and 6% as the same or worse, and 94% went back to work. Average sick leave after operation was 11 weeks (103). Improvement was seen in 46% at 4-month follow-up, in 59% at 1 year, and 63% at 2 years. Recovery was seen more often in patients who had preoperative symptoms for less than 1 year (104). Preoperative pain, surgical outcome, and neurologic recovery were similar in single- and double-level herniation (105).

Percutaneous Discectomy

Discitis, vascular injury, and hematoma are risks of percutaneous discectomy. Success rates for percutaneous discectomy range from 60 to 87% with two reports showing rates as low as 53 to 55%. Replacing microsurgical laminectomy with percutaneous discectomy is not open for discussion because of insufficient evidence and value (106).

Microdiscectomy

Patients undergoing microlumbar discectomy for lumbar disc herniation have less postsurgical pulmonary morbidity and temperature elevation than those treated by the lumbar laminectomy and discectomy (107). Seventy-five consecutive cases of outpatient disc excision found it a practical alternative for selected patients requiring disc surgery (108).

SPINAL FUSION—CONTROVERSIAL

This section begins with a discussion of lumbar spine fusion based on a published meeting paper on the subject by noted authorities from throughout the world. Advanced technology such as new spinal implants, including screws, rods, plates, cages, and biologic grafting materials have contributed to the growth of knowledge in the field of spinal fusion (109).

Rates of Lumbar Fusion

The rates of lumbar fusion procedures are increasing rapidly, particularly for lumbar spinal stenosis in older patients. Fusion rates vary remarkably between geographic areas and surgeons. Spinal stenosis with spondylolisthesis has higher costs and complications when a fusion procedure rather than decompressive surgery alone is performed.

Of an estimated 279,000 low back operations performed in persons older than 20 years in 1990, 46,500 (17%) were lumbar fusions. For comparison, in that same year hospital records show that approximately 120,000 total hip replacements, 20,000 revision total hip replacements, 500,000 cholecystectomies, and 400,000 coronary artery bypass graft procedures were performed. Lumbar spine fusion was performed for IVD disorders in 51% of patients, spondylolisthesis in 24.3%, spinal stenosis in 10.5%, spondylolysis in 10%, and vertebral fracture in 7.3% (109).

From 1979 to 1990, low back surgical rates increased by 55%, from 102 to 158 per 100,000 adults (at least 20 years old). Lumbar fusion rates increased 100%, from 13 to 25 per 100,000 adults. Lumbar spinal stenosis was the condition showing the greatest increase for fusion surgery (110).

Geographically in the United States, lumbar surgical procedures varied greatly. In the western United States, the surgical rate was 124 per 100,000 adults; it was 190 per 100,000 adults in the South. Lumbar fusion varied from 18 per 100,000 in the West to 30 per 100,000 in the South. The fusion rates were 40% lower in the West than in the South. Lumbar surgical rates were highest in the South and fusion rates were highest in the South and Midwest (111).

Among surgeons, fusion rates vary dramatically. For example, fusion rates in New Hampshire were 56% higher than the mean northern New England rate, whereas Maine was 57% lower. This suggests that professional uncertainty or differences in physician practice patterns are the reason (112).

The United States shows the highest rates of low back surgery of 12 Western nations. The likelihood of having surgery in the United States is 35% higher than in other countries, whereas back surgery rates in Sweden, England, Scotland, and Manitoba, Canada are less than one third of those in the United States (113).

Cost of Fusion

In-hospital cost of noninstrumented laminectomy fusion has exceeded laminectomy alone by 50%. Instrumented fusion at laminectomy is 100% more costly than laminectomy alone (114). Hospital bills of 40 patients (20 each in 1986 and 1993) who had undergone single-level and double-level lumbar arthrodesis showed the hospital cost (mean) for single and double-level spinal arthrodesis increased from $7457 (1986) to $19,712 (1993). In inflation-adjusted 1993 dollars the actual increase was 97%. Most dramatic was the 638% increase in implant costs (115).

Fusion Treatment of Degenerative Lumbar Disorders

Spinal fusion plays an important role in the treatment of degenerative disorders of the lumbar spine. Decompression laminectomy or foraminotomy for spinal stenosis yields 80% good or excellent results when the facet joints are preserved, with 20% showing increasing postoperative slip. With partial face-

tectomy, good or excellent results were found in only one third of patients, with two thirds showing poorer results and increasing spondylolisthesis slip. Fusion allowed 90% good to excellent results; improved outcomes were found with fusion for degenerative spondylolisthesis (116).

Discectomy without fusion at L4–5 and L5–S1 showed excellent and good results in 81% of patients in both groups. The L4–L5 disc is most susceptible to axial torsion and it is the most common site for lumbar instability; thus, discectomy at this level would yield less favorable results than discectomy at L5–S1 because of the perceived increased failure rate due to mechanical pain and instability (117).

Patients with spinal pain lasting 4 months or longer and unremitting to any treatment are candidates for spinal fusion if discography reproduces their pain syndrome (118). Positive discography pain has shown 88% of patients to receive satisfactory pain relief with spinal fusion (119).

Disc excision and posterior lumbar interbody fusion (PLIF) was performed on 39 men and 23 women who had low back pain symptoms longer than 6 months, were out of work for at least 4 months, and derived no relief from medication or rehabilitation. No patient had disc herniation, spinal stenosis, abnormal movements of spinal motion segments, or prior back surgery. The consistent finding was a positive pain response during discography combined with internal disc degeneration and disruption. Ultimately, 89% claimed to have satisfactory results and 93% returned to work, mostly to predisability assignments; PLIF was successful, as determined radiographically, in 94% of cases (120).

Conservative Treatment for Discographically Produced Low Back Pain

Although positive discography is viewed as a valid diagnostic technique, the results of fusion surgery are often disappointing. Conservative treatment results are comparable with or better than those reported for surgical treatment of nonradicular, discogram-positive low back pain. Pain can be expected to improve in up to two thirds of patients with simple conservative therapy for discographically induced low back pain (121).

Fusion surgery is often performed based more on clinical anecdote than on rigorous scientific study. The literature does support the use of spinal fusion in several degenerative disorders of the spine, including lumbar spondylolysis, discogenic pain, facet joint syndrome, lumbar degenerative disease, isthmic spondylolisthesis, and spinal stenosis (116).

Fusion Increases Complications

Lumbar fusion, when compared with surgery without fusion, is associated with a substantial increase in complications, mortality, and resource. A high short-term cost is found with this procedure, both medically and financially (122).

Spinal fusion for discogenic pain is based on the hypothesis that the disc is sensitive to painful stimulus and that a solid fusion relieves the pain by reducing the stimuli to the pain-receptive structures (123). In considering spinal fusion, lumbosacral supports have been used preoperatively to judge the effect of immobilization. If pain relief occurs while the patient wears the brace, theoretically, spinal fusion should also relieve the patient's pain. The posterior longitudinal ligament and the outer fibers of the anulus fibrosus are pain-sensitive structures capable of producing back pain. Recommended patient selection fusion criteria include (*a*) disability for more than 1 year, (*b*) failure of conservative treatment, and (*c*) a positive provocative discogram (123).

Severe Degenerative Disc Disease Benefits from Fusion

In 36 patients with lumbar degenerative disc disease 26 also had a degenerative spondylolisthesis, 18 had spinal stenosis, and 26 had lateral stenosis. They were operated on with an extensive root release, followed by a posterolateral fusion with a pedicle screw technique. Sixteen patient outcomes were classified as excellent, 8 good, 9 fair, and 3 poor. Patients classified as failed backs could probably benefit from further fusion surgery (124). Another study showed no advantage for fusion over surgery without fusion (125).

Fusion Doubles Second Surgery Rate

In a study of 388 patients undergoing spinal fusion, 68% were work disabled and 23% required further lumbar spine surgery 2 years postfusion. Five markers of severity predicted work disability outcome (older age at injury, longer time from injury to fusion, increased time on work disability before fusion, increased number of prior low back operations, and increased number of levels fused). Receiving instrumentation with fusion doubled the risk of reoperation. Most patients reported that back pain (67.7%) was worse and overall quality of life (55.8%) was no better or worse than before surgery. Outcome of lumbar fusion performed on injured workers was worse than reported in a published case series (126).

Fusion Heightens Blood Loss, Complications, Mortality

Patients undergoing fusion had a complication rate 1.9 times higher than those who had surgery without fusion. Rates were higher for blood transfusion (5.8 times), nursing home placement (2.2 times), and hospital charges (1.5 times) ($P < 0.0005$). Six-week mortality rate was 2.0 times higher for patients undergoing fusions (127).

Increased Intradiscal Pressure Above Fusion

Fusion increases intradiscal pressure above the fused level with a higher pressure noted with increases in both the number of levels involved in the simulated fusion and flexion motion itself. A greater increase was seen at the L4–L5 level than the L3–L4 level. This may explain the phenomenon of progressive degeneration of IVDs adjacent to a fused or fixed segment (128).

New Fusion Surgical Technique

Extraperitoneal anterior abdominal approach with a 4 to 5 cm incision on the midline at the umbilicus with an endoscope under video assistance allows disc resection and grafting for fusion. This is felt to lower morbidity and increase anterior fusion management for lumbar disc disease (129).

Lumbar Fusion: Helpful, Necessary, or Not?

Clinical outcomes for lumbar fusion did not differ by diagnosis or fusion technique, but were worse in studies with a greater number of previously operated patients (130).

Indications and contraindications for fusion include:

1. Absence of relief of sciatica after laminectomy is not an indication for fusion.
2. Significant remaining low back pain with a distinct morphologic explanation sometimes may be considered an indication for fusion.
3. Recurrent sciatica caused by fibrosus is not relieved by fusion.
4. Induced instability because of facetectomy responds well to fusion.
5. Fusion for unspecific remaining complaints after decompressive surgery, not explained by distinct morphologic findings, should be avoided (131).

Spinal Fusion for Relief of Low Back Pain

Follow-up discography of 62 chronically disabled low back pain patients treated with posterior interbody fusion showed 89% had satisfactory results, 93% returned to work, and 94% had a successful fusion. Concern has been raised that results of this study might lead to justification of fusion for low back (132).

Posterolateral intertransverse fusion can be used to successfully manage chronic discogenic back pain. However, patient selection remains a challenge, and successful outcome appears to be limited in the subset of patients receiving Worker's Compensation and those chronically disabled (133).

Fusion is not routinely required in patients undergoing repeat laminectomy and discectomy for recurrent disc herniation. In the absence of objective evidence of spinal instability, recurrent disc herniation can be adequately treated by repeat lumbar laminectomy and discectomy alone (134).

Orthosis Stabilization Produces Different Predictions of Fusion Success

Three weeks wearing a stabilizing orthosis to predict surgical success did not positively affect clinical outcome following fusion (135).

Another study of diagnostic external fixation was performed in 101 patients with disabling low back pain. In 47 patients, pain was relieved by stabilization but it returned after destabilization. These patients were considered good candidates for a fusion operation. Results after fusion are available for 34 patients: 14 (41%) patients had a good, 12 (35%) had a fair, and 8 (14%) had a bad result. Fifty-two patients did not respond positively to external fixation. Nine were operated on despite negative results with fixation. Of these, seven patients had a bad result, one a good result.

External skeletal fixation may be indicated for diagnostic purposes when all other diagnostic tools have failed to demonstrate the source of pain. In this difficult group of patients, external skeletal fixation helps to select the patients for a fusion operation with reasonable accuracy. No patient with inadequate response to external fixation should undergo spinal fusion (136).

Joint Spaces Adjacent to Fusion Sites Develop Spondylosis and Stenosis

Mobile spinal joints adjacent to fusion levels of the lumbar spine often develop problems that surface later and cause pain or require reoperation. Some of these problems include spinal stenosis, hypermobility, degenerative joint disease of disc or facets, and acquired spondylolysis or spondylolisthesis. The response of cartilage to the sudden increase in loading could be instrumental in the facet joint's biologic response (137).

Accuracy of Plain Radiographic Determination of Fusion

Plain radiographs are currently the most widely used modality for determining arthrodesis. When using static two-dimensional radiographs, the presence or absence of arthrodesis can be predicted in approximately two thirds of cases (138).

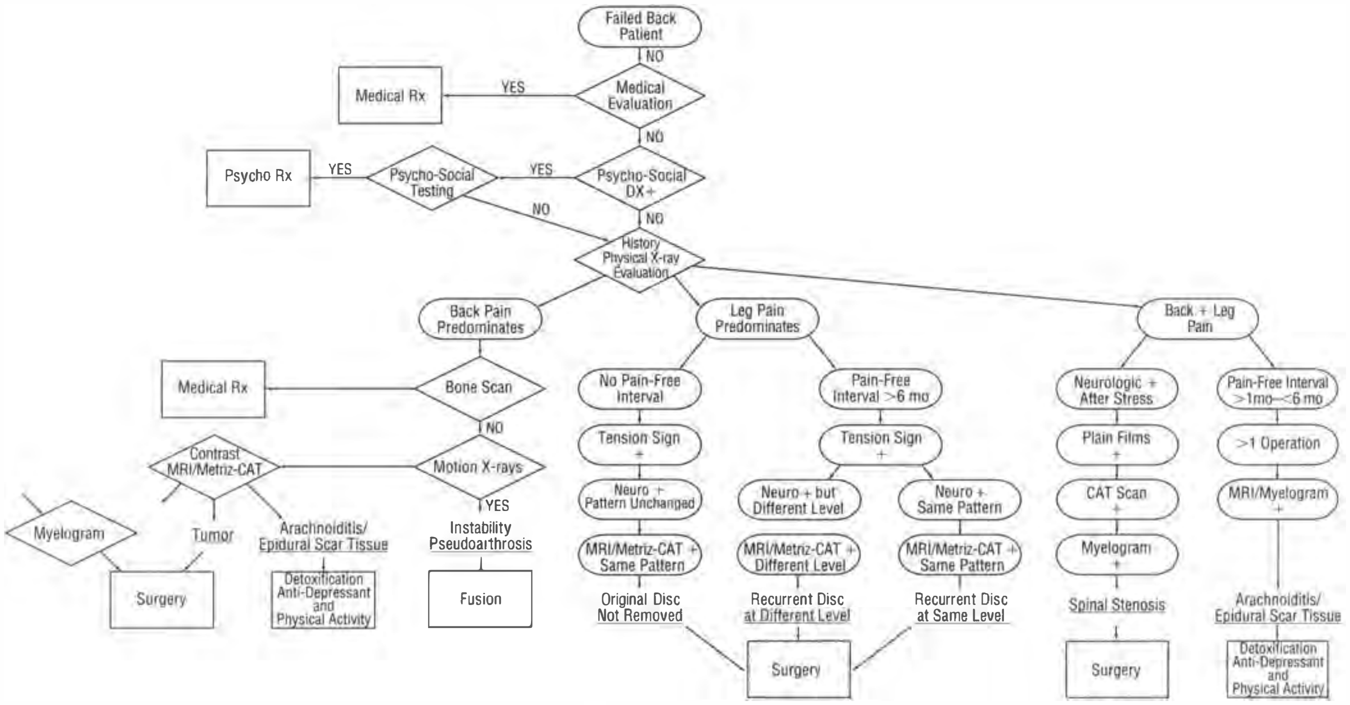
Failed Back Surgical Care—Spinal Implant Use and Benefit

Pain relief with spinal cord stimulation (SCS) in treating patients with failed back surgery syndrome (FBSS) is important for these unfortunate patients. Seventy-eight patients underwent trial stimulation. Thirty-five patients (55%) continued to experience at least 50% pain relief, 58 patients (90%) were able to reduce their medication, and 39 patients (61%) reported a change in lifestyle in that their ability to perform daily activities had improved significantly. Fifty-three patients (83%) continued to use their spinal stimulation device (139).

The greatest concentration of alpha and beta fibers lay in the dorsal columns and stimulation there would, according to the gate control theory, provoke inhibition of C fibers to afford relief (139).

Considering all patients selected for SCS (trial and definitive stimulation), the success rate has not been more than 41% at long term. Prevention of FBSS by more careful selection of patients for surgery would seem to be better than its treatment (139).

Approximately 50 to 60% of patients with failed back surgery syndrome report greater than 50% pain relief with the use of spinal cord stimulation (140).



Multiply operated back algorithm.

Figure 12.1. Multiply operated back algorithm. (Reprinted with permission from Wiesel SW, Boden SD. Diagnosis and management of cervical and lumbar disease. In: Weinstein JN, Rydevik BL, Sonntag VE, eds. Essentials of the Spine. New York: Raven Press, 1995:154–155.)

CARE OF THE MULTIPLY-OPERATED LUMBAR SPINE

Of the 300,000 new laminectomies performed each year in the United States, 15% will continue to have significant pain. A challenge in these failed back surgical cases is to determine the patient with recurrent disc herniation, spinal stenosis, or spinal instability. These are mechanical reasons for recurrent pain, as opposed to nonmechanical causes such as psychosocial instability, which will not be helped with further surgery. Figure 12.1 is an algorithm for decision-making when confronted with the multiply operated FBSS (141).

PRESENTATION OF FAILED BACK SURGICAL SYNDROMES

Recurrent L5 Dermatome Sciatica Post–L5–S1 Laminectomy Due to L4–L5 Far Lateral Herniation and Degenerative L5–S1 Stenosis

Case 1

A 46-year-old man was seen 3 years postsurgically for left L5 dermatome sciatic pain extending to the foot. Previous surgery was to remove a left L5–S1 herniated disc via laminectomy.

Figure 12.2 is the sagittal T1-weighted MRI study showing loss of signal intensity of the L1–L2 through L5–S1 discs. Note the anterior disc herniations at the L2–L3, L3–L4, and L4–L5 levels (*arrows*). Also note the type I degenerative pattern at the L4–L5 disc level, indicated by hyperintensity of the inferior L4 vertebral body plate and subchondral bone (*curved arrow*).

Figure 12.3 is a sagittal section of the left osseoligamentous canal showing the diminished vertical and horizontal diameter of the canal at the L5–S1 level (*curved arrow*). Hypertrophic degenerative changes of the facet joints at L5–S1 are seen (*arrow*), which narrow the upper third of the osseoligamentous canal housing the dorsal root ganglion.

Figure 12.4 shows the enhancement with Magnivest (gadopentetate dimeglumine) at the site of the previous laminectomy (*arrow*).

Figure 12.5 shows the facet hypertrophy of the left L5–S1 facet articulation on axial image (*curved arrow*). This creates stenosis of the osseoligamentous canal (*straight arrow*).

Figure 12.6 illustrates a left far lateral L4–L5 disc herniation (*arrow*), which could compress the L4 dorsal root ganglion and nerve.

Figures 12.7 and 12.8 are the anteroposterior and lateral projections of the lumbar spine and pelvis. Note the extensive disc and facet joint degenerative changes throughout the lumbar spine, especially L4–L5 and L5–S1. This allows excellent correlation with the above cited MRI findings.

The clinical impression of this case was that this patient may have recurrent left lower extremity fifth lumbar dermatome pain caused either by degenerative stenosis or perhaps to the L4–L5 left far lateral disc herniation, which would cause a different dermatome pain distribution, namely L4. This patient sought further surgical care after 2 weeks of distraction adjustments did not yield relief.

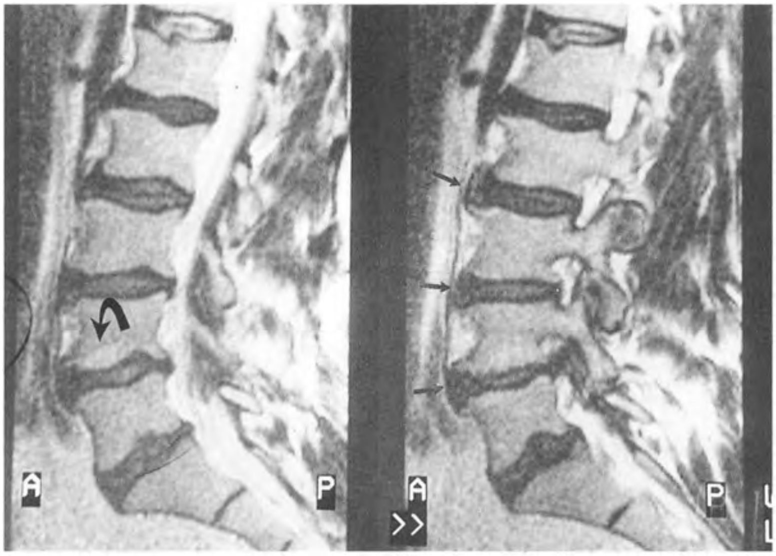


Figure 12.2. Loss of signal intensity is seen from L1–L2 through the L5–S1 disc levels. Note the anterior disc herniations at the L2 through L4 disc spaces (*arrows*). Type I degeneration of the L4–L5 disc space and vertebral plate and bone is shown at the *curved arrow* as an area of hyperintensity of bone.

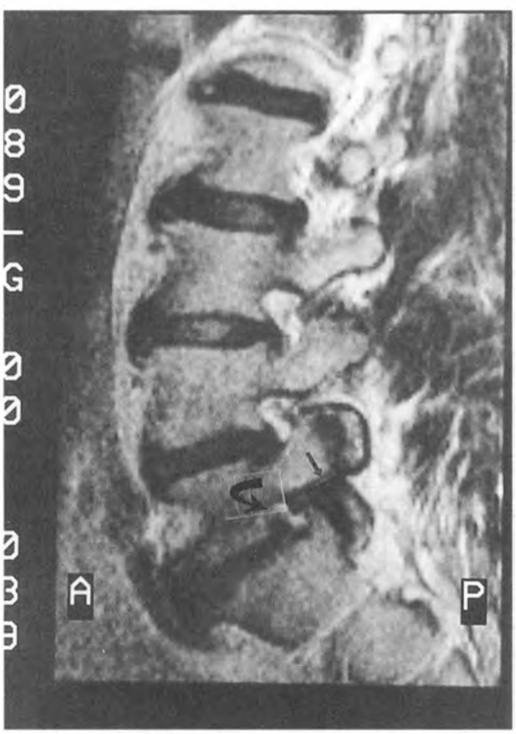


Figure 12.3. Note the diminished vertical and horizontal diameter of the L5–S1 left osseoligamentous canal (*curved arrow*) and the arthrosis of the facet joints (*straight arrow*).

Figure 12.4. The *arrow* points to the uptake of contrast medium at the site of the previous laminectomy.

Figure 12.5. The *curved arrow* points out facet arthrosis which creates foraminal stenosis (*straight arrow*).

Figure 12.6. The *arrow* points out the far lateral disc herniation. This patient had no leg pain, only low back pain.

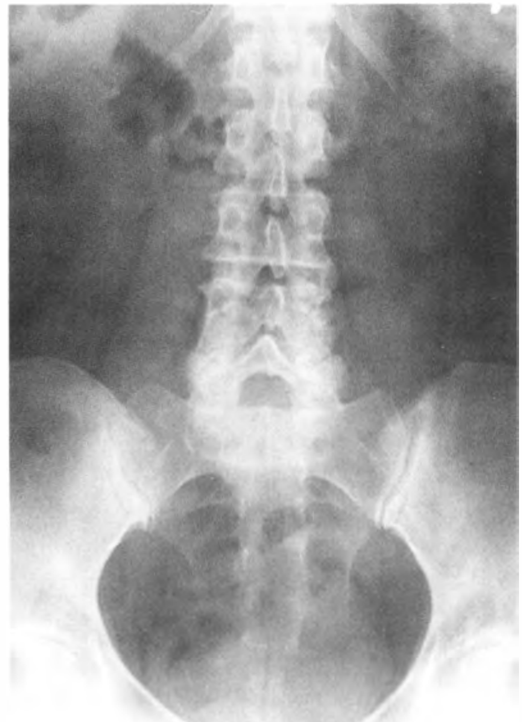


Figure 12.7. The anteroposterior lumbar spine study shows degenerative changes of the L4–L5 and L5–S1 facets with facet lamina syndrome.

Figure 12.8. The degenerative changes of the three lower discs are seen here, most especially at the L4–L5 level.

Pedicle Screw Removal for Probable Contact Irritation of the Nerve Root

Case 2

A 36-year-old man complained of right burning lumbosacral pain radiating into the right sacroiliac and buttock and down the posterior right leg into the foot as a pins and needles feeling.

This patient had previous decompression surgery at L5–S1 in 1994 with bilateral intertransverse process fusions on the left extending from L4 through the sacrum and on the right from L5 through the sacrum. This patient also had previous surgery to place screws into the pedicles at L4–L5 and L5–S1; these pedicle screws were felt to be the cause of recurrent right leg pain and they were removed.

Figures 12.9 to 12.11 are MRI images of this patient's lumbar spine. Figure 12.9 is the sagittal T2-weighted image showing the fifth lumbar vertebral body to be anterior on the sacrum with exostosis of the posterior inferior L5 plate extending into the vertebral canal (*arrow*). Signal intensity loss is seen at the L4–L5 and L5–S1 discs with normal intensity at the L3–L4 and L2–L3 levels. The fifth lumbar spinous process is missing from previous decompression laminectomy. Posterior to the lumbosacral spine is noted hyperintensity within the vertebral canal (*curved arrow*), which could represent Gelfoam or fat placed at the time of surgery, or more remote inflammatory fluid or cerebrospinal fluid.

Figure 12.10 shows precontrast T1-weighted image on the left and postcontrast on the right. Increased uptake of contrast is seen within the right lateral vertebral canal surrounding and displacing the first sacral nerve root laterally (*straight arrow*). This tissue displaces the thecal sac to the left and posterior, which is granulation uptake by contrast in the postcontrast study. Also is seen dis-

Figure 12.9. This T2-weighted image shows minimal L5 anterolisthesis with posterior L5 end plate hypertrophy (*arrow*). Also note the hyperintensity at the *curved arrow* posterior to the first sacral segment, which could represent fat or Gelfoam from past surgical intervention.

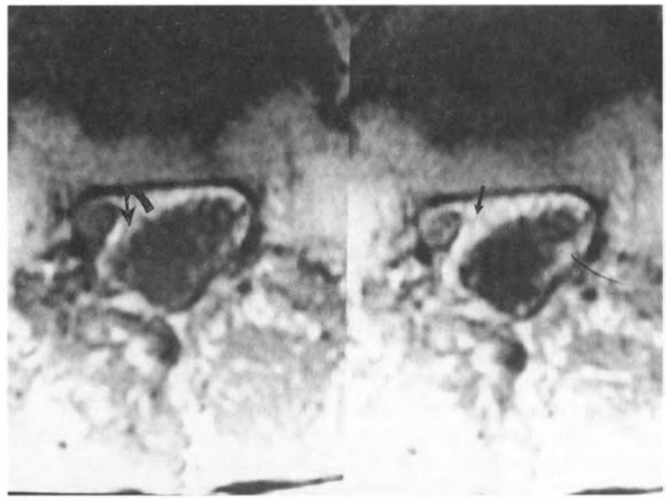


Figure 12.10. **Left** is a precontrast magnetic resonance image (MRI) showing cauda equina displacement by tissue mass (*curved arrow*); on the **right,** enhanced MRI shows uptake of contrast compatible with scar tissue presence (*arrow*).

Figure 12.11. At the *arrows* is shown the bone defect from previous pedicle screw placement.

placement of the thecal sac on the precontrast study by mixed areas of hyper and hypointense contrasting tissue (*curved arrow*).

Figure 12.11 is an axial T1-weighted image at the L4–L5 level revealing facet arthrosis (*curved arrow*). The *straight arrow* is the probable site of pedicle screw implant that was removed. It is noted that the remnant defect of the pedicle screw is parallel to the nerve root and, in fact, may have touched it and caused inflammation of the nerve.

Therefore, in this case, numerous persistent causes of continued low back and right lower extremity pain are possible. Scar tissue at the right L5–S1 level is seen to encompass the right S1 nerve root and to displace the thecal sac. Removal of the surgical pedicle screws and the approximate location of them to the exiting nerve roots raises the possibility that they were a cause of nerve root inflammation.

I only examined this patient, and did not treat him. I recommended a myelographically enhanced CT scan to rule out arachnoiditis and to detail nerve root compromise within the vertebral and osseoligamentous canals. Blood tests were also suggested to rule out possible inflammation of the subarachnoid space or meninges.

Pedicle Screw Fusion After Failed Surgery for Instability at L4-L5 in a Parkinson's Disease Patient

Case 3

This 59-year-old man with Parkinson's disease was seen in 1992 for thoracolumbar spine pain and anterior right and left thigh numbness and burning that began after a fall. Radiographic and CT study showed L4–L5 and L5–S1 advanced degenerative disc disease and spinal stenosis. Chiropractic distraction adjustments gave some relief, but with increasing falls, up to 10 times daily, the patient developed increasing bilateral thigh pain and also abdominal and groin pain.

In September, 1995 the patient underwent his first surgery with multilevel decompressive laminectomies at the L3–L4, L4–L5, and L5–S1 levels for stenosis. After surgery he was in severe right leg pain with loss of strength and difficulty walking. Significant leg weakness and numbness was reported. No bowel or bladder symptoms were noted. The pain increased on lying, sitting, standing, and walking, and he could not lift anything.

Examination Findings After First Surgery

The obvious Parkinson's disease symptoms were present. A well-healed scar from previous surgery was present. The patient could ambulate but was unsteady. Motor testing showed no left leg weakness, but the right leg demonstrated significant decrease in the right quadriceps and tibialis anterior muscles, 4 ± 5. No weakness of the extensor hallucis longus or gastrocnemius muscles was seen. Decreased sensation in the L4 distribution of the right leg was noted. The deep tendon reflexes at the knee were −1/−1; the ankle, −1/−1. Toes were downgoing. Otherwise, the examination was unremarkable.

Post–First Surgery Imaging After Decompression

Magnetic resonance imaging of the lumbar spine was performed 2 months postsurgery, which was too early to not mistake the usual postsurgical inflammatory changes for recurrent disc or scar tissue, but the patient's pain necessitated repeat MRI. The findings showed:

1. Figure 12.12 shows the plain lumbar x-ray study prior to decompression laminectomy and the postsurgical study showing L3, L4, and L5 level posterior spinal canal mixed signal intensity structure with a hypointense rim, predominantly hyperintense (*straight arrow*), which probably represents a combination of postsurgical changes including Gelfoam and postoperative edema. Retropulsion of the L4 vertebral body is noted (*open arrow*). A large posterior disc herniation at the L4–L5 level is seen (*curved arrow*). Flexion and extension studies also showed instability at that level.
2. Figure 12.13 shows diastasis of the right L4–L5 facet articulation with severe right L4–L5 foraminal stenosis (*open arrow*). The curved arrow shows the Gelfoam.
3. Sagittal MRI (Figure 12.14) shows a fracture of the pedicle of the L4 vertebra without displacement (*open arrow*).
4. Axial MRI (Figure 12.15) and CT (Figure 12.16) show the right L4 pedicle fracture as well as evidence of postoperative edema (see *arrow* for fracture).

The following diagnoses were made:

1. Postlaminectomy syndrome, multilevel, L3–L4 to L5–S1.
2. Lumbar instability, L4–L5 with retrolisthesis.
3. The instability with the L4 right pedicle fracture and retrolisthesis subluxation of L4 caused worsening spinal stenosis with probable impingement of the L4 nerve root on the right side.

Repeat decompression with partial facetectomies and fusion from L3–S1 with pedicle screw instrumentation and autogenous iliac crest bone graft was performed as a second surgery (Figs. 12.17 and 12.18). Postoperatively, the patient noted complete relief of his leg pain and was released on postoperative day 5. He wore a lumbosacral orthosis. Two years postoperatively he continues to have excellent relief of his leg pain and is active with signs of a solid fusion from L3–S1.

This case is presented with the cooperation of the orthopaedic surgeon on the case, Robert R. Shugart, MD, Fort Wayne, Indiana, who performed the second successful surgery.

Case 4

A 60-year-old man 2 years previously to being seen underwent intertransverse fusion from L4 to sacrum with repeat repair of pseudoarthrosis of the fusion. Postsurgical complaints were low back pain, left lateral thigh and anterior leg burning, left foot numbness, and right lateral and posterior thigh numbness extending to the toes and plantar foot area.

No cauda equina signs were present on examination. Loss of lumbar lordosis was noted with positive sitting SLRs producing leg pain. Range of motion was 30° of flexion, 10° extension, and 10° of bilateral lateral flexion of the thoracolumbar spine. Pain was noted over the sacroiliac joints and L2 to S1 segments.

Motor weakness of the right great toe flexion and left foot eversion were seen. Seen was 25 mm of atrophy of the left thigh compared with the right, with 55 mm loss of the left calf on mensuration. The deep tendon reflexes of the lower extremity were +2 bilaterally with hypesthesia of the L3 through S1 dermatomes noted. Vibratory sense was diminished at both great toes.

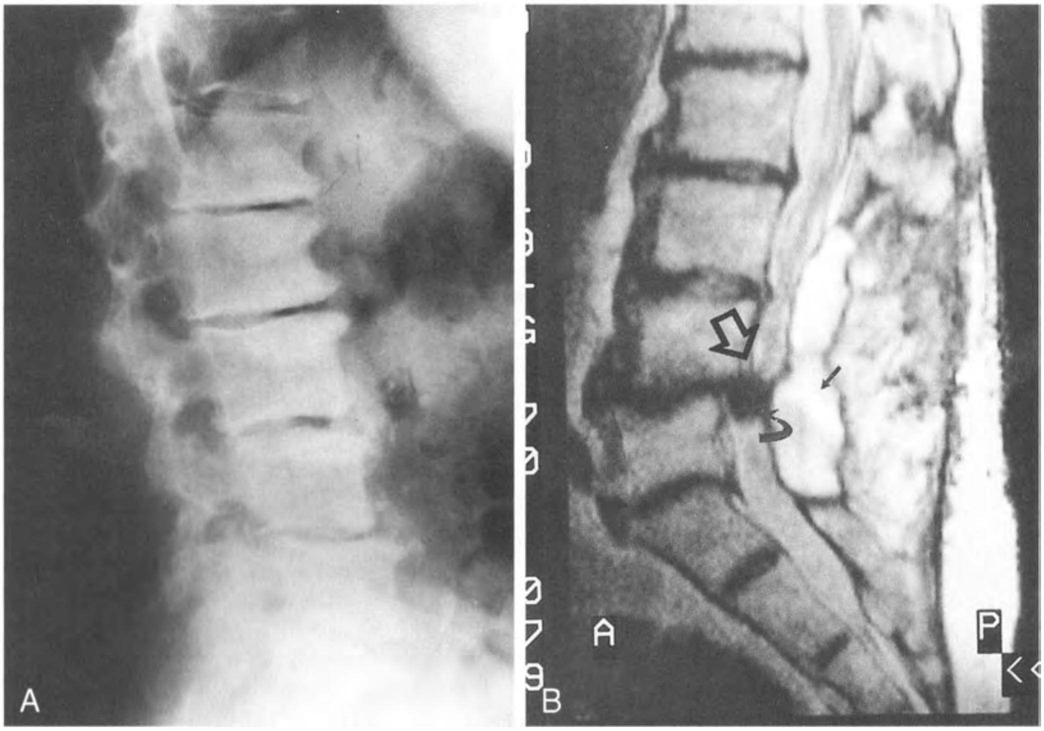


Figure 12.12. **A.** The plain lateral lumbar x-ray study before surgery. Note the vacuum changes within the discs and the marked advanced degenerative disc disease and retrolisthesis subluxation of L4 on L5. **B.** The first surgical postoperative magnetic resonance image showing the hyperintense area within the vertebral canal (*arrow*) representing Gelfoam from surgical placement. At the *open arrow* is shown the persistent posterior displacement of L4 on L5 with a disc bulge (*curve arrow*). This is instability of L4 on L5.

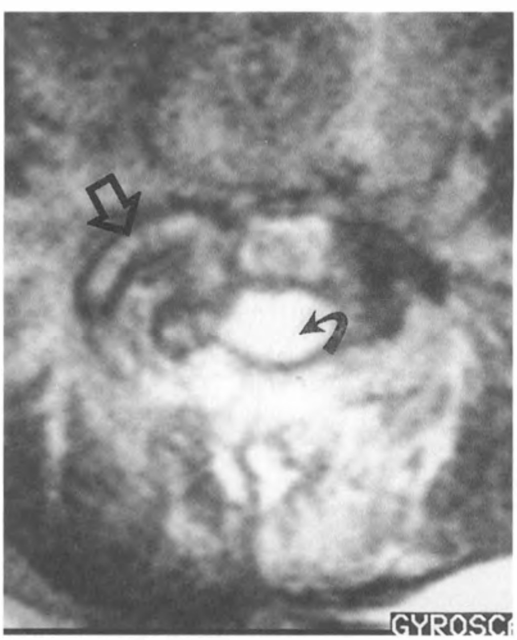


Figure 12.13. The Gelfoam defect (*curved arrow*) is seen within the vertebral canal, and the facet misalignment and diastasis (*open arrow*) of the L4–L5 facet joint are evident.

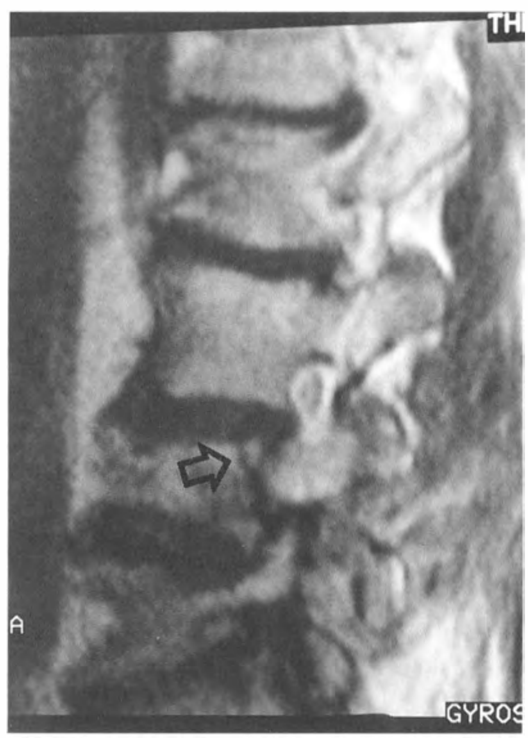


Figure 12.14. The pedicle fracture (*open arrow*) that occurred following the surgery.

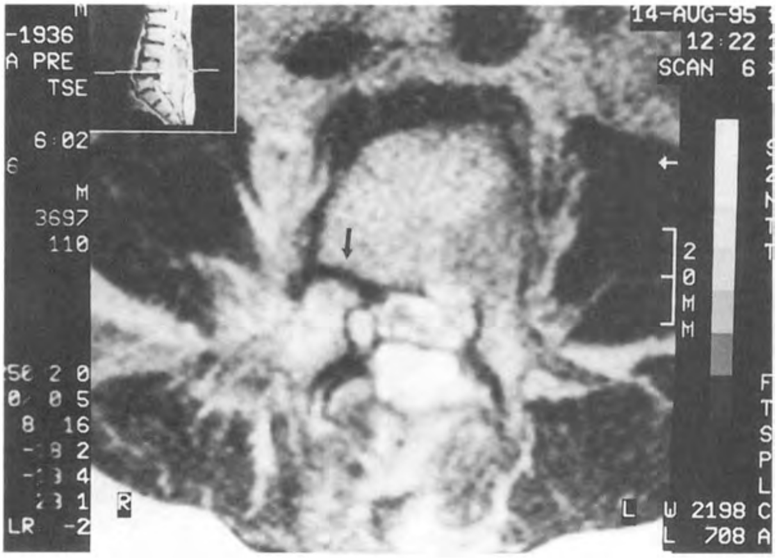


Figure 12.15. Axial magnetic resonance image shows the right L4 pedicle fracture (*arrow*) with postoperative edema.

Figure 12.16. Computed tomography image shows the right L4 pedicle fracture (*arrow*) with postoperative edema.

The hamstring muscles were tight bilaterally, at 30° on the left and 45° on the right side. No signs of hip disease were seen. Gluteal muscle strength was 5/5 bilaterally.

Figure 12.19 shows dextrorotatory scoliosis and the intertransverse fusion with laminectomy of the lumbar spine from the first surgery. Figure 12.20 shows an old healed L2 vertebral body fracture and degenerative disc disease throughout the lumbar spine, most marked at the L5–S1 level.

Figure 12.21 shows L5–S1 anterior and posterior disc protrusion with advanced degenerative change. Figure 12.22 shows the laminectomy (*arrowhead*) and the intertransverse fusion (*arrow*).

Figures 12.23 and 12.24 are myelograms showing generalized disc degeneration of the lumbar spine and in Figure 12.23 is shown amputation (*arrow*) of the right L5 nerve root and the opposite root is visualized. Figure 12.25 shows the intertransverse fusion (*arrows*) with the laminectomy (*arrowhead*). The cauda equina is not compromised. Figure 12.26 at the L4–L5 level shows facet arthrosis (*arrowheads*) and a generalized disc bulge into the central and osseoligamentous canals (*arrow*).

Conclusion in this case was that a right L5 nerve root amputation occurred at the L4–L5 level by disc protrusion, within the surgically fused spine. The L4–L5 disc was protruding into the spinal and osseoligamentous canals, but the cauda equina was not compressed.

Conservative care involved distraction adjustments at the L3–L4 level and above, trigger point therapy to the lumbar spine paravertebral levels, lumbar spine massage, electrical stimulation consisting of positive galvanism at the L4–L5 level on the right followed by tetanizing currents to sedate muscle irritation, home exercises, and low back school training, which gave this patient

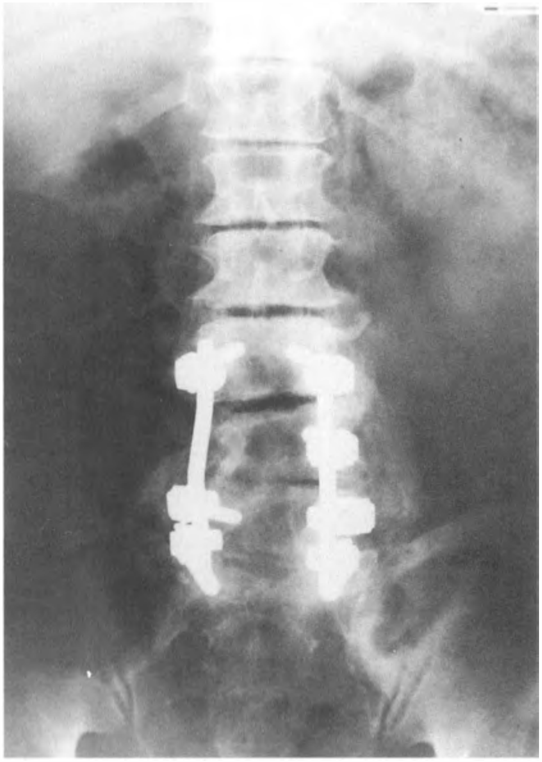


Figure 12.17. Anteroposterior lumbar spine radiograph showing the pedicle screw instrumentation following repeat surgery.

Figure 12.19. L5 laminectomy (*arrow*) with dextroscoliosis and L4 to S1 intertransverse fusion is seen.

Figure 12.18. Lateral lumbar spine radiograph showing the pedicle screw instrumentation following repeat surgery.

Figure 12.20. A healed compression fracture of L2 is noted (*arrow*), as well as extensive L5–S1 degenerative disc changes with discogenic spondylosis throughout the lumbar spine.

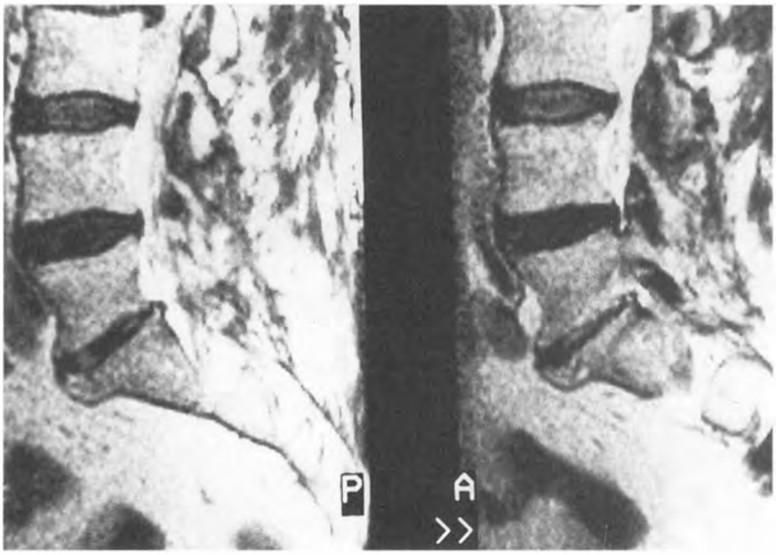


Figure 12.21. Extensive L5–S1 degenerative disc disease is noted with both anterior and posterior disc herniation.

Figure 12.22. Axial L5–S1 section shows the laminectomy (*arrowhead*) and the intertransverse process fusion (*arrow*). Note that the cauda equina is spared compression.

Figure 12.23. Myelography is performed showing a dextroscoliosis of the lumbar spine with the intertransverse fusion (arrowhead). Of special interest is the amputation of the right L5 nerve root (*arrow*); the opposite root is visualized with dye.

Figure 12.25. Myelographic-enhanced computed tomography scan shows the laminectomy (*arrowhead*) and the fusion (*arrows*). Note that the space available for the cauda equina is adequate with no thecal sac compression.

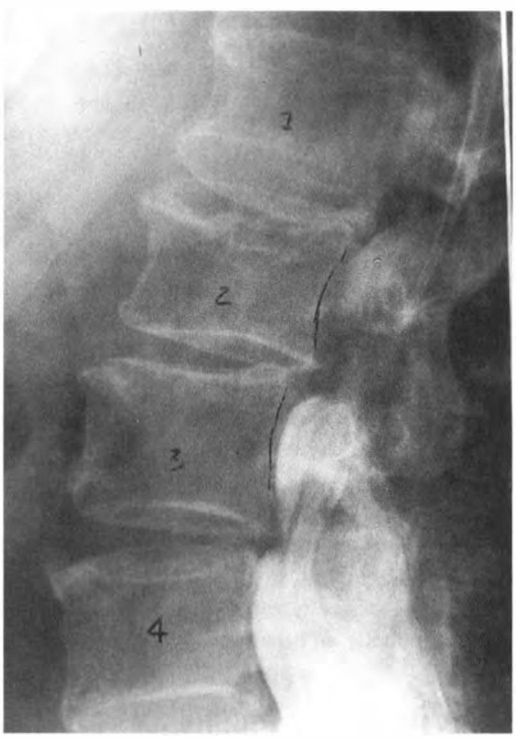


Figure 12.24. Note the posterior protrusions of the L3–L4 and L4–L5 discs into the anterior thecal dye-filled sac.

Figure 12.26. At the L4–L5 level, generalized disc bulging is noted (*arrow*), which narrows the vertebral canal and lateral recesses. Facet arthrosis is seen bilaterally (*arrowheads*), which does narrow the canal diameter.

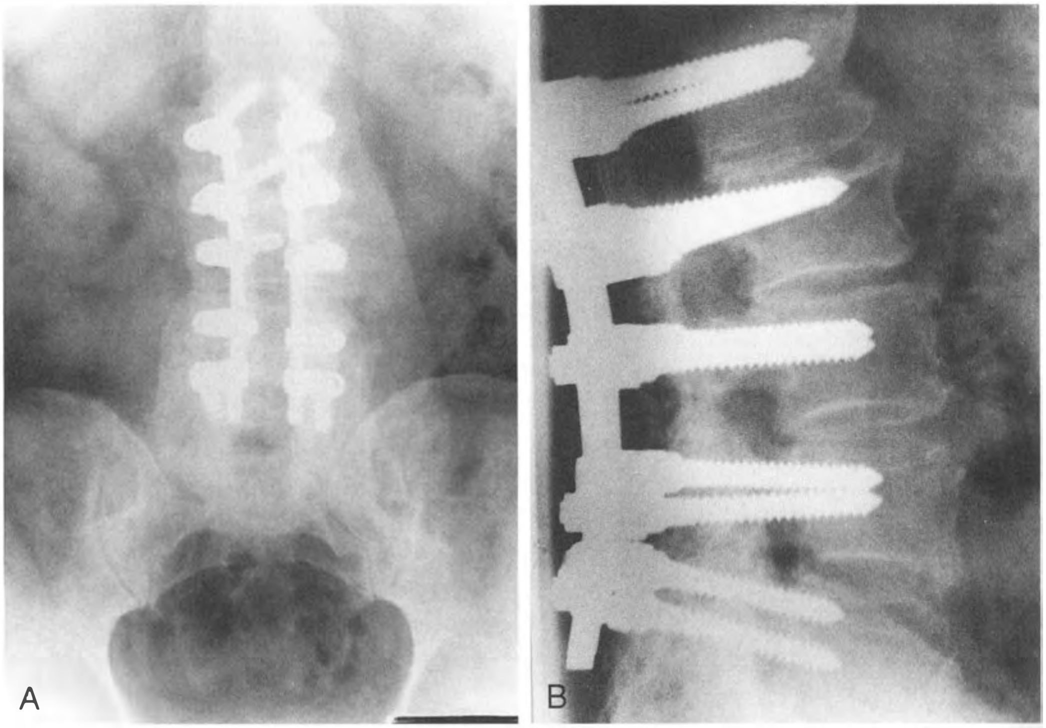


Figure 12.27. Anteroposterior and lateral lumbar spine radiographs showing the instrumented fusion performed.

about 50% relief of symptoms. This was a medicolegal case and the patient sought another decompression surgery with fusion, which gave him 60% relief of lower extremity symptoms and 10% relief of low back pain. The foot numbness was not relieved and he wears a transcutaneous electrical stimulation (TENS) unit to relieve the low back pain. Figure 12.27 shows the anteroposterior and lateral radiographs with the instrumented fusion.

OTHER LESS USED FORMS OF TREATMENT FOR LOW BACK PAIN

Intravenous Immunoglobulin Therapy for Nerve Root Pain

Lumbosacral plexopathy responds favorably to high-dose intravenous immunoglobulin therapy (0.8 and 0.4 g/kg), and represents a clinically and possibly pathogenetically distinct and treatable subgroup of patients (142).

Sclerotherapy

Sclerotherapy, also known as prolotherapy, identifies the anticipated function of an injection of glycerine (25%), dextrose (25%), phenol (2.4%), and water with an equal volume of local anesthetic to create a proliferation of fibrous repair tissue (143).

Arthroscopic Discectomy

Arthroscopic discectomy, a promising new procedure, is totally unproved at this time (144).

Four Preventive Treatment Regimens for Low Back Pain

The effectiveness of four strategies to prevent low back pain for asymptomatic individuals were compared (145):

1. Back and aerobic exercises
2. Education
3. Mechanical supports (corsets)
4. Risk factor modification

Limited evidence indicates that exercise aimed at strengthening back or abdominal muscles and exercise aimed at improving overall fitness can decrease the risk of subsequent low back pain, but the effect is modest and of unknown duration. Insufficient evidence is found to recommend that either back education programs or mechanical supports be used routinely to prevent back pain. These conclusions should be generalized cautiously because they are based primarily on studies conducted in the workplace and not in the clinical setting. Although no evidence shows that smoking cessation, weight loss, or attention to psychological risk factors can prevent the development of low back pain, recommendations to address these factors might be made on other grounds (146).

Artificial Disc Replacement

Artificial disc replacement was performed in six patients, average age of 55 years and average follow-up of 3.4 years. Four of

the six patients had juxtafusion degeneration, one had multilevel disc degeneration, and one patient had isolated disc resorption. The Acroflex disc (Acromed Corp, Cleveland, OH), which was used in the replacement, is composed of a rubber core vulcanized to two titanium end plates. Satisfactory results occurred in four of six patients. Poor results occurred in the presence of deformity that resulted in prosthetic failure and isolated disc resorption (146).

Disc replacement is probably not indicated in every disc-related problem or for every spinal patient. The most common diagnoses for artificial disc replacement include disc degeneration and postnucleotomy syndrome, two common diagnoses in the middle-aged population. Advanced and debilitating disc disease is difficult to treat surgically in the active middle-aged population because currently no methods are available for maintaining motion segment function and thus prolonging the immobility that is often associated with more advanced disease or aging. Artificial disc replacement also represents an addition to the surgeon's armamentarium when considering problems associated with failed back syndrome. In patients with nonresolving symptoms after a discectomy procedure, artificial disc replacement could be an alternative. It can be inferred that because the potential exists for dislocation or gross migration of the prosthesis, spondylolisthesis should be a contraindication. Care should be taken to avoid patients with metabolic bone diseases such as osteoporosis and osteomalacia (147).

Patients being treated by disc replacement actually achieve good symptom relief with respect to back and leg pain. Also, parameters such as the ability to walk and the straight leg raising sign improve. Patients who underwent total disc replacement experienced few complications, and they did not experience the comorbidity factors that have been previously enumerated. Routine or indiscriminate use of artificial disc replacements is not warranted and further investigation into the utility of spinal arthroplasty is needed (147).

Artificial disc implants in 46 patients produced excellent clinical results in 24% of patients, good in 39%, and fair or poor in 27%. Clinical results were satisfactory in 69% of patients who had a disc prosthesis at one level and 40% of those operated at two levels. None of the artificial discs dislocated or loosened (148).

A lumbar disc prosthesis made of silicone rubber was found to be biomechanically applicable for human use and to restore function and improve the curative results of disc excision (149).

A polyurethane fiber reinforced disc prostheses that demonstrated properties similar to those of natural discs produced good outcome (150). Manufactured synthetic disc prostheses that matched the mechanical behavior of a natural disc have been produced (151).

Disc Transfer

Lumbar disc transplantation was studied in eight mature mongrel dogs in which the L2–L3 and L4–L5 intervertebral discs, with a small segment of adjacent superior and inferior vertebral body, were removed and transposed. The structure and function of autograft IVDs were maintained after disc transfer surgery; the transplant discs, however, were not completely normal in either their morphology or their metabolic functioning (152).

Cadaver Transplants of Damaged Discs

Bone bank organizations have been encouraging research on the use of cadaver transplants for replacements of damaged IVDs. The research is currently at the animal stage, and is years away from human investigation (153).

Growth Factors in Repair of Anulus Fibrosus Damage

Two growth factors to combat disc dehydration, transforming growth factor-β and basic fibroblast growth factor play a role in the regulation of cartilage and bone formation, and they have been used in several animal investigations of anulus fibrosus repair with interesting results (153).

Preventing Nerve Compression by Disc Herniation

A tiny "cage" made of carbon fiber mesh and packed with soft bone matter is placed between spinal discs to prevent them from pinching a nerve. The cage is ten times stronger than bone and fuses to the existing discs better than hard bone. The operation could be beneficial to people suffering from failed back surgery, degenerative disc disease, osteoporosis, and disc slippage (154).

EPIDURAL STEROID INJECTIONS

Mixed opinions on the beneficial role of epidural steroid injections exist. Let us look at some differing opinions, because currently this procedure is a clinician's decision and chiropractic physicians are involved in caring for patients who have or will undergo the procedure.

Positive Role

Favorable outcomes from some controlled and many uncontrolled studies suggest that epidural steroid injections ease lumbar radicular pain caused by common structural abnormalities, such as lumbar disc herniation and spinal stenosis. Current knowledge of enzymatic and neurochemical mediation of pain and inflammation supports the use of steroids in managing noncompressive lumbar radicular pain and possibly lumbosacral pain (155). One study showed that at 3 months, 83% of patients were satisfied with the clinical outcome (156). Lumbar epidural injection or periradicular infiltration at the appropriate level, confirmed under image intensifier, was the next step before considering surgical decompression in 154 patients with sciatica. Twenty-three patients (14%) underwent surgical de-

compression. All conservatively managed patients made a satisfactory clinical recovery: average reduction of pain on the visual analog scale was 94% (range, 45–100); 64 of the 84 (76%) disc herniations and 7 of the 27 (26%) disc bulges showed partial or complete resolution (157).

Epidural Steroid Action

Epidural local anesthetics produce a differential inhibition of somatosensory and motor functions, where perception of temperature and pain is more easily blocked than perception of tactile stimuli and motor function. A block of three consecutive nodes of Ranvier, or partial blockade of more nodes, is necessary for conduction block of myelinated fibers.

Epidural bupivacaine (BUP) use, alone or with morphine, induced differential blockade of both nonpainful and painful somatosensory functions, and did not result in inhibition of motor function (158).

Patients with true cervical radiculopathy showed a 62% probability of obtaining 50% pain relief and at least a partial return to normal activities after epidural steroid injection (159).

Thirty-five patients with sciatic nerve compression receiving epidural steroid injection showed 85% received some improvement at 1 week, and 43% had improvement lasting 3 months. At 3 months, 83% were still satisfied with the treatment (160).

Intramuscular Steroid Is Superior to Epidural Steroid Injection

Few studies have demonstrated superiority of epidural steroid injections to placebo. The efficacy of intramuscular and epidural steroids for treating 31 patients with chronic low back pain were compared. Epidural injections of triamcinolone acetonide (1 mL) and intramuscular injection with saline and epidural injection with saline and intramuscular injection with 1 mL of triamcinolone acetonide were studied. Subjective and objective tests showed superiority of the intramuscular steroid over epidural steroid injection for treating chronic back pain caused by disc degeneration. The only instance of superiority of epidural steroid injection over intramuscular injection was spine mobility, where epidural injection was superior (161).

Placebo Contribution to Pain Relief

Surgery is the most potent placebo effect that can be exercised in medicine. A double-blind trial with a sham operation on 19 patients with angina in contrast with those treated with ligation of the internal mammary artery showed no difference between the two groups, most of whom showed a marked improvement of their angina and exercise tolerance and some of whom improved the shape of their electrocardiograms. The effect of ultrasound on the pain, trismus, and swelling that can follow wisdom tooth extraction was tested with use of the machine turned on and off. No difference in reduction of swelling or pain relief in the two instances was noted (156).

Three reasons for the placebo effect are (*a*) the effect is produced by a decrease in anxiety; (*b*) expectation leads to a cognitive readjustment of appropriate behavior; and (*c*) it is a classical Pavlovian response.

Intraoperative Epidural Corticosteroid Use—Helpful or Not?

Intraoperative epidural corticosteroids after microsurgical lumbar discectomy for unilateral disc herniation did not lessen postoperative morbidity or improve functional recovery (162). Intraoperative infiltration reduces postoperative pain and morphine requirements. It is a quick, simple, safe, and effective means of improving the patient's comfort (163).

Epidural Steroid Injection Is Not Effective

The National Health and Medical Research Council of Australia does not endorse epidural steroid injection use, stating it is essentially an unproved procedure that has potential risks. Desperate patients in pain and physicians eager to try something that might work is not a good clinical recommendation (164).

Although epidural corticosteroid injections remain a relatively safe treatment modality, their efficacy remains to be tested in a properly controlled prospective randomized, double-blinded clinical trial with adequate numbers (160). The efficacy of epidural steroid injections has not yet been established. The benefits of epidural steroid injections, if any, seem to be of short duration only (165).

Negative Opinion on Epidural Steroid Injection for Prolapsed Disc Lesions

Epidural injection of steroids was given to 16 patients less than 50 years of age with no evidence of degenerative changes on standard plain radiographs (e.g., no disc space narrowing, osteophyte formations, facet joint hypertrophy, and so forth). Other patient characteristics included an attack of low back pain with sciatica with signs of nerve root entrapment, no significant past history of disc disease, intractable predominantly sciatic pain despite at least 3 weeks of conservative treatment with bed rest and analgesia, and clear evidence of a prolapsed IVD with root impingement on either radiculography or CT. Ten had temporary relief of pain but all 16 ended in surgical care with prolapsed discs.

The results of this study cast doubt on lumbar epidural injection of steroids to treat patients with acute proved prolapsed IVDs in the absence of degenerative spinal disease who persist with severe sciatic pain despite a trial of bed rest (166).

Epidural Steroid Injections to Differentiate Physical (Organic) Pain from Psychogenic (Nonorganic) Pain

Epidural injection of steroids were given to 100 consecutive patients with low back and leg pain first with 10 mL of saline.

If this reduced the patient's pain, the procedure was terminated. If it did not, 6 to 10 mL of lignocaine was injected as far proximally as the T12–L1 level. Greater than 60% subjective relief on visual analogue scale (VAS) represented a positive result.

Fifty-one patients had a positive result as did 19 of those injected with saline solution (the placebo effect). Epidural injections are not a reliable complementary test to standard radiologic practices in the investigation of lumbar spine disorders. The placebo response can be from 35 to 90% positive, making clinical evaluation impossible (167).

In another study comparing lignocaine with saline epidural injection, the placebo response accounted for 35% of patient improvement. In some cases, higher responses of 70 and 90% have been recorded. Again, findings were that differential epidural injections with saline and lignocaine is not a reliable complementary test to standard radiologic techniques in the investigation of lumbar spine disorders (168). Intradiscal steroid injections had no statistically significant benefit in another study (168).

Predicting Surgical Outcome with Temporary Relief Following Nerve Root Injection

A steroid injected into the patient's symptomatic nerve root should provide temporary pain relief if the patient is expecting a favorable surgical outcome. Prolonged structural compromise of spinal nerve roots can lead to chronic changes that surgical decompression might not be able to reverse (169).

Complications of Lumbar Epidural Anesthesia and Analgesia

Lumbosacral radiculopathy, polyradiculopathy, or myelopathy have developed during epidural anesthesia or analgesia. The local concentrations and time of exposure of the medications are high and the following factors act to increase the risk of neurologic complications:

1. Lumbar stenosis is probably a significant risk factor.
2. Inadvertent subarachnoid administration.
3. Only medications deemed safe for intrathecal injection should be used during the combination of general and epidural anesthesia.
4. Older patients undergoing long surgeries may be at increased risk for neurologic complications (170).

RETURN TO WORK FACTORS FOLLOWING LOW BACK INJURY

Younger age and early referral for rehabilitation, but not the severity of the injury, were associated with greater likelihood of return to work (172). Eight percent of workers with acute low back pain returned to work within 6 weeks, whereas the 10 to 20% who did not recover within 3 months contributed 80% of the cost of work-related back injuries (173–176).

Return to Work Following 1-Year Hiatus

After 6 months of disability, patients have a 50% likelihood of successful rehabilitation; at 1 year this figure reduces to 20%, and at 2 years the chances of successful rehabilitation are virtually nil (177). One year after seeking care, 82% of 1128 low back pain patients reported having back pain in the previous month (178).

How Soon Do Disabled Chronic Low Back Pain Patients Return to Work?

Although most low back pain patients recover within 2 months, 2 to 3% eventually develop disabling chronic low back pain (179). Fifty-five patients, average age 37 years old, referred by occupational physicians were evaluated and followed successfully for at least 6 months. Overall, 12.7% of the patients returned to work within 1 month of injury, 40% returned within 2 months, 54.5% within 3 months, 69% within 4 months, 74.5% within 5 months, 76.3% within 6 months, 80% within 7 months, and 83.6% after 7 months. Approximately 16% never successfully returned to work within the follow-up period. Married patients returned to work more quickly than single patients. Predicting which patients presenting with acute low back troubles are likely to become chronic cases, the optimal prediction equation would appear to be a perception that low back trouble is work-related plus absence from work for more than 2 weeks, which equals a high-risk case (179).

Compensation

Workers (1191) with low-back pain who were injured on the job were compared with 389 workers who were injured away from work on variables of disability time and pain intensity. Injury on the job is associated with prolonged disability time, irrespective of the type of job performed (180).

Patients receiving Workers' Compensation benefits report significantly greater levels of pain, disability, and psychological distress than do those not receiving benefits, irrespective of diagnosis. Patients suffering from myofascial pain were significantly less likely to report periods of pain relief than patients with herniated disc syndrome. Patients with myofascial pain and Workers' Compensation benefits demonstrated the highest levels of somatization and phobia. These findings suggest that the effects of low back pain of myofascial origin have comparable, if not worse, consequences than disc herniation. These findings also reaffirm the importance of Workers' Compensation benefits in understanding the differences in patients with chronic low back pain (181). Worker's Compensation patients are 1.37 times more likely to undergo surgery involving fusion and almost twice as likely to have a subsequent reoperation within 3 years of the index surgery (182).

Legal Awards Are Based on Word Selection of Disc Problem

In disc problem cases where the "H" word [herniation] was used, the average dollar amount of the legal award was $82,000. Where the "P" word [protrusion] was used to describe a similar degenerative abnormality, the average value of the award was $30,000. When "disc bulge" or "normal MRI" were used to describe an abnormality, the average claim was $12,000 (183).

Approximately 2% of all workers injure their backs annually, resulting in $16 billion in direct costs; but incredibly, 10% of the injuries account for 80% of the costs. Only 50% of individuals with low back pain disability have any objective findings (184).

Insurance does influence the use of medical care for musculoskeletal conditions that require increased expenditures (consulting a doctor, using medication, or physical therapy), but it does not affect aspects of care that do not require additional expenditure (type of physician, recommendation of unsupervised exercise) (185).

CHIROPRACTIC CARE IS EFFECTIVE, LESS COSTLY, SAFER, AND ACCEPTED

Chiropractic care is more effective, less costly, safer, and results in greater patient satisfaction than other types of medical care. Furthermore, greater patient satisfaction is found with chiropractic than medical management of low back pain. For these reasons, the following recommendations are made: governments should encourage the greater use of chiropractic services for low back pain with full public insurance of chiropractic services. Chiropractors should be employed in hospitals and hospital privileges should be extended to them. Chiropractors should be further involved in Workers' Compensation boards. Chiropractic education should be integrated with a university. Finally, cooperation between chiropractors, physicians, and other providers should be encouraged (186).

Chiropractic Care Costs 50% that of Medical Allopathic Care

A 2-year retrospective comparison of medical and chiropractic care for 8928 low back pain patients in the private fee-for-service sector was made. Total insurance payments were substantially greater for medically initiated episodes, especially for episodes of care lasting longer than 1 day. For the medically initiated category, inpatient payments were nearly seven times as great for the medically initiated cases and their outpatient payments were nearly 50% higher. The wide disparity in costs suggests that the role of chiropractic deserves careful consideration in strategies adopted by employers and third-party payers to control health care spending (187). The high-cost dilemma is further complicated by claims from the medical profession itself that about 90% of the 250,000 back surgeries performed each year can be avoided.

In addition to relative costs and outcomes, the role of the chiropractic doctor as a primary point of contact for episodes of lumbar and low back neuromuscular disorders needs careful consideration. The growing importance that patients attach to nonmedical treatment, particularly to chiropractic therapies, adds urgency to these tasks (187).

A comparison of the health care costs of patients who received chiropractic treatment in insurance plans that do not restrict chiropractic or medical benefits with those treated solely by medical and osteopathic physicians showed that patients receiving chiropractic care experienced significantly lower total health care costs ($291 to $1722) as represented by adjusted third-party payments in the fee-for-service sector (188).

Chiropractic Care Is of Renewed Interest in Medicine

Until recently, organized medicine has vigorously debunked chiropractic as an "unscientific cult." Now held more discreet in its criticism by the resolution of the Wilk antitrust suit, which alleged that organized medicine has improperly restrained the practice of chiropractic, allopathic medicine is reevaluating the role of spinal manipulation in the treatment of patients with spinal pain. Yearly, 1 in 20 Americans visit a chiropractor, whose numbers have grown from 32,000 in the 1970s to 45,000 in 1990. Such reputable medical practitioners, such as Cyriax, Mennel, Maigne, and Greenman, as well as other members of the North American Academy of Manipulative Medicine, have also taken the lead in advocating the use of spinal manipulation in the conservative treatment of low back pain (189).

As long as political and economic factors continue to color scientific judgement with respect to the efficacy of manipulation, a literature review of this subject will continue to defy objectivity. Meanwhile, let the manipulist always be prudent and correct in diagnosis and, as is always true in our consumer-driven economy, "caveat emptor," let the buyer beware (189).

The chiropractic profession is involved in treating a significant number of postsurgical back patients. The prevalence of these types of cases in the primary care chiropractic practice was found to be above the anticipated level in the general public (190).

Duodenal Ulcer Treated with Spinal Manipulative Therapy

The outcome of 40 cases of uncomplicated ulcerous disease treated by usual therapeutic methods were analyzed and compared with spinal manipulative therapy (SMT) treated patients. In patients with an almost identical initial condition status, the use of SMT resulted in clinical remission on an average of 10 days earlier than traditional care. It must be also noted that patients in the SMT treated group did not report having any pain after from 1 to 9 (average 3.8) days of therapy. SMT resulted in clinical remission with full epithelialization of ulcerous defects or with cicatrization (191). I insert this study as it emphasizes the chiropractic paradigm in the treatment of diseases other than neuromusculoskeletal problems—a concept that often strains relations between chiropractic and allopathic medicine.

Chiropractic Value

Expenditures on health care in America now exceed $800 billion annually. This is around 13% of the national output, which is roughly double the share that health care occupied two decades ago (192).

> "Spinal manipulation applied by chiropractors is shown to be more effective than alternative treatments for low back pain.
>
> "There is no clinical or case-control study that demonstrates or even implies that chiropractic spinal manipulation as unsafe in the treatment of low back pain" (187).

> "Chiropractic management of low back pain is more cost-effective than medical management.
>
> "Chiropractic services should be fully integrated into the health care system.
>
> "A very good case can be made for making chiropractors the gatekeepers for management of low back pain in the workers' compensation system in Ontario" (192).

A blinded randomized clinical trial comparing the effectiveness of manual therapy, physiotherapy, placebo (detuned ultrasound and detuned short wave diathermy), and a general practitioner (GP) for 256 patients with chronic nonspecific back and neck complaints had physical outcome measures (spinal mobility and physical functioning) presented for 3, 6, and 12 week follow-ups. For example, respectively, 36% in the GP group, 43% in the placebo group, 54% in the physiotherapy group, and 62% in the manual therapy group had an improvement score of three points or more.

The manual therapy group showed the best outcome for patients with improvement scores of less than six points, whereas the GP group showed the least improvement. The cumulative distributions of physical therapy and placebo therapy were in between (193, 194).

Controlled clinical trials comparing manipulations to placebo or more conservative treatments in low back pain have produced variable (32 to 92% experiencing relief) and generally short-lived results. Most studies have demonstrated improved outcome with manipulation when compared with other treatment modalities for low back pain. However, the most efficacious treatment appears to be a protocol combining manual therapy and other therapies (195). Acute severe low back pain is helped by chiropractic manipulation within the first 4 weeks (196).

Disc Size Change Under Conservative Care

Larger disc herniations show the greatest reduction in size under successful conservative care, whereas patients not responding to conservative care have the most herniations that do not reduce. Central disc herniations show a lower incidence of diminution (197).

Migrating free nuclear fragments resorb or disappear, and the mechanism for this may be exposure to the vascular supply (198). Neovascularization at the periphery of 30 sequestrated discs with no fibrous scar formation was observed, suggesting that a kind of "absorption" process occurs predominantly in the healing stage (199).

Chiropractic and McKenzie Treatment Equally Effective

Reportedly, McKenzie assessments have been questioned (200). Five hundred and six patients were invited into a study and randomized to treatment by a McKenzie therapist, a chiropractor, or a control treatment consisting of an educational pamphlet.

Four experienced chiropractors delivered the chiropractic care. The manipulations involved side-posture, high-velocity thrusts. Chiropractors also were allowed to provide up to nine visits over the month, with the exact number up to the chiropractor.

The McKenzie therapists saw their patients for an average of 4.6 visits over 1 month, whereas the chiropractic patients, on average, had two visits more each. In terms of total contact time, however, the McKenzie therapists spent more time with their patients than the chiropractors (201).

McKenzie and chiropractic treatments both provided modest levels of pain relief, compared with a control group whose only treatment was an education pamphlet. The control group functioned just as well at the end of a month as did patients who had the more expensive McKenzie or chiropractic therapy.

Seventy-two percent of patients treated with the McKenzie protocol said that they would treat their next episode of back pain themselves and not seek professional care. Only 39% of the chiropractic group expressed a similar view (201).

MANUAL THERAPY SUPERIOR TO CONVENTIONAL MEDICAL CARE FOR LOW BACK PAIN PATIENTS

Fifty-three acute or subacute patients with low back pain were given conventional treatment by primary health care teams. Forty-eight patients received an experimental treatment that included specific mobilization, muscle stretching, autotraction, and cortisone injections. The experimental manipulation was thrust techniques or specific mobilization. Treatment also was performed by seven physical therapists, more or less specialized in manual therapy.

Control patients received active, optimal and standardized conventional treatment. This approach is consistent with modern official recommendations for low back pain management in Sweden. In addition, they received drugs, verbal and written ergonomic advice, low back pain school training, sick-leave, active back exercises, corsets, taping, shortwave, ultrasonic waves, transcutaneous neuromuscular stimulation (TNS), transcutaneous electrical muscle stimulation (TEMS), electric stimulation, heat (Steam-pac), cold (Cold-pac, ice), postural instructions, postural exercises, and in some cases plunge bath training and massage (202).

After 4 months, the experimental group had increased range of motion, less pain in the back and lower extremity, and a less positive straight leg raising test (both sides) than the conventionally treated group. Manual treatment was superior to the conventional activating treatment in normalizing pathologic findings on physical examination of the lower back. These results agree with the positive influence on pain, drug consumption, sick leave, disability rating, and quality of life reported in other reports from the same study.

The study shows that manual therapy reduced the presence of clinical findings in a low back pain population more effectively than did conventional treatment supplied by primary health care teams. An essential component of the treatment might be the steroid injections, which have not been used with manual treatment in previous investigations (202).

Chiropractic care is at least as effective as medical care in reducing low back pain and functional disability caused by low back pain. Chiropractic patients were more likely to perceive their treatment to be successful in reducing low back pain as compared with medical patients (203).

One hundred forty five chronic low back pain patients demonstrated the clinical utility of spinal manipulative therapy by immediate reduction of reported pain after 2 weeks of treatment. This randomized trial successfully accounted for a number of the more serious criticisms of earlier studies on spinal manipulative therapy by using a methodologically more rigorous protocol. There appears to be treatment available to benefit patients with chronic low back pain (204).

Of 59 patients diagnosed with a lumbar disc herniation who received chiropractic treatment, 90% (56 of 59) reported improvement of their complaint after a median of 18 treatments (range 3 to 90 treatments) over a median duration of 45 days (range 2 to 180 days). Side posture lumbar spinal manipulation and electrotherapy (interferential current) were used in 93 and 97% of the cases, respectively. Seventy-eight percent of the patients were provided instructions on how to perform William's flexion exercises. No complications from the treatment were reported by the patients on follow-up visits or by the medical doctors on follow-up correspondence (205).

"Hands-On" Effect

A measure of patient satisfaction and confidence from manipulation is the potential benefit in patient expectation and satisfaction in the outcome. The success of manipulation may lay in the "hands-on" approach (206).

Facet Joint Effect of Spinal Manipulative Therapy

A total of 86 manipulations (46 fast and 40 slow) were conducted on the thoracic spine (vertebra level T3, T6, and T9) of 11 asymptomatic subjects. The fast treatments consisted of a high-velocity, low-amplitude "thrust" of short duration. For the slow treatments, force was gradually built up over a period of 3 to 4 seconds until peak force was achieved, and peak force was then held for another 2 to 3 seconds.

Manipulations were given in the following order: First, one or two slow treatments were administered to T3, followed by a fast treatment on the same level. After the fast spinal manipulative therapy (SMT), a fast burstlike electromyographic (EMG) signal was observed, which probably originated from type II articular receptors. During slow SMT, a continuous gradual increase in EMG activity occurred, which was probably the result of the activation of type I articular receptors. However, it has to be kept in mind that various types of mechanoreceptors are embedded in a joint and its related soft tissues. An SMT will likely activate a wide variety of receptors, including proprioceptors and pain receptors in muscles and skin. Thus the beneficial effects associated with SMT may be the integrated action of the simultaneous activation of multiple receptors (207).

Joint cavitation was not associated with a reflex response. Joint cavitation has often been considered a sign of a successful treatment. Anecdotally, joint cavitation has been assumed to evoke reflex responses which may cause a reduction in pain and muscle relaxation. Audible sounds do not evoke a measurable reflex response in muscles. They may still produce reflex activities that do not show themselves in EMGs (207).

Distraction Manipulation Stimulates Group III Afferents for Pain Relief

Distraction of the facet activates group III sensory receptors more often than compression. Manipulation of a lumbar facet stimulates group III afferents with receptive endings located in or near the tissues of the facet and endings located in lumbar paraspinal muscles distant from the facet joint capsule. Stimulation or modulation of this system may explain the beneficial effects many patients receive through physical therapy, bracing, and spinal manipulation. Future research is necessary to determine the precise role played by the activation of these receptors (208).

Opioid Production

Repetitive stimulation of small-diameter somatic afferents likely effect a release of opioids in an area such as the substantia gelatinosa (lamina II), where the overwhelming majority of these small fibers terminate (209). Passive joint movement relieves pain of spinal origin by arousing to clinically effective levels a pain control system encoded by opioid peptides (210).

Decreased Intra-articular Pressure

Gate control theory is that selective excitation of larger diameter somatic afferent units in peripheral nerves will suppress the rostral transmission of the painful information at the level of the dorsal horn of the spinal cord. Passive joint movements could produce pain relief by effectively reducing afferent input, reducing intra-articular pressure, and inhibiting reflex muscle contraction. The level of pressure in human lumbar apophyseal joints could be subsequently reduced by passively moving these joints at the end of their range for a few minutes. Such a de-

crease in intra-articular pressure could reduce discharges in joint afferents. Relaxation of reflexly contracted muscles about a joint would help abolish both the pain of muscle ischemia and any additional discharges produced in joint afferents caused by tension placed on periarticular tissue (211).

DIAGNOSTIC IMAGING CHANGES FOLLOWING TREATMENT OF HERNIATED LUMBAR DISC PATIENTS

Nonsurgical Care

Of 47 lumbar disc herniation patients, 42 underwent conservative medical therapy consisting of bed rest, lumbar support, epidural steroid injections, twice-daily physical therapy, analgesics, and nonsteroidal anti-inflammatory agents. Five of the cases went to surgery. Repeat CT showed herniation reduction from 25 to 100%. Figures 12.28 to 12.31 show the classification of size, disc size reduction, and the plot showing the percentage size decrease from initial and second CT scan. Most lumbar disc herniations do reduce under conservative care and the largest herniations seem most likely to undergo significant reduction in size (212).

Bozzao et al. (213) reported that 48% of 69 lumbar disc herniations showed greater than 48% reduction in size, 63% greater than 30% size reduction, and 8% were worse under conservative care. They concluded that lumbar disc herniation may be primarily a nonsurgical problem.

Resolution of a patient's pain can occur with or without resolution of the hernia. Up to 36% of the population have asymptomatic disc herniations; it is not just the structural presence of a disc hernia that is responsible for a patient's radicular pain (214).

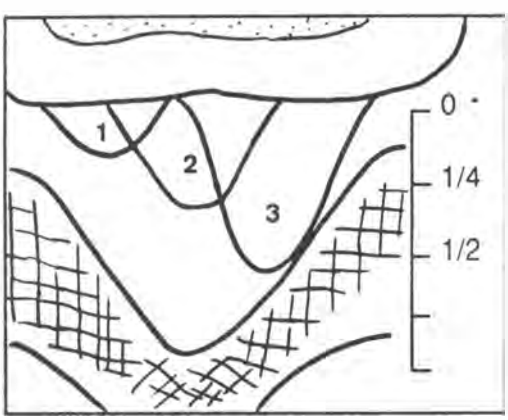


Figure 12.28. Classification of the size of disc herniations observed during the initial computed tomography scan. Size is determined with respect to the anteroposterior diameter of the lumbar canal. 1: small herniation (less than one fourth); 2: medium herniation (between one fourth and one half); 3: large herniation (more than one half). (Reprinted with permission from Maigne J, Rime B, Delignet B. Computed tomographic follow up study of forty-eight cases of nonoperatively treated lumbar intervertebral disc herniation. Spine 1992:17(9):1071–1074.)

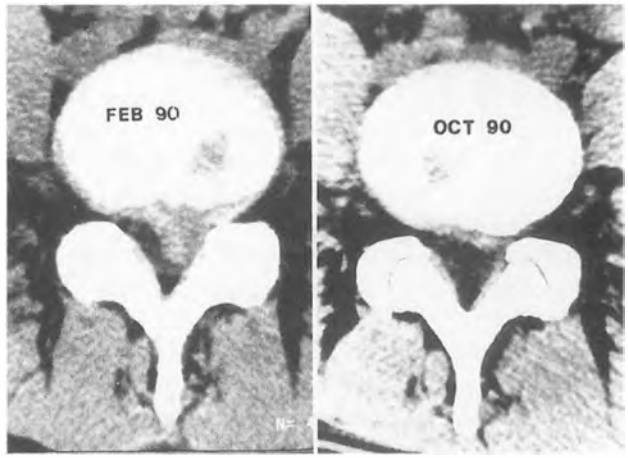


Figure 12.29. Large herniation having decreased by 95% in 9 months. (Reprinted with permission from Maigne J, Rime B, Delignet B. Computed tomographic follow up study of forty-eight cases of nonoperatively treated lumbar intervertebral disc herniation. Spine 1992:17(9):1071–1074.)

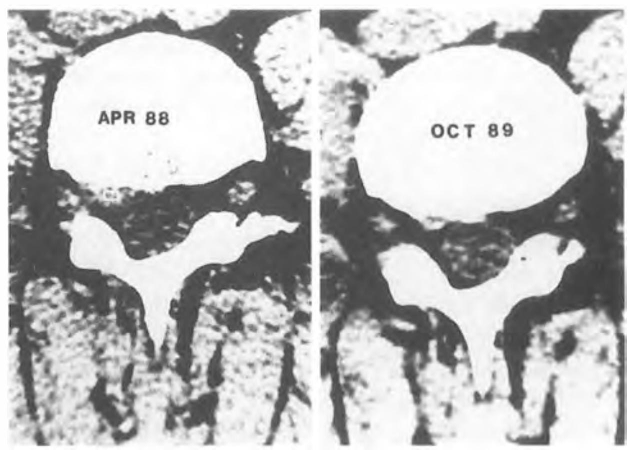


Figure 12.30. Small herniation that decreased in size by 75% in 18 months. (Reprinted with permission from Maigne J, Rime B, Delignet B. Computed tomographic follow up study of forty-eight cases of nonoperatively treated lumbar intervertebral disc herniation. Spine 1992:17(9): 1071–1074.)

Eighteen subjects with lower extremity pain or paresthesia, positive straight leg raising, weakness in a myotomal distribution, reflex asymmetry, or electromyogram evidence of radiculopathy underwent conservative care with complete clinical improvement and resolution of the disc herniation in 78% of cases. Patients without demonstrated disc resolution or improvement in the disc herniation can still show a complete clinical improvement without recurrence over a 2.5-year follow-up (215).

Follow-up CT scan of 21 patients with herniated lumbar discs treated with steroid injection or oral medication showed definite decrease in size of the herniated nucleus pulposus in 14 patients: disappearance in 5, obvious decrease in 5, and moderate decrease in 4. No definite change was observed in seven patients. Major changes on CT scan occurred significantly more frequently in a large herniated nucleus pulposus than in a small

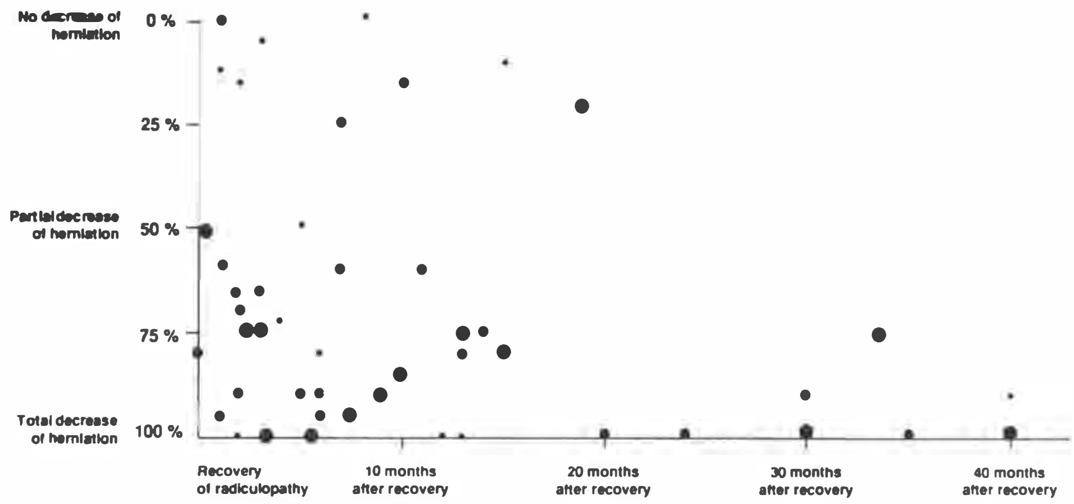


Figure 12.31. Each point represents a case. The horizontal axis represents the number of months that have elapsed between the cessation of treatment and the second computed tomographic (CT) scan. The vertical axis represents the percentage (%) decrease in herniation size between the initial and second CT scan. The size of each point corresponds to the size of the herniation on the initial CT scan: small herniation, medium herniation, and large herniation. (Reprinted with permission from Maigne J, Rime B, Delignet B. Computed tomographic follow up study of forty-eight cases of nonoperatively treated lumbar intervertebral disc herniation. Spine 1992:17(9):1071–1074.)

one. Large lumbar herniated nucleus pulposus can decrease and even disappear in some patients treated successfully with conservative care (216).

Steroid and local anesthetic injection at the intervertebral disc nerve root interface of 84 cases of disc herniation and sequestrations showed 64 (76%) had either complete or partial resolution on follow-up CT examination. Of 22 cases with either a generalized or focal bulge of the disc, 18 (82%) were unchanged on follow-up.

Even if patients have a marked reduction of SLR, positive neurologic signs, and a substantial IVD herniation or sequestration, a potential exists for making a natural recovery. Indeed, disc herniation, the abnormality that might seem best suited to surgical resection, is the type of disc lesion showing the most significant incidence of natural regression. Acute disc herniations in young patients are the category of disc pathology most likely to show greatest resolution on follow-up CT examination (217).

Symptoms and Signs Do Not Correlate with the Degree of Disc Herniation Reduction

At 2 years after conservative care, herniated nucleus pulposus patients showed 16.5% reduction in the size of the herniation. Reduction was found in 57% of the patients, no change in 40%, and 3% were enlarged (218).

Thirty-two patients with herniated lumbar discs, treated conservatively, were studied. MRI was performed in the acute stage, then 6 months and 1 year later. On axial images, the proportion of the cross-sectional area of the spinal canal occupied by the herniated disc was 31.9% on the average on the initial scan, 28.7% at 6 months, and 25.3% 1 year later. The size of the herniation decreased by more than 20% in 11 patients (34%), by 10 to 20% in 8 (28%), and was unchanged in 12 (38%) (219).

Herniated discs occupying more of the spinal canal on the initial study showed a more marked reduction in size. Most reduction occurs in the latter half of the first year.

The size of the herniated material reduced significantly more in the patients developing severe degeneration than in those with mild degeneration, suggesting that degeneration might participate in reducing herniation via dehydration, shrinkage, and degeneration followed by scar contraction.

Patients whose symptoms disappeared with short-term rest but whose reduction of herniation was mild, and others who had achieved slight improvement in symptoms and marked reduction of herniation are reported. Symptoms might not always parallel the reduction of herniation because reduction occurs considerably later than symptoms (219).

Patients with multiple lumbar disc herniation (22) and those with single lumbar disc herniation (37) receiving conservative treatment (mainly spinal manipulation) were compared before and after a period of conservative treatment. *No obvious changes were seen in herniated nucleus pulposus size, position, and volume, even after clinical improvement.* However, structural and functional recovery in the group with multiple segments was less satisfactory than that of the group with single segment involvement (220).

Possible Mechanisms of Disc Resorption Under Conservative Care

Chemokines

Monocyte chemotactic protein-1 (chemokine) is supposedly produced by damaged anulus fibrosus and epidural vessels, which in turn promotes recruitment of monocytes, resulting in further production of other chemokines including macrophage inflammatory protein-1. These chemokines may contribute to activation and recruitment of macrophages in a paracrine or an autocrine fashion in the initiation of the resorption process of herniated nucleus pulposus (221).

Phagocytosis

An extruded or sequestrated disc has the potential to be resorbed by phagocytes (macrophages and T lymphocytes) by creating inflammatory change (e.g., cell infiltration, neovascularization, and granulation) seen in 16.9% of protruded discs, 81.8% of subligamentously extruded discs, 100% of transligamentously extruded discs, and 80% of sequestrated discs (222).

Hypervascularity of the Herniated Fragment

After IVD injury or as a result of degenerative changes, the disc becomes vascularized as blood vessels grow into ruptured areas in the anulus and may be involved in the absorption of herniated disc tissue, which in turn would cause a decrease in symptoms (223).

Percutaneous Lumbar Discectomy Pre- and Post-MRI Findings

Little change in appearance is seen in the post percutaneous lumbar discectomy (PLD) disc even in patients with a successful outcome. The mechanism by which pain relief is accomplished by the procedure remains to be elucidated (224).

A small absolute decrease in the size of disc herniations on the average (5% of the anteroposterior diameter of the spinal canal) is noted in PLD with success likely when 10% absolute change in size is seen postoperatively. Pain relief without a measurable change in size may be due to the specific pressure-volume dynamics (bulk modulus of elasticity) of the IVDs, to the removal of chemical irritants, or to placebo effect (225).

POSTSURGICAL RECURRENT DISC OR SCAR TISSUE FAILS TO CORRELATE WITH PAIN

Contrast-enhanced CT during the first postoperative week, after 1 to 2 months, and after 1 year was studied in 50 patients with single level disc herniation. At 1 year after surgery, 16 patients showed posterior disc protrusion, 47 scar tissue, and 13 nerve root displacement. Microsurgically operated patients did not show less scar tissue than laminectomy patients. None of the postoperative radiographic changes definitely correlated with remaining back or leg pain (226).

MRI Does Not Correlate with Postoperative Back Pain or Radicular Leg Pain

A prospective study of 36 patients with radicular leg pain and lumbar herniation who underwent single level disc resection showed in clinical follow-up with a gadolinium-DPTA MRI examination 1 year after surgery that the disc herniation was still present in eight patients and four of these did not have any significant radicular pain. Another 15 patients had a small protrusion at the site of the former herniation. Twenty-three patients showed evidence of scar tissue. The nerve root was displaced in 12 patients and was thickened in 16 patients, respectively. Clinically, 19 patients recovered from leg pain, 14 patients improved, and 3 patients remained unchanged compared with preoperative symptoms. No consistent correlation was found between postoperative back pain or radicular leg pain and MRI findings (227).

Disc Contour Change Persists After Successful Surgical Removal

Successful discectomy patients may show localized disc contour abnormalities that simulate recurrent disc herniation for months after surgery (228). Asymptomatic lumbar disc herniation patients show residual mass effect on the neural elements simulating recurrent or residual disc fragment after surgery. An orderly progression of imaging changes occur during the first 6 months after lumbar surgery that limits the interpretation of MRI examinations during that period (229).

Unilateral nerve root enhancement at the operated level is seen in most patients on the initial (3-week) postoperative study. Extradural root enhancement (excluding the dorsal root ganglion) is seen in 81% of the levels initially and is still present on the 6-month study in approximately a third of the patients. Intradural nerve root enhancement can be seen tracking cephalad toward the conus medullaris on the operated side in 62% of the disc levels on the initial postoperative study, but this enhancement is no longer present on most images at 3 months (230).

The amount of soft tissue seen in the anterior epidural space postoperatively is often greater than that found preoperatively. No correlation is found between the clinical outcome and the size or nature according to MRI of the postoperative soft tissue masses. Edema and scar tissue formation is the probable reason for difficulties in interpreting postdiscectomy MRIs. *Early postoperative MRI after lumbar discectomy must be interpreted carefully; edema and scar formation are probably reasons for difficulties in interpretation* (230).

An Objective and Accurate Correlative Assessment System Needed

Computed tomography examinations were done before and at 3 and 24 months after nonoperative treatment in 30 patients with lumbar disc herniations. The size of the herniation was described by different indexes and related to the degree of sciatica.

Because the degree of sciatica is thought to be an important clinical parameter, it seems that the sagittal diameter of the canal and the herniation are the two most important dimensions to be measured (231).

In 16 patients with total regress of sciatica, a disc herniation was still visible on CT3, although an obvious reduction in absolute and hernia size had occurred from CT1 to CT3. Seemingly, an individual threshold was found to relative hernia size under which sciatica disappeared. As long as the relative hernia size was above this level, the mechanical stimuli caused by the herniation was an important factor contributing to the degree of sciatica by triggering a local inflammatory painful reaction in the adjacent nerve root. If the hernia size decreases under the threshold level, the degree of pain will be reduced disproportionally because of the reduction of the painful inflammatory reaction. This is in accordance with previous findings in which no relation was found between hernia size and the straight leg raising test, indicating other factors such as an inflammatory reaction and not pure mechanical pressure by the herniation on the nerve root as causative for the degree of sciatica.

The need for an objective and accurate assessment system when describing the size of disc herniations is obvious. By such a system, the radiologic evaluation of the effect of different treatment programs on the size of the herniation would become much more precise (231).

Cox (232) described a method to measure the size of the disc hernia as a percent of the sagittal canal diameter. This report allowed before treatment and posttreatment comparison of the disc herniation size and nerve root or cauda equina compression with a patient's subjective and objective symptoms. Little correlation between the herniation size and symptoms could be made from this study.

The following two cases illustrate chiropractic treatment showing limited disc reduction following successful relief of sciatic and back pain under flexion distraction adjustments.

Case 5

Figures 12.32 to 12.35 are pre- and post-MRI studies of a 37-year-old woman treated under distraction adjusting for the chief complaint of low back pain and right leg pain extending to the heel. The patient did have motor weakness of the right calf muscle, an SLR positive at 40 ° on the right, and a diminished right ankle reflex. Hypesthesia of the right S1 dermatome was noted.

Figures 12.32 and 12.33 are the axial and sagittal images before treatment started showing the right paracentral disc herniation. Treatment of distraction adjusting with positive galvanism and tetanizing currents to the L5–S1 disc and right sciatic nerve at the gluteus maximus level was administered. Tetanizing current to the right calf muscles was also given to aid in regaining muscle strength. The patient took glycosaminoglycan and glucosamine sulfate for disc supplementation, started rehabilitation exercises, and attended low back wellness school. The rule of attaining 50% relief within 1 month of care or a surgical consultation obtained was explained to her.

One month of care resulted in 80% subjective relief as measured on Oswestry pain index, Roland Morris Low Back Disability questionnaire, and VAS. The deep tendon reflexes, ranges of motion, and SRLs were normal.

Repeat MRI shown in Figures 12.34 and 12.35 reveals reduction

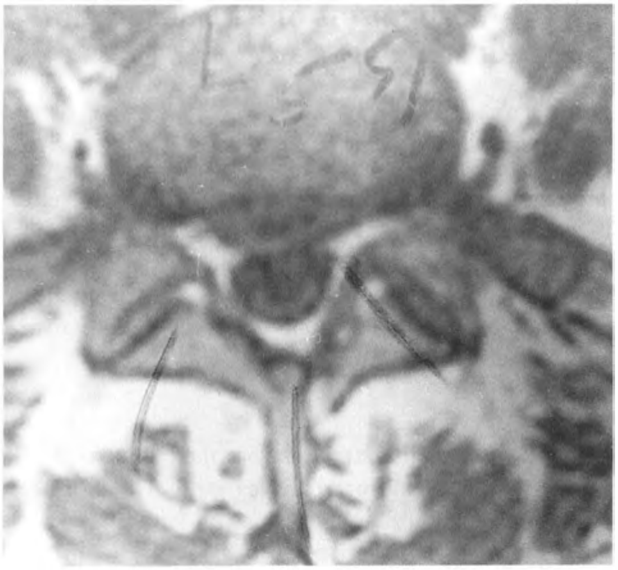


Figure 12.32. Axial view showing the right paracentral disc herniation at L5–S1 prior to distraction adjusting.

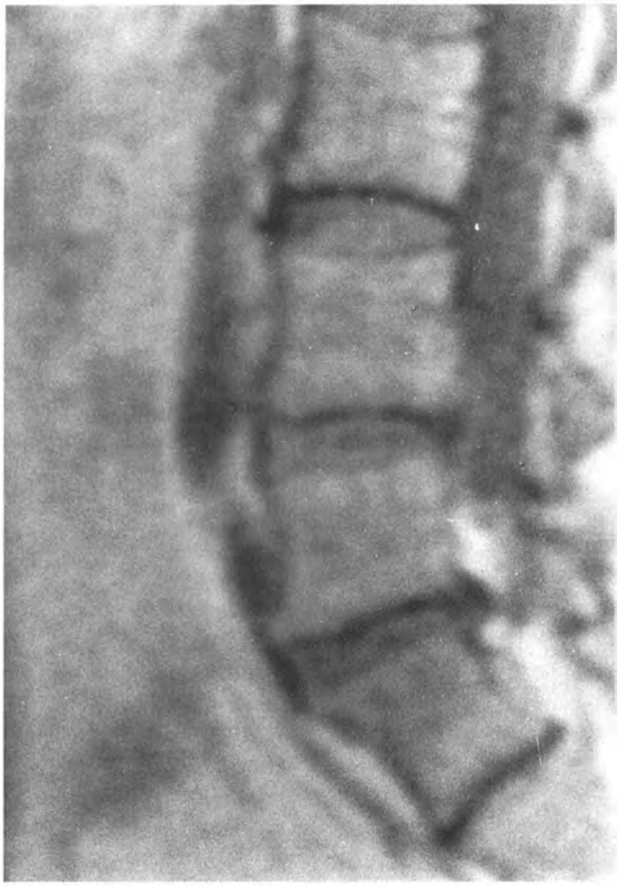


Figure 12.33. Sagittal view of the L5–S1 disc herniation prior to treatment.

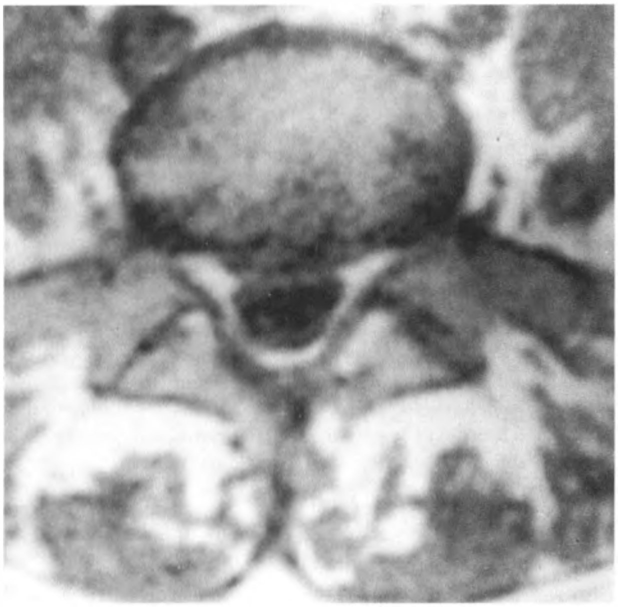


Figure 12.34. Axial view of the L5–S1 disc shown in Figure 12.32 after relief of pain. The disc herniation is diminished but definitely present.

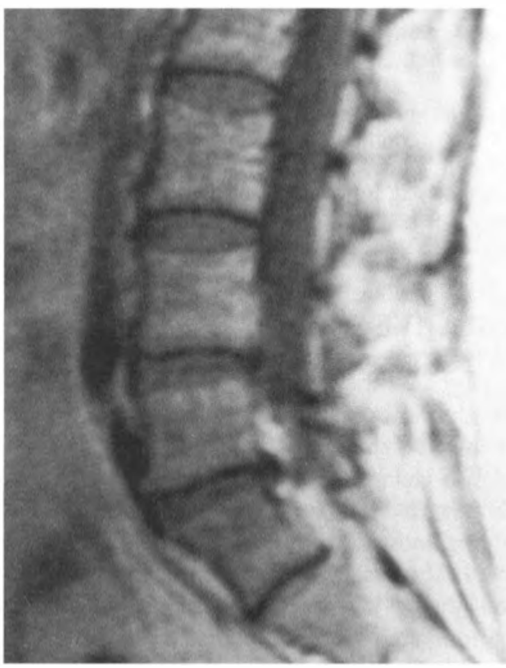


Figure 12.35. Sagittal view after the patient is asymptomatic still showing the L5–S1 disc herniation. A good example that disc size and location mean little concerning clinical presentation.

in the size of the disc herniation, although it is still evident. This case is an example of complete relief with disc herniation still present.

Case 6

Figure 12.36 is an L5–S1 left paracentral focal disc herniation in a patient with left S1 dermatome sciatica. Following successful management with flexion distraction adjustments, Figure 12.37 shows remaining disc herniation, although it is diminished in size.

Transdural Disc Herniation

Case 7

Three surgeries and cauda equina syndrome before transdural herniation is diagnosed—a cause of concern for a doctor.

A 45-year-old automobile mechanic with a 20-year history of untreated low back pain and a few slight episodes of left leg sciatica, felt a sudden onset of severe low back pain while attempting to lift a 100 L oil drum. An L5 laminectomy was performed. The patient awoke from surgery with greater low back and sciatic pain, bilateral loss of dorsiflexion of his feet, loss of sensation in the perianal area, and retarded micturition (233).

A myelogram showed a complete stop at the L3–L4 disc (Fig. 12.38). An L4 laminectomy was performed with resection of scar tissue and possible inspection of the L3–L4 disc.

Again, the pain was worse postoperatively, and the man had developed anal and urinary incontinence with loss of sensation from L4 distal and loss of Achilles tendon reflexes and dorsal flexion of both feet. Bilateral leg pain was present and he was wheelchair confined. MRI (Fig. 12.39) showed a voluminous intradural tumor of the same density as the nucleus pulposus at the same level as the L3 disc.

The patient was taken back to surgery and laminectomies of L2 and L3 were performed, after which a large mass became ev-

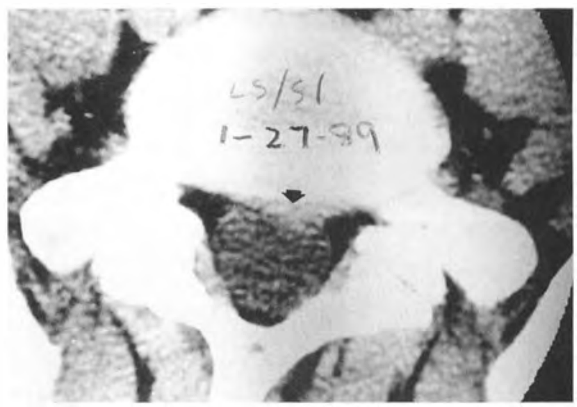


Figure 12.36. The left paracentral L5–S1 disc herniation prior to distraction treatment.

Figure 12.37. The same L5–S1 disc herniation 3 months after that seen in Figure 12.36. The patient is relieved of sciatica but a definite disc lesion remains that is not pain-producing. (Case presented by Terry Sandman, DC, DACBR.)

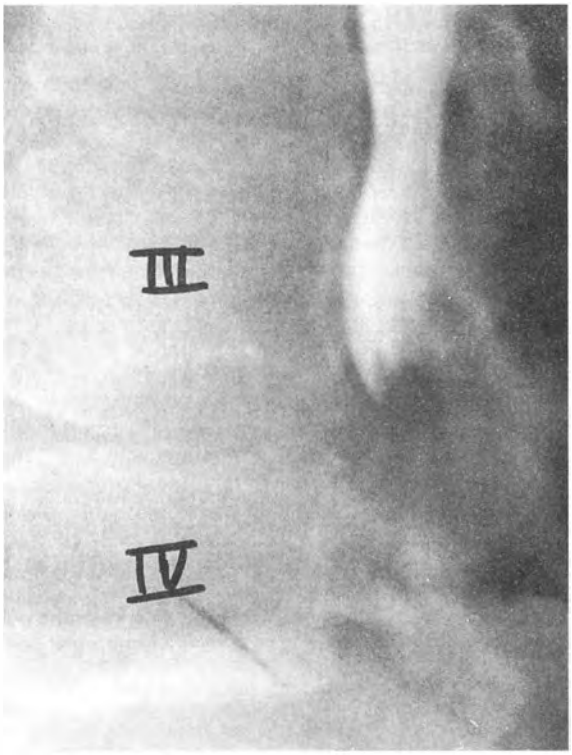


Figure 12.38. Lateral view of myelogram showing complete stop at L3–L4 disc. (Reprinted with permission from Reina E, Calonge ER, Heriot RPM. Transdural lumbar disc herniation. Spine 1994;19(5):617–619.)

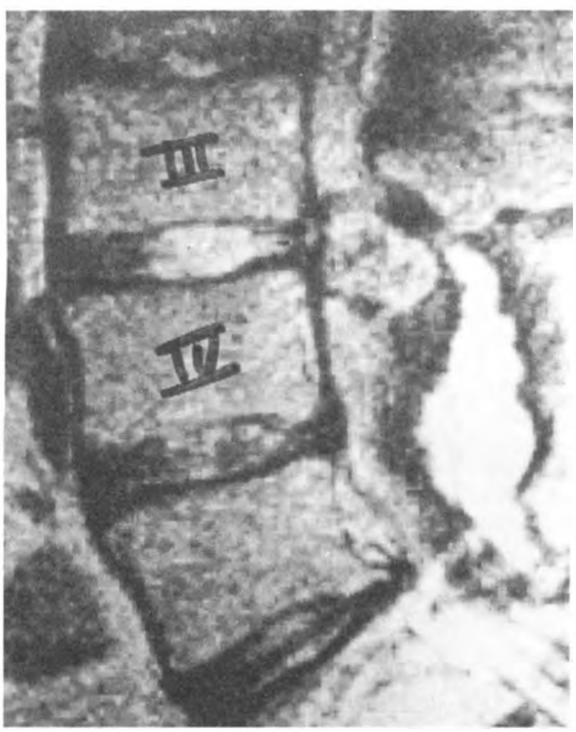


Figure 12.39. Sagittal magnetic resonance image showing a mass occupying the neural canal from disc through to lamina at L3–L4 level. (Reprinted with permission from Reina E, Calonge ER, Heriot RPM. Transdural lumbar disc herniation. Spine 1994;19(5):617–619.)

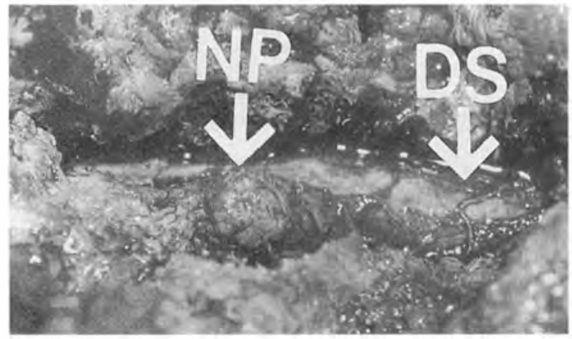


Figure 12.40. Photograph at surgery showing large mass (*NP*) protruding through posterior aspect of the dural sac (*DS*) medial to the axilla of the left fourth lumbar nerve root. (Reprinted with permission from Reina E, Calonge ER, Heriot RPM. Transdural lumbar disc herniation. Spine 1994;19(5):617–619.)

ident as it extruded through the dura medial to the axilla of the left fourth lumbar nerve root (Fig. 12.40). The dura was opened without opening the arachnoid membrane, and on inspection the tumor seemed to be coming from the anterior wall of the canal. On examination, it measured 3 × 1.5 / 1 cm and had the consistency of nucleus pulposus.

In the postoperative period, the pain disappeared almost completely, and the patient partially recovered urinary and anal function.

The mechanism of penetration for a dural disc protrusion is such that the posterior longitudinal ligament must be perforated for the disc herniation to reach the anterior dura. Much force is required for this to occur and it is suggested that the mechanism is that of sustained pressure, causing necrosis of the dura over a prolonged time (221).

MANIPULATIVE EXPECTATIONS IN THE CLINICAL SETTING

Today, the clinical expectations for manipulative approaches are good compared with the results reported in the early studies in the 1950s to 1980s.

Splendid research on the biomechanics of the low back is presently being performed, and varying techniques in the surgical treatment of low back conditions are being investigated and tried. Therefore, it is incumbent on chiropractic to develop manipulative care of the low back to its utmost perfection.

According to Dommisse and Grabe (234), a spinal surgeon rather than an orthopaedic or a neurosurgeon is the appropriate leader of the surgical team in an operation on the spine. This reflects the idea that a surgeon whose training is primarily in spinal surgery is the appropriate physician to enter the spine. It might also be said that some chiropractic physicians should specialize in the care of the low back and make it their primary study and practice. To this end, the manipulative care of other specialists throughout the world is briefly examined.

Hadler et al. (235) reported a randomized controlled study in which 54 subjects were placed under manipulative care. The patients were divided into two subgroups: those with acute low back pain of less than 2 weeks duration, and those whose dis-

comfort had persisted for 2 to 4 weeks. Outcome was measured with a functional impairment questionnaire, which showed that those patients who had suffered a backache were afforded more rapid improvement if they were subjected to spinal manipulation. Because of the extreme prevalence of low back pain in society today, this finding of relief in fewer days than without such care was pointed out to be a major ramification. I find this controlled study to provide a more realistic appraisal of manipulation than previous studies of more questionable design and purpose.

Arkuszeqski (236) allocated 100 patients with lumbago or sciatica alternately into two groups, all of whom received standard drug treatment and physiotherapy, undergoing manual examination twice a week. Traction, mobilization, and/or manipulation was applied to all parts of the spine in the manual treatment group. In 60% of patients, there was concomitant neck pain. Blockages of the cervical segments were found in 95% of the patients, the atlanto-occipital segment being the one most frequently affected. In the manual treatment group, the treatment period was shorter, and posture, pain intensity, and neurologic signs showed greater improvement both on discharge and 6 months later. When manipulation was compared with standard conservative medical care, the manipulation group had a 30% reduction in hospitalization time, a greater number who remained well at 6 months, and greater improvement in neurologic findings.

Ongley et al. (237) contrasted two patient groups: an experimental group of 40 patients receiving manipulation along with proliferant injections, and a control group of 41 patients receiving parallel treatment with less forceful manipulation and saline solution instead of proliferant. Disability scores then showed that the experimental group had greater improvement than the control group at 1, 3, and 6 months after treatment ended. At 6 months post-treatment, an improvement of 50% or more was recorded in 35 of the experimental group versus 16 of the control group, with 15 of the experimental group free of pain versus four of the control group. The experimental group receiving manipulation showed significant advantages over the control group, who had not received manipulative care.

Rupert et al. (238) carried out a chiropractic controlled clinical trial to evaluate the efficacy of chiropractic adjustments in the treatment of low back pain among 148 Egyptian workers. The patients were randomly assigned to one of three treatment regimens: chiropractic adjustments, drugs and bed rest, or placebo. Treatment results were evaluated by the VAS, active and passive SLR, and the finger-tips-to-floor assessment of forward flexion. Chiropractic treatment was associated with the greatest improvement.

Cox and Shreiner (239) carried out a chiropractic multicenter observational pilot study of 576 patients with low back and/or leg pain to compile statistics on examination procedures, diagnosis, treatments types rendered, treatment results, number of days and treatments required to arrive at a 50% and a maximal clinical improvement. This study showed that 275 (50%) of the patients showed an excellent outcome, 74 (14%) very good, 60 (11%) good, 36 (6%) fair, and 22 (4%) poor; 17 (3%) underwent surgery, 57 (10%) stopped or did not undergo treatment, and 7 (1%) were examined but not treated. Fifty percent relief of pain was seen after 14.4 days and 9.5 treatments (mean values). Maximal improvement was obtained after 42.8 days and 18.6 treatments (mean values).

Potter (240) reported chiropractic manipulation in 744 cases of neck and back pain, whether acute or chronic, with or without radicular signs. Overall statistical results reported were recovery in 268 cases (36%), much improvement in 257 (34.5%), slight improvement in 54 (7.3%), no change in 161 (21.6%), or worsened condition in 4 (0.6%).

Nyiendo and Haldeman (241) found that 80% of low back pain patients seen at a chiropractic college teaching clinic were diagnosed as having lumbosacral strain, with 23% of 2000 patients receiving one visit, 54% receiving two to five treatments, and less than 1% receiving more than 20 visits. The range was 1 to 81 visits, with a mean of 5.3 visits.

Bronfort (242) reported on 298 patients with acute or chronic low back pain from 10 different chiropractic clinics who were selected for study. Fifty-three percent of them had consulted a medical doctor or had received other types of treatment because of their current episode of pain. Seventy-five percent of these patients reported being free of symptoms or feeling much better following chiropractic care.

Waagen et al. (243) performed a double-blind study of the efficacy of spinal adjustment therapy in a college clinic. Nineteen patients with low back pain underwent a 2-week treatment period, with nine patients receiving chiropractic adjustments and ten in a control group receiving a comparable series of manual interventions. It was reported that the experimental nine patients improved significantly compared with the control group.

Wooley and Kane (244) stated that major areas of difference were found between allopathic and chiropractic care for low back pain with respect to the number of visits and duration of treatment given. Patients were seen an average of approximately 13 times by chiropractors, as opposed to approximately 7 times by allopaths. However, the treatment by allopaths took more than 9 weeks, as opposed to 6.5 weeks for chiropractors, which averaged 1.2 visits per week for allopaths and 2.5 visits per week for chiropractors.

Chrisman et al. (245) found that 10 of 27 patients with positive myelograms had good to excellent results 3 years or more after manipulation. Fifty-one percent of patients with clinical evidence of lumbar disc rupture had good to excellent results from manipulation.

A full-scale multicenter trial to include 2000 patients was proposed, which was found feasible after a study for a randomized controlled trial of chiropractic and hospital outpatient management for low back pain of mechanical origin (246). Patients who were eligible for this study were interviewed by a nurse coordinator who explained the purpose of the study and pointed out that agreement to take part involved an equal chance of being treated by chiropractic or by conventional hospital methods, the decision being made at random. The study involved the Northwick Park Hospital and a local chiropractic clinic.

Vernon et al. (247) found manipulation to be associated with a statistically significant increase in serum β-endorphin levels when blood testing was performed before and after spinal manipulation. This allows a hypothesis that pain relief induced by manipulation is due, in part, to a short-term increase in β-endorphin levels.

MANIPULATION AND DISTRACTION TECHNIQUES AND CONCEPTS FOR LOW BACK AND LEG PAIN TREATMENT

In considering some of the manipulative treatments of low back and leg pain resulting from IVD lesions, two approaches could be responsible for relief attained by manipulative efforts. These are, first, the reduction of anular and nuclear disc protrusion, with relief of the anular irritation that can cause back pain; and second, the possible effect of manipulation on stimulating circulation and causing resorption of the inflammatory effects of free nuclear material within the spinal canal.

Burton's Concepts of Traction Reduction

Burton (248) performs nonsurgical treatment of back and leg pain patients with acute contained disc herniations by using a chest harness to suspend the patient and using patient's body weight as the distractive force. He finds the technique of benefit in the following three entities:

1. Disc "bulging" producing distention of the anulus and posterior longitudinal ligament. This entity produces pain by stimulating branches of the sinuvertebral nerve. A dorsal ramus pain syndrome, typically referred to the low back, hips, and knees, is produced. Pain is rarely referred as far as the ankles.
2. A herniated disc, in which nuclear material extends beyond the anulus but is contained by the posterior longitudinal ligament (sometimes called a "roof disc"). Compression of a spinal nerve either exiting or traversing the interspace produces sciatic pain that radiates to the toes and feet, and neurologic findings that are associated with this compression.
3. A herniated disc in which nuclear material extends beyond the anulus and is beginning to erode through the posterior longitudinal ligament but has not yet become a free protrusion.

Experience has shown that when herniated disc material extrudes past the posterior longitudinal ligament (free protrusion) or migrates in the spinal cord (sequestered fragment) the application of gravity traction accentuates pain and neurologic deficit rather than alleviating it. This phenomenon occurs during the first few days of treatment and is most important to document, because it signals the need to discontinue the traction program and consider more aggressive treatment modalities such as chemonucleolysis or surgery.

Figure 12.41 shows Burton's schematic observation of what

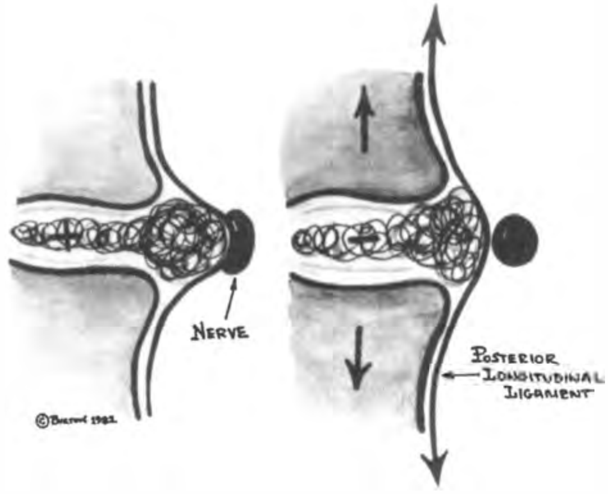


Figure 12.41. Computed tomography scan showing that the application of axial traction on the vertebrae, anulus fibrosus, and longitudinal ligaments caused the protruding disc to diminish in volume but rarely to return to its normal state. The clinical problem relates to distension of anular and ligamentous dorsal ramus nerve fibers and spinal nerve compression. It is believed, on the basis of biomechanical calculation, that significant intradiscal negative pressures may be produced. The intermittent reduction appears to allow reparative processes to re-establish support. (Reprinted with permission from Burton CV. Gravity lumbar reduction. In: Kirkaldy-Willis WH, eds. Managing Low Back Pain. Edinburgh: Churchill Livingstone, 1983:196.)

"before and after" CT scans show when gravity reduction is applied to contained discs. This indicates substantial change in spinal nerve root compression and suggests that the total reduction of disc protrusion is of secondary importance to nerve root decompression.

According to Hirschberg (249), herniation of a nucleus pulposus causing nerve compression can heal spontaneously, provided that low intradiscal pressure can be maintained for 3 months.

Neugebauer (250), who has treated more than 30,000 patients in 14 years, has proved that a disc prolapse can be converted into a disc relapse. He has achieved a 99% incidence of healing and believes that decompression treatment provides the only lasting recovery for the patient with a disc prolapse. Neugebauer has found that, as evidenced by x-ray measurement, he can increase the height of the L5 disc; he has increased the IVD distance from 3 mm dorsally and 9 mm ventrally to 6 mm dorsally and 15 mm ventrally over a course of treatment of 6 months. He is the first person to document that a disc can be re-established by decompression treatment.

Neugebauer achieves three therapeutic effects by his decompression treatment:

1. The disc is re-established.
2. The intervertebral foramen is enlarged, giving enough space for the nerve root to escape the prolapse.
3. Restretching of the anterior and posterior longitudinal ligaments brings the vertebra back into its normal position.

Other Concepts of Disc Treatment

In principle, traction stretches the back so that vertebrae are pulled away from each other, and radiographic studies have suggested that spinal traction is capable of distracting vertebrae and diminishing disc protrusion in patients with herniated discs (251, 252).

Tien-You (253) believes that manipulative reduction is the key to the treatment of patients with a protruded nucleus but asks the question: "Can a protruded nucleus be reduced by simple manipulation?" His answer is that a specific feature of the nucleus pulposus is its strong elasticity. This elasticity has been used during manipulative reduction to change the shape of the space between the affected vertebrae and to produce a retractile force by which the prolapsed nucleus is pulled back to its original position.

Others (254–261) using similar techniques have provided strong documentary evidence to the effectiveness of manipulative treatment and the nonsurgical approach to the care of patients with myelographically proved disc protrusion who are awaiting surgery.

How much can the IVD space be opened on distraction? Gupta and Ramarao (262) write that traction by various methods was a popular form of treatment for lumbar disc prolapse in the early years of this century. Subsequently, it fell into disrepute until the middle of the century, when more modern and sophisticated traction techniques were introduced and became popular. For example, Mathews and Yates (257) are reported to have demonstrated the efficacy of traction in reducing lumbar disc prolapse in three patients, with the help of epidurography. In this series, symptoms persisted and no change was seen in the patterns on epidurograms in only two of 14 patients, supporting the popular belief that disc protrusion can be safely treated by traction.

Others have reported a distraction of 1.5 mm per disc space after lumbar traction (Fig. 12.42), and a vertebral distraction of 2 mm/disc after traction (262). However, a vertebral distraction of only 0.5 mm per disc space was reported.

Lind (263) documented a 20.7% increase in the IVD space during manipulative reduction of lumbar disc protrusion. Furthermore, of 20 patients awaiting surgery for lumbar disc protrusion, 14 received complete relief from pain within 1 hour of application of the autotraction technique.

Discs absorb shock in two ways: (*a*) by squeezing fluid out of the nucleus, and/or (*b*) by allowing the fibers of the outer shell to stretch (264). Hukins and Hickey (265) show that the disc fibers have limited elasticity and suffer irreparable damage at 1.04 times their initial length. When a person is standing upright, the discs can withstand 10 times more compression than the vertebrae can, so a heavy load crushes the bones before it ruptures the disc. Disc fibers are less capable of coping with torsion because the stress then concentrates at points of maximal curvature. It has been reported that astronauts are 5 cm taller on their return to earth than they were when they left (264). Nachemson (266) reports that they are 10 cm taller.

Protrusion or rupture of the disc is usually preceded by degenerative changes characterized structurally by radiating cracks in the anulus that develop and weaken its resistance to nuclear herniation. As Tindall (267) points out, the sinuvertebral nerve supplies the posterior longitudinal ligament, periosteum, meninges, articular connective tissue, anulus, and vascular structures of the vertebral canal. The characteristic clinical features of back and leg pain, therefore, are related to irritation and stretching of the sinuvertebral nerve by the bulging anulus and by direct pressure on the nerve root, respectively (267).

Effects of Distraction of the Intervertebral Disc

According to Cyriax (268), three effects result from traction and its attendant distraction on the IVD (Fig. 12.43):

1. Increase in the interval between the vertebral bodies, thus enlarging the space into which the protrusion must recede.
2. Tautening of the joint capsule, which allows the ligaments joining the vertebral bodies to exert centripetal force all around the joint, thus tending to squeeze the pulp back into place.
3. Suction.

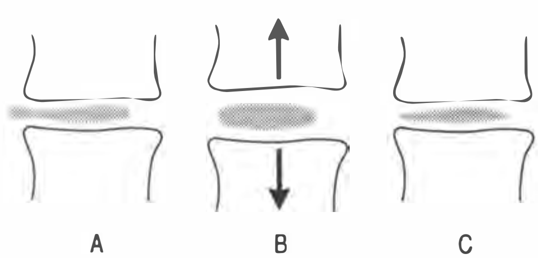


Figure 12.42. Effect of traction after discography. **A.** Disc herniation: dye protruding backward (to the left). **B.** On traction, the protruding disc material returns to the center of the disc. **C.** On relaxing the traction, the disc material tends to remain in the center of the disc. (Reproduced from Kirkaldy-Willis WH. Managing Low Back Pain. Edinburgh: Churchill Livingstone, 1983:179.)

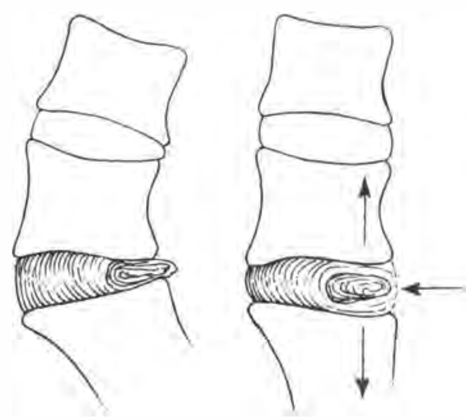


Figure 12.43. Positive effect of traction (lumbar flexion) on protruding fragment of disc. (Reprinted with permission from Finneson BE. Low Back Pain, 2nd ed. Philadelphia: JB Lippincott, 1980:312.)

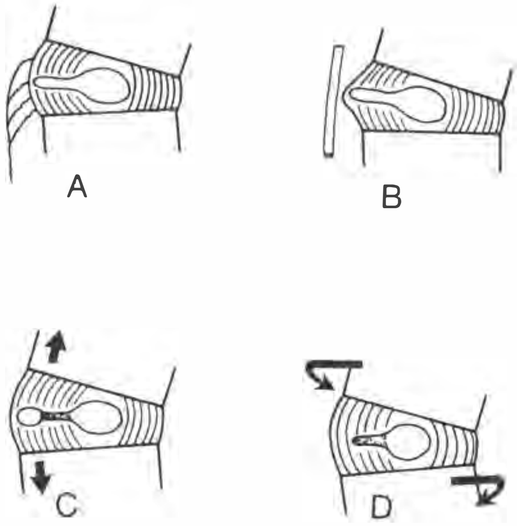


Figure 12.44. Explanation of the way in which manipulation can reduce pressure of a disc herniation on a nerve. **A.** Herniation with irritation of branches of the sinuvertebral nerve. **B.** Herniation with pressure on a spinal nerve. **C.** Traction separates the vertebral bodies and allows the herniated material to return to the nucleus. **D.** Rotation encourages further return of herniated material to the nucleus. (Reprinted with permission from Kirkaldy-Willis WH. Managing Low Back Pain. Edinburgh: Churchill Livingstone, 1983:179.)

Levernieux's experiments (269) on spines under distractive forces were done on cadavers whose discs were injected with an opaque dye and then placed under tractive forces. Radiographs made before, during, and after traction showed that an internally disrupted disc, with the nuclear material protruding posteriorly into the vertebral canal, shows the dye return into the center of the disc as the disc space is widened. After traction was complete, part of the contrast material was retained in the center of the disc (Fig. 12.42**C**).

De Seze (270, 271) feels that low back pain is caused by nuclear fragments becoming lodged within the anular cracks. This creates anular bulging and pressure on the sinuvertebral sensory nerve innervation of the anular fibers. This explanation of how manipulation corrected disc protrusion is shown in Figure 12.44.

IS THE DISC THE SOURCE OF LOW BACK PAIN?

Graham (272) explored the question of whether back pain emanated from disc, facet joint, adjacent musculature, or the lateral recess. Two hundred consecutive patients underwent discographic examination of their discs, and during the procedure their pain responses were monitored. In 111 of the 200 patients, their original pain was precisely intensified during discography, whereas an additional 43 patients who had no pain during discography had an increased intensity of the presenting pain during the ensuing 24 hours of mobilization. Graham felt that these findings suggested that in most patients, *low back pain did emanate from the disc, as suggested by Mixter and Barr in 1934.*

Acute Symptomatic Disc Protrusion

Kessler (273), in a discussion on the effects of pelvic traction, states that "*static pelvic traction must not be used in the acute stage of a disc prolapse.*" The patient may feel less pain while the distractive force is applied, but as the traction is released, a marked increase in pain occurs, and the patient may even have some difficulty in rising from the treatment table. Such an effort is probably caused by absorption of additional fluid by the nucleus while the traction is applied and the development of a high intradiscal pressure as the distractive force is relaxed. This unfortunate result is less likely to occur with intermittent traction, but few patients in the acute stage tolerate this well. We often hear of the patient with low back pain who enters the hospital and is placed in pelvic traction. How often these patients are the same or even worse following such traction. In clinical practice we often see patients who have been hospitalized, undergone every test, and were discharged in the same or worse condition. Static traction actually opens the IVD space, allowing the nucleus to imbibe fluids, and can thereby increases the intradiscal pressure, which worsens the pressure against the already compressed nerve root.

According to Kessler (273), if a patient is hospitalized or can attend therapy sessions without risking worsening of the lesion from increased intradiscal pressure, treatment can include specific segmental manual distraction techniques applied by a therapist skilled in such techniques. Oscillatory techniques can relieve pain by increasing large fiber and proprioceptive input, thus relieving some of the protective muscle spasm. Possibly, decreasing the longitudinal slack in the posterior longitudinal ligament and anular lamellae overlying the bulge in the disc effects a centripetal movement of the disc material away from the pain-sensitive structures. Thus, Kessler (273) believes that the management of the patient with an acute disc protrusion should include:

1. Bed rest with short periods of ambulation.
2. Avoidance of positions or activity that can increase intradiscal pressure, especially sitting, forward bending, and the Valsalva maneuver.
3. Relaxation of reflexed muscle splinting.
4. *Specific segmental distraction techniques.*

Note that the *Cox technique is a specific intermittent distraction.* Distraction of the disc provides a push-pull pumping effect on the IVD space as the caudal section of the table is gently moved up and down during traction. This movement creates a milking action on the IVD space. *Remember that in the acute stage of a disc lesion, the patient may not tolerate traction until some of the swelling and inflammation has dissipated.*

ANOTHER OPINION ON TRACTION APPLICATION

McElhannon (274) gives four basic purposes for traction:

1. Enlargement of the intervertebral disc space.
2. Tautening of posterior longitudinal ligament to create a centripetal force on the anulus fibrosus.

3. Separation of apophyseal joints.
4. Enlargement of intervertebral foramina.

He listed 12 contraindications for traction: malignancy, cord compression, infectious disease, osteoporosis, hypertension or cardiovascular disease, rheumatoid arthritis, old age, pregnancy, active peptic ulcer, hiatal hernia, aortic aneurysm, or hemorrhoids.

The "rule of three" is advocated by McElhannon. It says that the patient must be seen for distraction for 3 consecutive days at the beginning of care. The patient may feel some discomfort after the first session, but it should diminish on the second or third session. The lordotic curve must be flattened to distract the vertebrae. McElhannon advocates static traction for the first three sessions, to adapt the muscles and ligaments to the force, and then intermittent or kinetic traction, holding for 30 seconds and releasing for 10 seconds. Acute discs are, in his opinion, best handled by static traction until the spasm and radiculitis begin to subside; then treatment should be changed to intermittent traction.

STODDARD'S OSTEOPATHIC TECHNIQUE

Stoddard (275) has described his osteopathic technique as follows:

> "The treatment of intervertebral disc herniation should start long before it occurs. We should manipulate and mobilize osteopathic spinal lesions long before they lead to these degenerative changes and not leave them to take their course. If on examination of the spine we find areas of restricted mobility or even single lesions, our duty is to release the restricted joints and insure normality as far as is within our power.
>
> "I am of the firm opinion that a herniated disc can sometimes be replaced by manipulation, but when a true prolapse of the disc occurs, I am convinced that it is impossible to replace the nuclear material by manipulation. At that stage all that can be achieved by manipulation is the empirical attempt to shift the position of nerve root and prolapsed nuclear material so that less pressure occurs on the nerve root.
>
> "By herniation of a disc I envision a bulging of the anulus sufficient to press on and irritate the posterior longitudinal ligament and dura mater without a complete rupture of the anulus and the posterior longitudinal ligament. If there is sufficient outer annular fibers and posterior ligaments to hold the herniation from protruding right through them I think it ought to be possible to reposition the nuclear material—not that such a state of affairs is desirable, it is a highly vulnerable condition—but clinically at least such cases are rewarding in that the patient obtains a dramatic relief of symptoms. Even though at a later date he may well have a relapse. After all, a track has been formed in the circular fibers of the anulus and such a tract does not repair well, if at all, because cartilage once torn is not repaired with cartilage but merely with fibrous tissue. At best we can hope for fibrous tissue repair and provide additional support either by improving the muscles surrounding the joint or by using artificial external supports.
>
> "When nuclear material has escaped into the spinal canal and has become wedged between the nerve root and the intervertebral foramen, manipulation can sometimes alter the site of pressure or shift the prolapsed material to another site where there is less irritation of the nerve roots. If the technics are designed to achieve this and they are sufficiently gentle to avoid further damage, they are well worth attempting because in roughly half of the cases the attempts succeed. If the attempts are successful, the patient has still to observe caution; the hope is that the prolapsed material will in time shrink and cause less trouble. In the meantime a laminectomy has been avoided. If the attempt is unsuccessful and the technic is designed to avoid further damage, the patient is no worse off and, if necessary, can still take advantage of surgical procedures" (275).[a]

Stoddard's guide to the prognosis of manipulation on the disc lesion is based on straight leg raising tests. If the test is positive at 30° or less, the prospects of success are distinctly limited. The smaller the angle, the less likely is manipulation to be successful, and the lower the level of disc lesion, the less chance of success. A probable reason for this observation is that the lowest intervertebral foramen has the smallest hole and the largest nerve root. Therefore, less opportunity presents for maneuver and alternation of position.

Given a patient with a disc prolapse at the L4–L5 level, and an SLR that is positive at 45°, the chances of success by manipulation are more than 50%, success not meaning complete relief of pain but a substantial reduction of pain and a reduction of physical signs.

Stoddard's technique of stretching the sciatic nerve involves placing the patient on the side while stretching the lower extremity over the side of the table. The idea is to make sure that the articular facets are at least mobile and that adhesions are released on the nonpainful side. The technique is applied on both sides. Stoddard believes that this procedure alters the position of the nerve root and the prolapsed disc. According to him, during application of the flexion and extension technique with use of the McManis table, "*the lower leaf of the table ought not be pressed far down into too much flexion in case gapping of the joint causes a herniation of the disc*" (275) (italics added).

The technique is useful on both the thoracic and the lumbar spine, but patient cooperation and ability to grip the top of the table firmly and yet relax the spine must be relied on. Such controlled relaxation is not easy by any means but it should be possible for the average cooperative patient.

A combination of movements can be obtained by using the lower leaf of the McManis table to open to two of its ranges, but such combined movements are complex, not easy to control, and rarely indicated anyway. The goal is to place the pivot of movement just below the level of the lesion and, while articulating all levels of the lower thoracic and lumbar joints, to pay special attention to those joints at which there is a *restricted range of movement*.

[a] From Stoddard A. A Manual of Osteopathic Technic. New York: Harper & Row, 1969.

DUAL DERMATOME SCIATICA TREATMENT

According to most authorities, 90% of all disc lesions occur at one level and involve one nerve root. For that other 10%, the following discussion is presented (Figs. 12.45 to 12.47).

Figure 12.45 shows the fourth lumbar disc compression of the fifth nerve root.

Figure 12.46 shows the fourth lumbar disc compressing the fourth lumbar nerve root. This unusual presentation has occurred in two patients who required surgery for repair, which I documented. A differential diagnosis I have encountered in practice is that, at about 15° SLR in a patient with disc protrusion into the intervertebral foramen, the entire pelvis lifts off the table instead of flexion occurring at the hip as in normal patients (Cox sign).

It is possible for a large disc protrusion to compress two nerve roots: both the one exiting at its intervertebral level as well as the nerve root originating at its level to exit at the foramen one level below (Fig. 12.47). The indication for the disc protrusion demonstrated in Figure 12.47 would be a patient who has nerve root dysesthesias in two dermatomes of the same extremity. This would indicate either two disc protrusions or a prolapse, such as is demonstrated here, impinging on two nerve roots at one level. If the former were the case, treatment of both disc protrusions would result in a closed reduction of both discs. This would afford relief even if it were not known whether one or two disc protrusions were involved. Keep in mind that if the patient failed to show 50% relief in 3 weeks of conservative care, a neurosurgical evaluation would be sought. This would then allow discovery of such an unusual situation. An attending physician's duty is to be aware of this clinical possibility.

A difficult diagnostic situation is encountered when, during treatment of a third lumbar disc protrusion, an L4 nerve root dermatome pattern is found. If no response is found in treating the third lumbar disc, keep in mind the possibility of an L4 disc fragment into the L4--L5 intervertebral foramen. My opinion is that intervertebral foramen free fragment encroachment by disc prolapse is often a surgical case.

How Long Should Conservative Care Be Administered Before Surgery Is An Option?

In the absence of progressive neurologic, motor, or bowel and bladder dysfunction, conservative therapy can be continued because no significant difference in recovery of function has been reported between patients whose herniated discs resolved spontaneously and those whose herniated discs were surgically removed (277).

Only 5 to 10% of patients with radicular pain require surgery. Surgery should be considered if symptoms have not been significantly alleviated after 6 weeks of conservative therapy.

Although intractable back and radicular pain are indications for surgery, the operation can be deferred for longer than 6 weeks if the patient prefers. The length of time elapsed without relief, however, is probably the one best indication that the pain will not remit without surgery.

Patients should be advised that the prognosis in disc herniation is good, with a 90 to 95% recovery rate for conservative therapy alone, and that permanent disability is rare (277).

It is commonly accepted that in the treatment of patients suffering from symptoms of herniated nucleus pulposus (lumbar disc lesion), conservative management should be tried before resorting to surgical procedure. The danger of surgical complications, the certainty that laminectomy will damage spine stability, and the occasional failure of surgical procedures to relieve symptoms indicates the advisability of an initial trial of conservative treatment (278).

Differentiation of Supine and Prone Distraction Benefits

Less lumbar sacrospinalis muscle activity is recorded during traction in the prone position than during traction in the supine position (279).

Cervical Spine Effects on the Lumbar Spine

Cervical spinal manipulation can have significant effects on the tone of the lumbopelvic musculature, presumably by facilitating tonic neck reflexes involving intersegmental spinal pathways (280).

Lumbar disc herniation frequently coexists with cervical disc disease. A recent retrospective study of 200 patients who had cervical discectomy found that 31% required lumbar disc surgery (277).

Transcutaneous Electrical Stimulation

Randomized studies show transcutaneous electrical stimulation (TENS) units reduce both the sensory-discriminative and motivational-affective components of low back pain in the short term but that much of the reduction in the affective component may be a placebo effect. TENS should be used as a short-term analgesic procedure in a multidisciplinary program for low back pain rather than as an exclusive or long-term treatment (281).

Return to Work Decision-Making of Lancourt

This is described in Chapter 9 under "Low Back Wellness School Principles" and you are referred to it to determine patient preparedness to return to work.

Chronic Low Back Pain Patients Do Not Have Restricted Lumbar Flexion

Lumbar flexion was not reduced in chronic low back pain patients. This may explain some of the current thought casting doubt on the presence of any true anatomic or structural impairment in chronic low back pain patients (282).

TREATMENT OF THE INTERVERTEBRAL DISC PROTRUSION PATIENT

Chapter 9 outlines the care of the intervertebral disc herniation patient under Protocol I. The chapter also covers the pre and postmanipulative care of the patient with disc herniation to include physiologic therapeutics of galvanism and tetanizing currents, nutrition, exercise, low back wellness school, home care, bracing, and return to work principles.

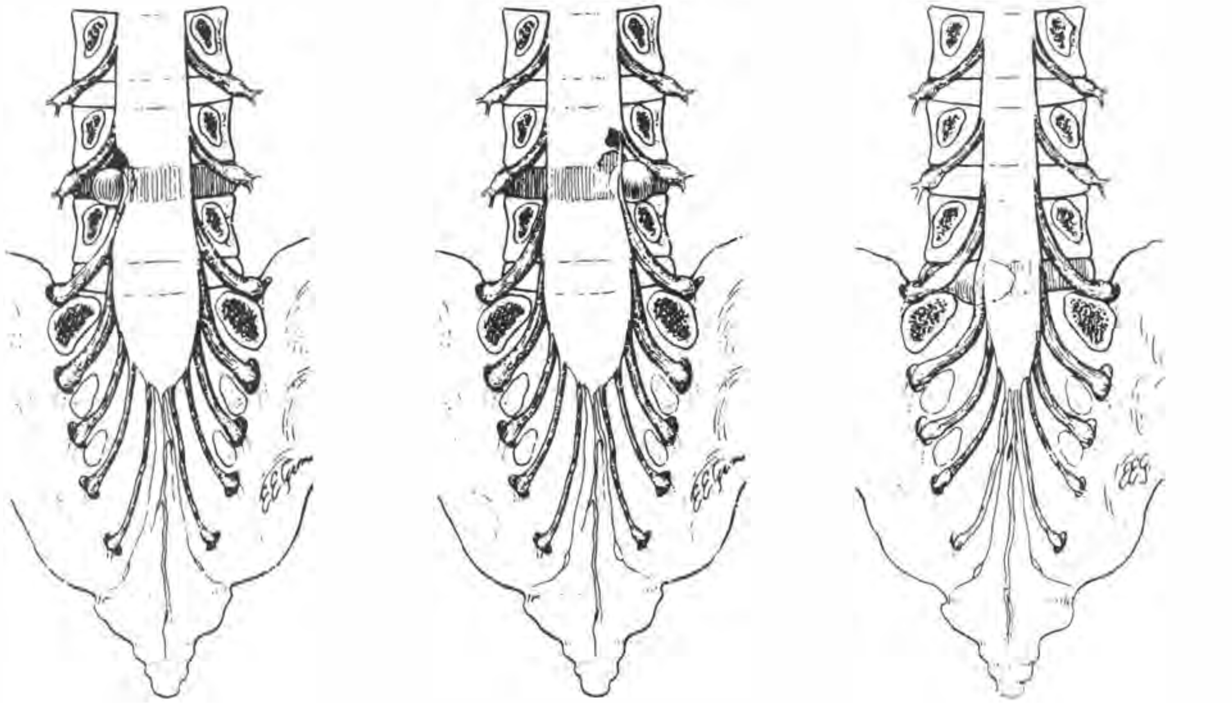


Figure 12.45. **Above left.** Usual relationship of fourth lumbar disc compression of L5 nerve root. The schematic illustration of the disc hernia pushing the L5 nerve root aside medially was predicted analytically and verified at operation. This patient had suffered from an intermittent, extremely painful left-sided L5 syndrome for 18 years. During periods of serious pain, the patient walked with a definite lean toward the right. Although the pain had been longstanding, the only nerve root involved in this overall picture of the disease was the left L5 nerve root. A year earlier, the extensor paresis had become severely involved. Before that time, the extensor paresis had been variable and involved to a moderate degree. This patient had surgery previously in which the fourth lumbar disc was exposed, but no hernia was found.

In this patient, the unnatural lean to the right indicated that the L5 nerve root was pushed medially. The patient recovered rapidly and became free of pain as he gradually regained satisfactory extensor muscle function. (Reprinted with permission from Herlin L. Sciatic and Pelvic Pain Due to Lumbosacral Nerve Root Compression. Springfield, IL: Charles C Thomas, 1966:42.)

Figure 12.46. **Above middle.** Unusual lateral position of the disc prolapse extending into the intervertebral foramen to compress the L4 nerve root. This 32-year-old woman developed an acute attack of right-sided L5 syndrome 3 years prior to seeing me. Six months prior to admission, she had experienced an acute recurrence of the right-sided L5 syndrome with severe pain and extensor paresis.

During examination, the L5 syndrome was confirmed, but slight symptoms and signs from L4 were also noticed as minor radiating pain on the anterior side of the thigh. In addition, pain occurred during palpation over the muscular attachments of the adductor muscles. The Lasègue sign was positive, at a low angle, for L5. The knee jerk was normal. No sciatic scoliosis was apparent. When the patient bent to the right, distinct pain in the L5 distribution area was elicited; less pain was provoked by bending to the left. *Myelography was negative.* The diagnosis indicated a nerve root compression by a lateral disc protrusion on the right side in the fourth lumbar disc. This condition exerted a slight compression on L4 and severe compression together with a slight medial displacement on L5.

At surgery, the fourth disc level was explored, and the L5 nerve root was displaced a little medially by the disc lesion. The intervertebral joint was resected to explore the L4 nerve root exiting the cauda equina through the intervertebral foramen. A major portion of the disc protrusion had been hidden by the intervertebral joint. Also found was the cranially displaced fragment of the nucleus pulposus that had pushed its way from the cavity and became lodged under the posterior longitudinal ligament of the spinal canal. It produced a sharp-angled cone that pinched the L5 nerve root at its angle of departure from the cauda equina.

The patient was immediately free of pain, and the extensor power returned quickly. (Reprinted with permission from Herlin L. Sciatic and Pelvic Pain due to Lumbosacral Nerve Root Compression. Springfield, IL: Charles C Thomas, 1966:42.)

Figure 12.47. **Above right.** L5–S1 disc protrusion prolapse compressing both the L5 nerve root at the intervertebral foramen and the S1 nerve root at its origin at the cauda equina.

This schematic is of a 32-year-old housewife who had had two children and who had a history of intermittent low back pain and lumbago for several years. She had suffered two spontaneous miscarriages, the latest 4 months prior to admission, which had been immediately followed by the onset of left-sided sciatica.

Examination indicated mixed nerve root syndromes of a painless lateral L5 syndrome, an ordinary dominant S1 syndrome, and a left-sided S2 syndrome. The left S3 was also involved. Left S2 pain could be provoked at palpation over the inguinal region, the tuber ischii, the medial part of the fossa poplitea, and medially over the soleus muscle of the calf. S3 pain was provoked over the symphysis and the most median part of the gluteal musculature. No obvious scoliosis was seen.

Diagnosis was a large hernia in the fifth lumbar disc extending from the left lateral to the median line with its maximal bulk where the S1 nerve root runs over the disc. Surgery confirmed the presence of a large disc hernia. The disc was evacuated and recovery was excellent.

Comment: The two miscarriages appear to have resulted from a lower sacral nerve root compression that caused the onset of the lumbago, with the sciatica developing later.

Herlin (276) documents other urogenital diseases caused by lumbosacral nerve root compression. On page 79, he discusses a situation he had encountered in which severe pelvic pain and urogenital infection might be caused by a factor similar to that of sciatica-nerve root compressions from the outside because of different types of disc degeneration. He believes that the possibility exists that whole pelvic diseased states depend on multiple nerve root compressions of the S2 and the lower sacral nerve roots. Surgical relief of lumbosacral nerve roots resulted in normalization of diseases such as salpingitis, painful irregular menstruations, vaginal discharge, sluggish frequent urination, cystitis, prostatis, urethritis, infertility, impotency, and vertigo. (Reprinted with permission from Herlin L. Sciatic and Pelvic Pain Due to Lumbosacral Nerve Root Compression. Springfield, IL: Charles C Thomas, 1966:42.)

PRESENTATION OF DISC HERNIATION PATIENTS FROM THE AUTHOR'S CLINICAL PRACTICE

L5–S1 Disc Herniation

Case 8

Figures 12.48 and 12.49 show a right L5–S1 paracentral disc herniation. This patient had surgery with a good clinical outcome.

L3–L4 Disc Herniation

Case 9

Figures 12.50 and 12.51 show an L3–L4 left paracentral to posterolateral disc herniation that contacts the thecal sac and the lateral recess where the L4 nerve root exits. This case involved is a 31-year-old chiropractor with low back pain and left anterior thigh numbness that started after a lifting incident.

Distraction adjustment was given at the L3–L4 disc followed by positive galvanic treatment of the disc and exiting lateral recess. Tetanizing current was applied to the paravertebral muscles. Exercises of stretching the hamstring muscles, knee chest flexion exercise, abdominal strengthening, and stretching of the abductor and adductor muscles were performed. Complete remission was obtained.

L4–L5 Synovial Cyst and Disc Herniation

Case 10

Figures 12.52 to 12.54 show advanced L5–S1 degenerative disc disease with an L4–L5 disc herniation. A synovial cyst is seen of the left L4–L5 facet joint, which accompanies facet arthrosis. The coronal image reveals T11–T12 disc herniation anteriorly and posteriorly. This case had a successful outcome with distraction adjustment of the L4–L5 level and positive galvanic current into the left facet articulation.

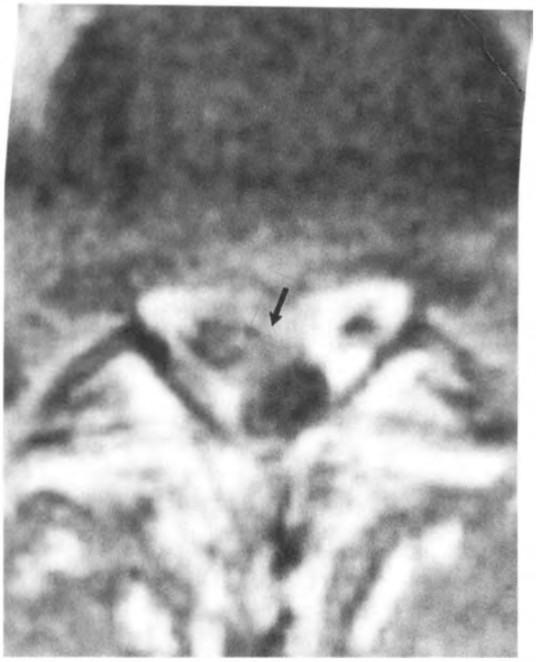


Figure 12.48. A large right paracentral disc herniation (*arrow*) at the L5–S1 level that contacts the thecal sac and surrounds the right S1 nerve root.

Figure 12.49. Sagittal magnetic resonance imaging shows the L5–S1 disc protrusion with loss of signal intensity at the L4 and L5 disc levels caused by degenerative changes.

Dual Disc Herniations

Case 11

This was a case of bilateral sciatica with two-level disc herniation. Figure 12.55 shows a left L4–L5 posterolateral disc herniation and Figure 12.56 a right L5–S1 herniation. Figure 12.57 shows Schmorl's nodes throughout the lumbar spine and Figure 12.58 shows the large L4–L5 and L5–S1 herniations in sagittal image.

This patient had foot drop in the left leg and weakness of the right gluteus maximus and hamstring muscles with a diminished Achilles reflex on the right. Bilateral sciatica, left L5 and right S1, was present. Distraction adjustments saw complete return of motor strength and relief of pain.

An interesting point in this case is that had the two discs herniated on the same side, resulting in unilateral L5 and S1 nerve changes, the outcome would have been much more in question, I feel. The reason is that two nerve root involvement on one side does not permit the axon sprouting to allow one nerve root to assist in rehabilitating the lost nerve function. This results in more permanent disability.

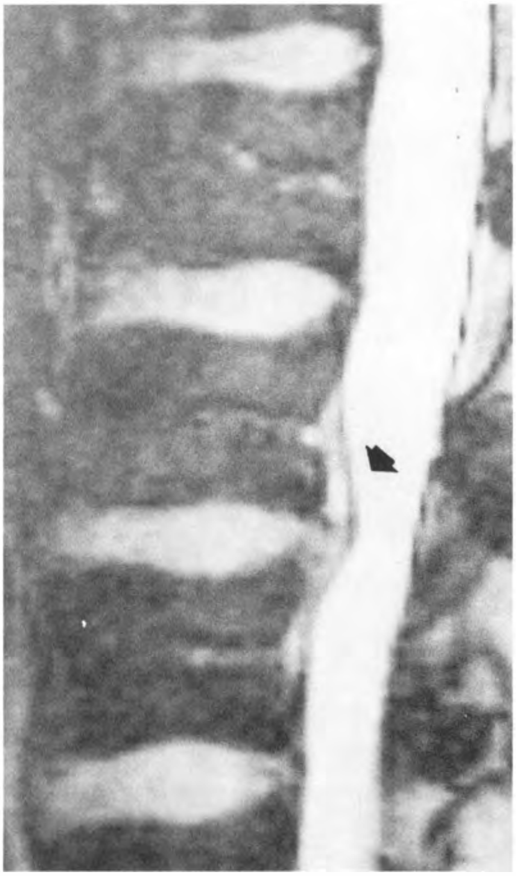


Figure 12.50. Sagittal magnetic resonance imaging shows the L3–L4 disc herniation with the outline of the posterior longitudinal ligament. (*arrowhead*).

Figure 12.51. Axial magnetic resonance imaging shows the left posterior lateral L3–L4 disc herniation (*arrowhead*) that contacts the thecal sac and narrows the foramen.

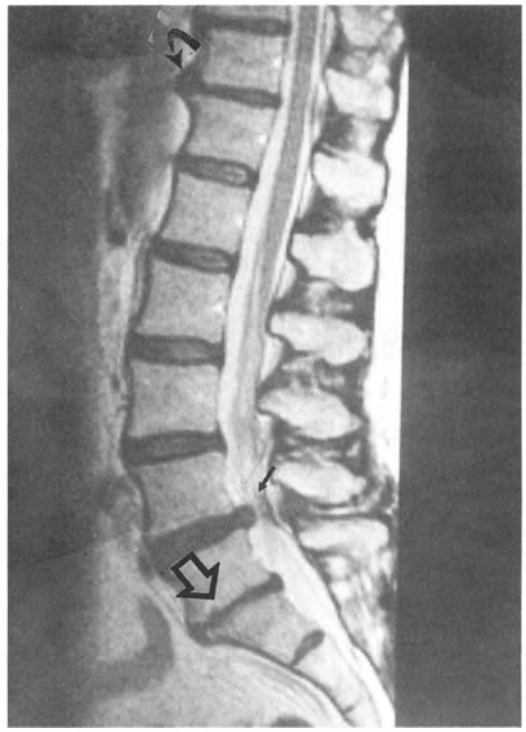


Figure 12.52. Extensive L5–S1 degenerative disc disease is seen (*open arrow*) with an L4–L5 disc protrusion (*curved arrow*). Also note the T11–T12 anterior disc herniation.

Figure 12.53. A synovial cyst is seen at the left anterior facet joint (*arrow*).

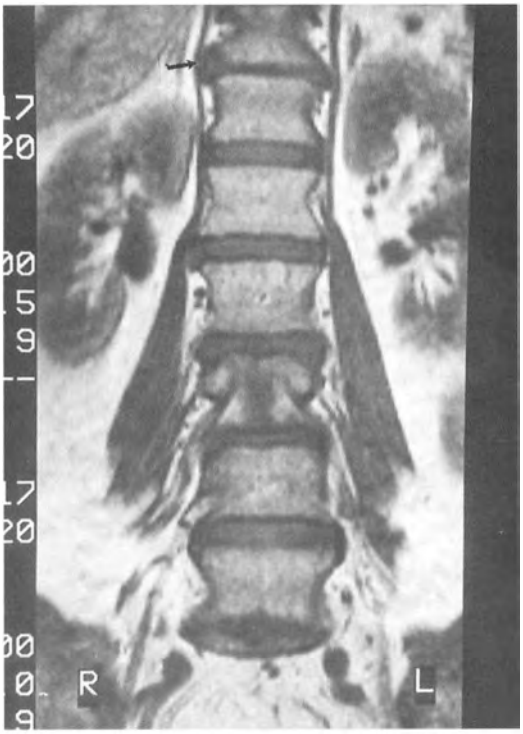


Figure 12.54. Coronal section shows the T11–T12 disc herniations (*arrow*).

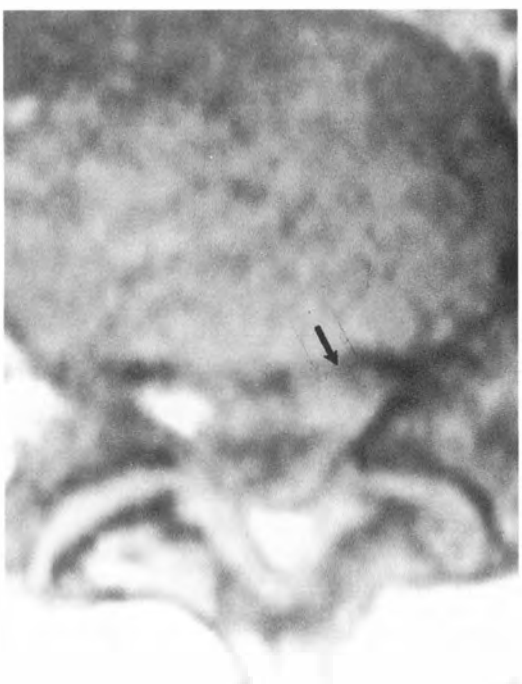


Figure 12.55. *Axial magnetic resonance imaging shows a left L4–L5* disc herniation (*arrow*) that occludes the lateral recess.

Figure 12.56. Axial magnetic resonance imaging shows a right L5–S1 disc herniation (*arrow*) that occluded the right lateral recess.

Figure 12.57. Sagittal views show multilevel Schmorl's nodes and anterior disc invaginations of the end plates resulting in somewhat trapezoid-shaped vertebrae.

Figure 12.58. Sagittal magnetic resonance image showing L4–L5 and L5–S1 degenerative disc changes with large disc herniations at both levels (*arrows*).

L5–S1 Sequestered Disc Fragment

Case 12

Figures 12.59 and 12.60 show a large free fragment in the right lateral recess and central canal at the L5-S1 level. Note the disc herniation fragment completely obliterates the right S1 nerve root (*arrow*). This large disc fragment responded to distraction adjustment with relief.

L5–S1 Disc Herniation

Case 13

Figures 12.61 and 12.62 reveal a right L5–S1 disc herniation in a 45-year-old woman who developed right first sacral dermatome pain following an attempted manipulation of her low back by her husband. Distraction adjusting of the L5–S1 disc space with trigger point therapy and positive galvanism of the L5–S1 disc and S1 nerve root yielded 90% relief within 3 weeks of care.

L4–L5 Large Disc Herniation

Case 14

Figure 12.63 shows a large L4–L5 disc herniation on sagittal MRI in a 32-year-old man with both L5 and S1 left lower extremity pain radiating to the foot and toes. He was originally treated with medications for a diagnosis of inflammation of his tailbone. Left plantar flexion and great toe flexion were grade 4/5 with a diminished left Achilles reflex. Deep tendon reflexes were 2/2 in the remaining lower extremity. Hypesthesia of the left L5 and S1 dermatomes was noted.

At 3 weeks of distraction adjustments, 50% relief of the left lower extremity pain was attained. A neurosurgical opinion found a good response to chiropractic distraction adjusting and care continued with complete pain remission and return of motor power.

L5–S1 Large Disc Fragment

Case 15

Figures 12.64 and 12.65 show a large right free fragment of L5–S1 disc that occludes the right lateral recess and extends behind the first sacral body.

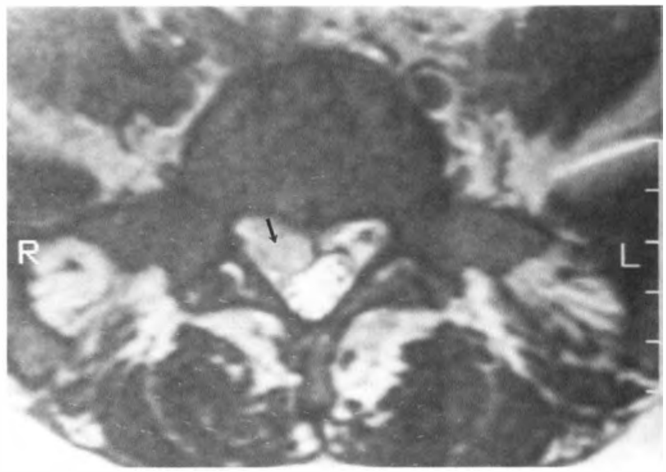


Figure 12.59. Axial image shows a large right disc fragment lying within the lateral recess and central canal (*arrow*), which totally obliterates the visualization of the right S1 nerve root. (Case is given by David Puentes, DC.)

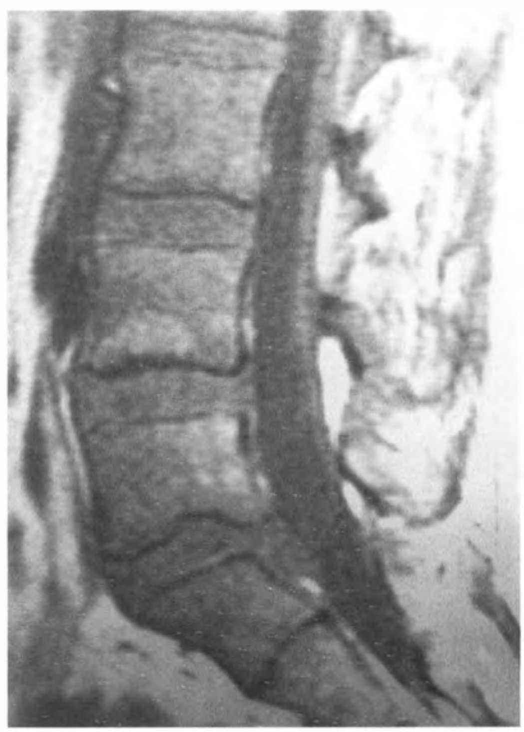


Figure 12.60. Sagittal view shows the fragment seen in Figure 12.59 lying posterior to the first sacral segment (*arrow*). (Case is given by David Puentes, DC.)

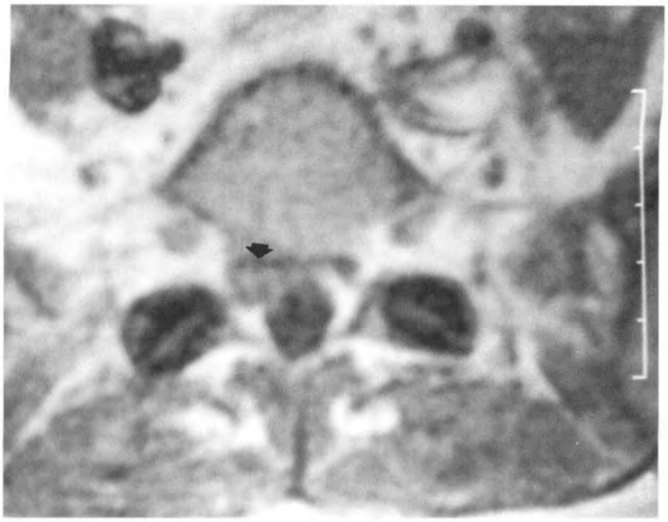


Figure 12.61. Axial image shows a large right L5–S1 disc prolapse that denies visualization of the right S1 nerve root.

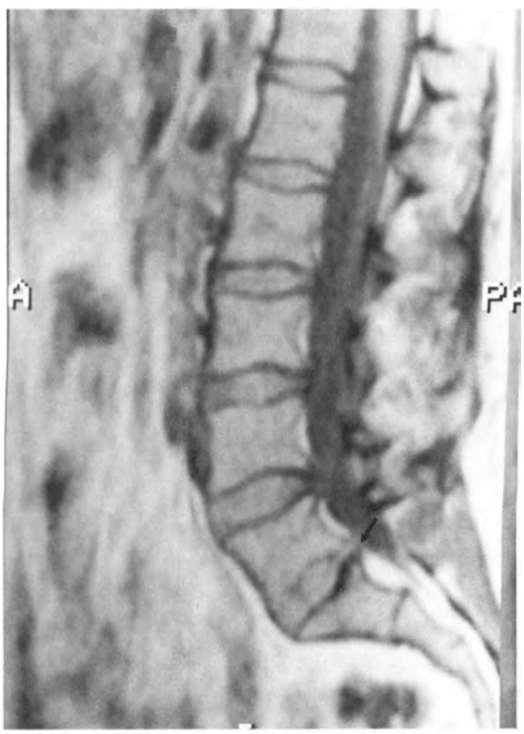


Figure 12.62. Sagittal section shows the L5–S1 disc protrusion (*arrow*).

Figure 12.63. Sagittal magnetic resonance imaging shows a large L4–L5 disc protrusion (*arrow*).

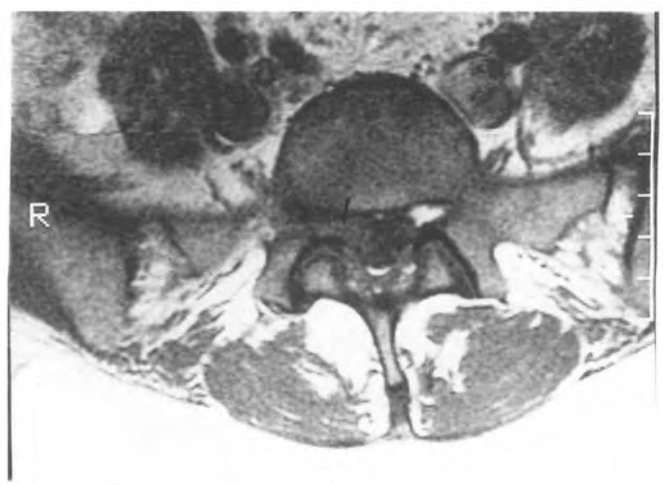


Figure 12.64. Axial magnetic resonance imaging shows the large free fragment of L5–S1 disc material within the lateral recess (*arrow*).

Figure 12.65. Sagittal image shows the fragment to be lodged behind the first sacral body.

This 31-year-old man was told by two surgical groups to have surgery, but he wants nonsurgical care. He is treated with the understanding that 50% relief must be attained within 1 month of care or surgery will be performed. At initial examination, the right SLR was positive at 35°, ranges of motion at the thoracolumbar spine were limited to 60° flexion, 20° extension, and 10° lateral flexion. The ankle jerk was absent on the right side and hypesthesia of the right S1 dermatome was noted on pinwheel examination.

Treatment consisted of distraction adjustments, positive galvanism into the L5–S1 disc space with heat, and tetanizing current to the paravertebral muscles while ice was applied to the spine, exercises of knee chest and pelvic tilt, and home care of alternating hot and cold fomentations to the low back followed by massage and the wearing of a lumbosacral brace. Slow steady relief occurred until the patient was back to work after 1 month of care. The Oswestry Pain index went from 40% when first seen to zero in 6 weeks. The VAS went from initial 7 to 1. The patient attended low back wellness school to learn proper ergonomics for his spine. Complete remission of the symptoms occurred.

L4-L5 Disc Prolapse

Case 16

The following case is presented from the practice of Charles C. Neault, DC, of Simi Valley, California. It is a case report in which a diagnosed L4–L5 lumbar nuclear prolapse, verified by MRI and treated with Cox distraction manipulation, was managed successfully and the reduction verified with a post-treatment MRI scan.

A 58-year-old woman presented complaining of low back and left leg pain of 1.5 weeks duration following moving a couch in her home. She indicated that she had some minor back problems previously, for example, when doing gardening. The pain usually lasted only a day or two and would go away. On this occasion, not only did the low back pain not go away, but she had left leg pain, which she had never had previously.

On examination, the patient was in severe distress. She weighed 180 pounds and was 5 feet, 9 inches tall.

She indicated that her low back pain was not aggravated by coughing or sneezing; however, the pain occasionally was worse on sitting. She also indicated that her leg pain appeared simultaneously with the back pain, and that it went all the way to the foot. The patient exhibited a left limp, a left antalgic lean, and severe paravertebral muscle spasm.

Bechterew's test was normal. Minor's sign was positive on the left, and the Valsalva maneuver was negative. The Neri's bow test and Lewin's standing tests were normal. Palpation revealed pain and tenderness on the spinous process of L4 with percussion positive. There was loss of lumbar lordosis. Range of motion was limited to 60° flexion with pain. Other motions were normal. Kemp's sign was positive on the left, with the right normal. Heel and toe walk were normal. Straight leg raise sign was left positive at 45° with a negative well leg raise sign. Patrick-Fabere sign was positive on the left and negative on the right.

Muscle testing revealed weak dorsiflexion of the foot, and great toe and foot eversion on the left. Milgram's sign was positive on the left low back. The deep reflexes at the patella and heel were +2 bilaterally and were equal. Sensory dermatome testing revealed a decrease of the left L5 dermatome as well as a slight decrease of the left S1 dermatome. Circulation of the lower extremities was normal. The clinical impression following workup was an L4–L5 left subrhizal nuclear prolapse.

An initial MRI–CT combined study was made. It showed a degenerative L4–L5 disc and a posterior central and left-sided disc herniation measuring 6 mm (Figs. 12.66 and 12.67). Also seen was a free sequestered fragment of disc material posterior to the L5 vertebral body, measuring 5 mm by over 1 cm in height. This had escaped from the L4–L5 space and migrated caudally behind the L5 body.

Treatment originally was for 2 weeks on the basis of daily care as the patient continued to work. On the day of the MRI–CT report, she was placed on disability and was treated twice a day for 9 days, at which time her pain decreased by 80% subjectively, and the SLRs were negative bilaterally. Prior to stopping work, she still had low back pain and a positive SLR of 65°.

At the end of the ninth day of treating the patient twice daily while she was off work, she complained of only occasional leg numbness and tingling, which was felt to be compatible with residual healing of the disc lesions. The patient returned to work 1 month later and was totally asymptomatic. No neurologic or orthopaedic findings of a positive nature could be elicited from the patient at that time.

Follow-up MRI and CT scans (Figs. 12.68 and 12.69) were made 21 days following the initial study. The radiologist reported that the size of the sequestered fragment had decreased significantly since the initial study shown in Figures 12.61 and 12.62. In addition, disc space height increased at L4–L5. Not only had the free fragment decreased in size, but also the L4–L5 posterior herniation had decreased as well, as shown on CT scan.

This was a severely sequestered disc fragment, and the patient had been told by a neurosurgeon to have surgery for its removal. Seeking chiropractic care resulted in Cox closed reduction distraction manipulation being administered, which enabled the patient to get well and resume her activities full time with no partial disabilities.

Although a disc bulge and a small sequestered fragment were present on the second MRI, it certainly indicated and provided concrete proof that the size of a patient's spinal canal is more important than the size of the disc lesion, which must certainly be

Figure 12.66. Sagittal (**A**) and axial (**B**) section magnetic resonance image prior to manipulation shows decreased signal of the L4-L5 disc, indicating a degenerating disc. The L4–L5 disc extends 6 mm beyond the vertebral body end plate and compromises the cauda equina. A large free fragment of sequestered disc is seen to lie posterior to the L5 vertebral body, measuring 5 mm in diameter sagittally and more than 1 cm craniocaudally (*straight arrow*) on the sagittal and on the axial view (*curved arrow*).

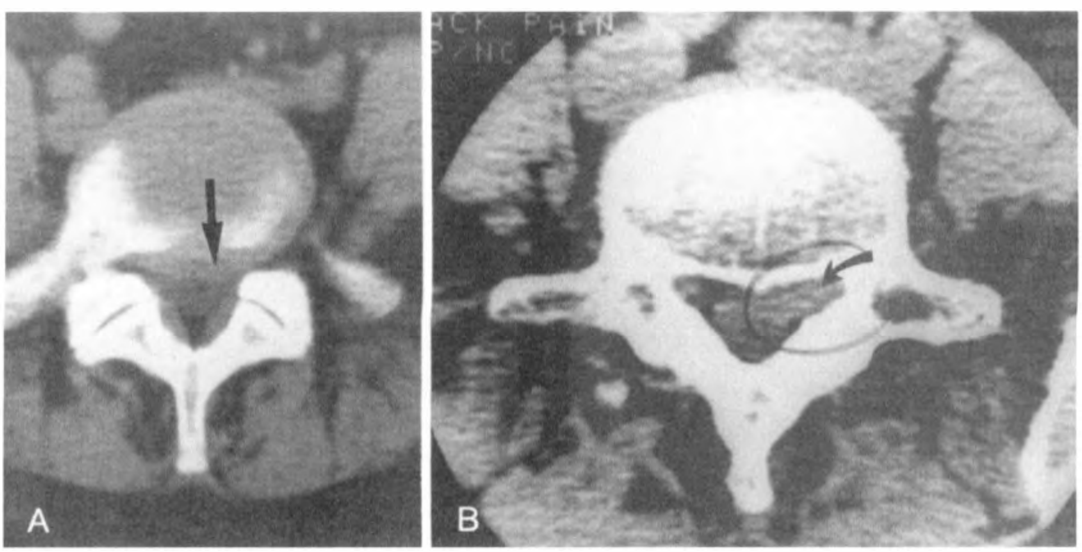


Figure 12.67. **A.** Computed tomography scan shows the large, left central disc protrusion (*straight arrow*). **B.** Disc protrusion seen compromising the left lateral recess and thecal sac (*curved arrow*).

Figure 12.68. Repeat magnetic resonance imaging, performed 21 days after the initial study shown in Figure 12.66, shows the disc sequestered fragment to be significantly smaller (*arrow*), and the patient now has no symptoms of low back or left leg pain.

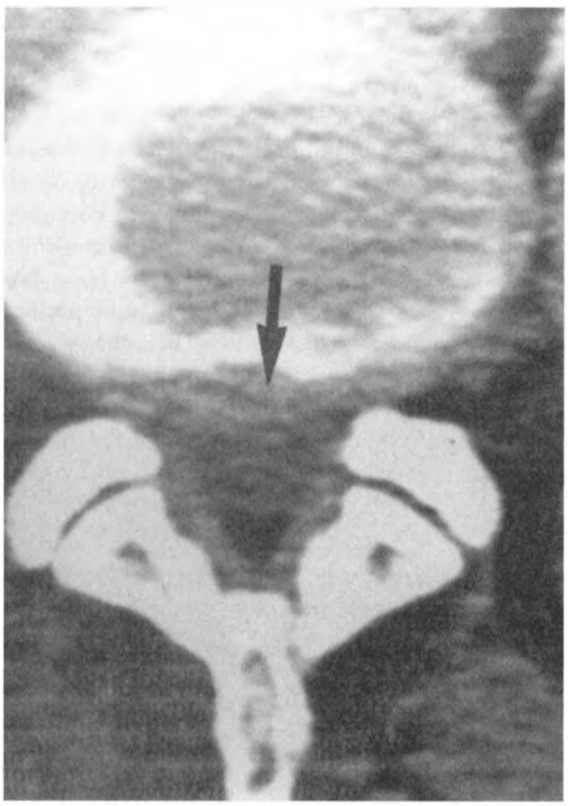


Figure 12.69. Repeat computed tomography scan also shows marked reduction of the disc protrusion (*arrows*) as seen in Figure 12.67.

taken into consideration when a sequestered fragment or a disc bulge is reported.

This was a unique case with "before and after" scans that demonstrated the possible effectiveness of flexion distraction as applied and taught in this textbook. In the past, patients with fairly large disc herniations and ruptures of this nature had to resort to either conventional methods of chiropractic manipulative therapy or open surgery for relief of pain.

L4–L5, L5–S1 Disc Degeneration and Facet-Generated Foraminal Stenosis

Case 17

A 40-year-old woman with chronic low back pain and right knee pain was seen; her MRI studies are shown in Figures 12.70 to 12.72. Distraction manipulation was given. The result, following 3 weeks of care, was absence of leg pain and isolation of pain to the right L5–S1 facet articulations. Treatment then consisted of full range of motion to the facet joints of the lumbar spine, with a vigorous home exercise program of stretching and Cox exercises. The patient was left with right L5–S1 facet joint pain on prolonged sitting, bending, lifting, or twisting movements of the waist. She attended low back wellness school to learn ergonomic control of her low back pain.

This is an excellent case of a patient with an unstable disc at L4–L5 with anular tearing and herniation, as well as vertical stenosis by facet imbrication of the L5 superior facet into the upper aspects of the L4–L5 intervertebral foramen where the L4 nerve root exits the cauda equina and vertebral canal. This patient must maintain constant care in using her low back to prevent more serious disc damage and nerve root compression irritation, which could necessitate surgery.

LUMBAR SPINE TREATMENT ENDING

This chapter on low back treatment concludes with an exciting dissection performed by Chae-Song Ro, MD, PhD, of the anatomy department of the National College of Chiropractic, followed by an algorithm of treatment selection dependent on

Figure 12.70. Magnetic resonance imaging sagittal study shows posterior bulging of the L4–L5 and L5–S1 discs, with degenerative changes of the discs noted (*arrows*).

Figure 12.71. Note the foraminal narrowing (*arrow*) at the L4–L5 level and the compromised space for the L4 nerve root.

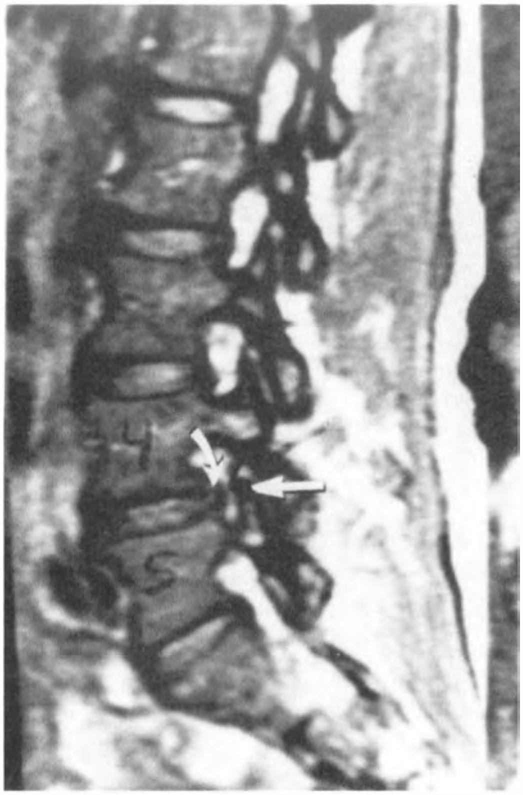


Figure 12.72. Observe the L4–L5 disc protrusion (*curved arrow*) and the vertical stenosis of the L4–L5 foramen by the telescoping of the L5 facet into the upper foraminal space (*straight arrow*).

patient objective and subjective findings. I conclude with this beautiful dissection (Figs. 12.73 and 12.74) because it so well capsulizes the probable pain pathways of the lumbar spine. It shows the spinal nerve within the intervertebral foramen and its divisions into the dorsal and ventral ramus. The dorsal ramus will supply the multifidi, sacrospinalis, aponeurosis of the latissimus dorsi, iliac crest, and buttock as cutaneous nerves (cluneal nerves L1, L2, L3), and the articular processes. The ventral ramus of the lumbar, sacral, and coccygeal nerves will form the lumbosacral plexus. This plexus will form the lumbar, sacral, and pudendal plexi. The lumbar plexus will form the iliohypogastric, ilioinguinal, genitofemoral, lateral femoral cutaneous, femoral, obturator, and accessory obturator nerves. The sacral plexus will form the sciatic nerve, and the pudendal plexus will form the pudendal nerve, perineal nerve, dorsal nerve, inferior hemorrhoidal nerve, and the scrotal branches.

The communicating ramus from the sympathetic gangl ionated chain (gray ramus communicans) will join the ventral ramus, and the recurrent meningeal nerve will be formed, which gives off the nerve supply to the disc inside the vertebral canal, the posterior longitudinal ligament, the ligamentum flavum, the facet capsule, and the epidural vascular plexus of the medulla spinalis and its membranes.

The bottom line in care of the intervertebral disc patient is treatment selection and the proper chronology of such care. Physicians need to start with conservative care, being constantly aware of the changing faces of patient symptoms and findings that dictate and demand diagnostic action and treatment regimens. Table 12.1 summarizes my basic decision-making protocol in dealing daily with clinically positive low back disc cases. It is hoped that it will aid in leading you through this often demanding and complex patient syndrome of low back pain and sciatica. Table 12.2 is a flow chart summarizing treatment selection procedures based on the diagnosis of the patient's complaint. I use Tables 12.1 and 12.2 as clinical decision-making parameters in daily practice.

Thoracic Spine Herniated Disc Diagnosis and Treatment

Increasing awareness and care of thoracic spine disc herniation dictates that chiropractic care be discussed. This chapter will conclude with this condition.

Incidence and Occurrence

Thoracic disc herniation is an uncommon entity and it is difficult to diagnose. Its incidence is reported at two to three cases per thousand patients with disc protrusion or one patient per million population per annum (283); 4% incidence has also been reported (284). It accounts for 1.5 to 1.8% of all disc operations (283, 285–287). It is most common in the fourth to sixth decades of life and has a predilection for the lower thoracic levels (286).

Equal sex incidence is reported. The location is usually below the sixth thoracic level. Trauma is found in a significant percent of cases (283, 288, 289). T11–T12 level is reported as

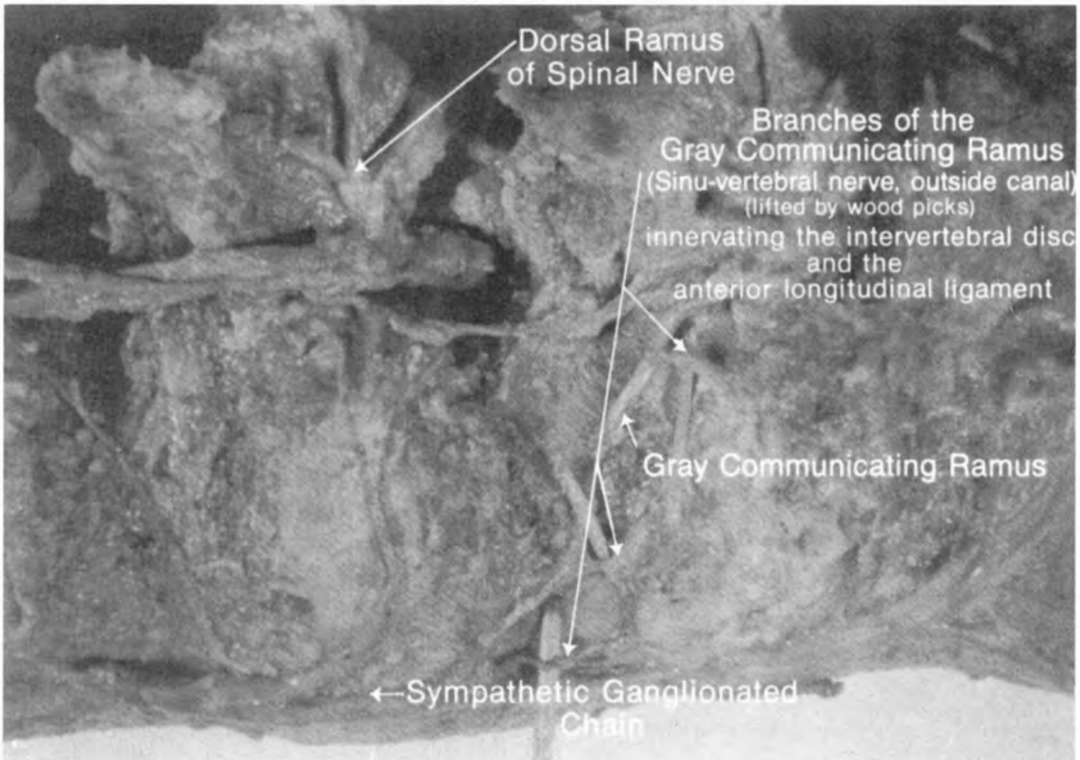
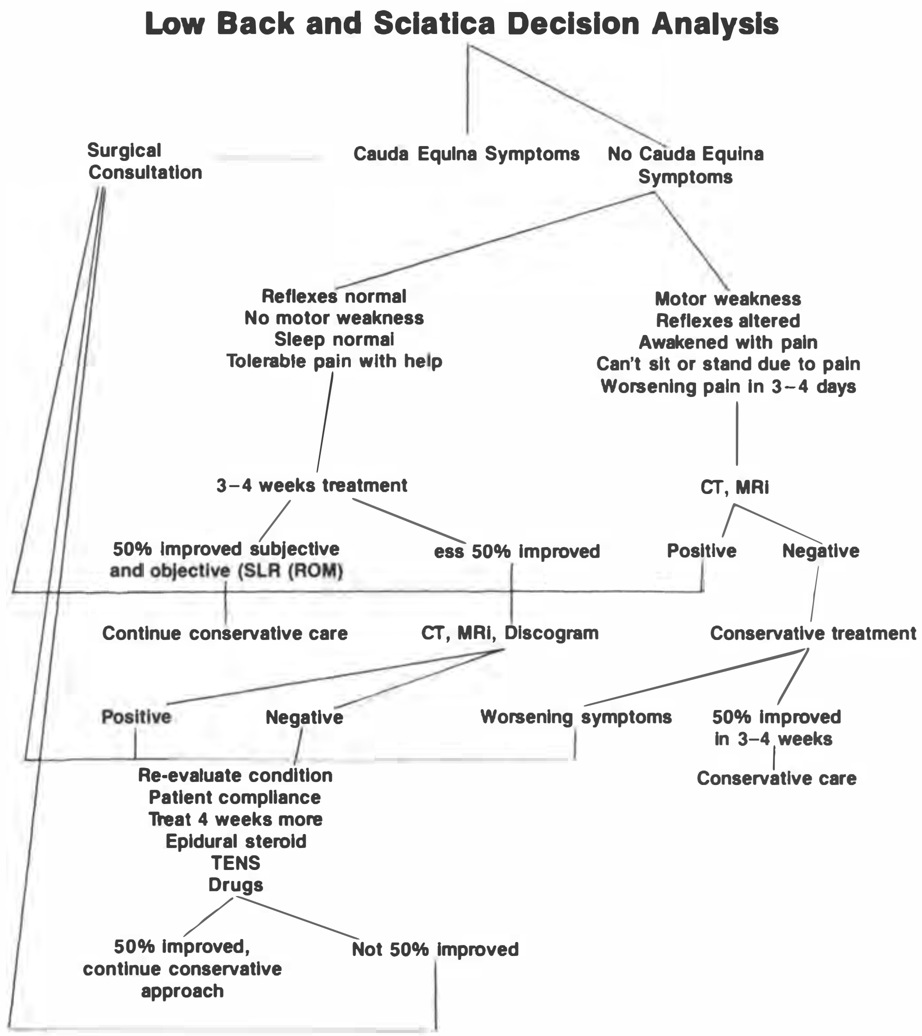


Figure 12.73. Dissection of the outside of the spine showing the sympathetic ganglionated chain giving rise to the gray rami communicantes that form the nerve supply to the circumference of the intervertebral disc and anterior longitudinal ligament. Note also the dorsal and ventral ramus of the spinal nerve.

Figure 12.74. Vertebral bodies have been carefully removed to allow visualization of the recurrent meningeal ramus. The sympathetic ganglionated chain is seen, with the gray ramus from it joining the ventral ramus to form the recurrent meningeal nerve (sinuvertebral nerve) that will enter the vertebral canal to supply the structures within it.

Table 12.1

Algorithm of Diagnosis and Treatment Protocol for Decision Making in the Sciatica Patient



CT, Computed tomography; MRI, magnetic resonance imaging; R●M, range of motion; SLR, straight leg raising; TENS, transcutaneous electrical neuromuscular stimulation.

the most common level (290). Occupations such as weight lifters and paratroopers show high incidences (291).

Torsional force is suggested as the cause of lower thoracic and upper lumbar disc degenerative changes (292).

Symptoms

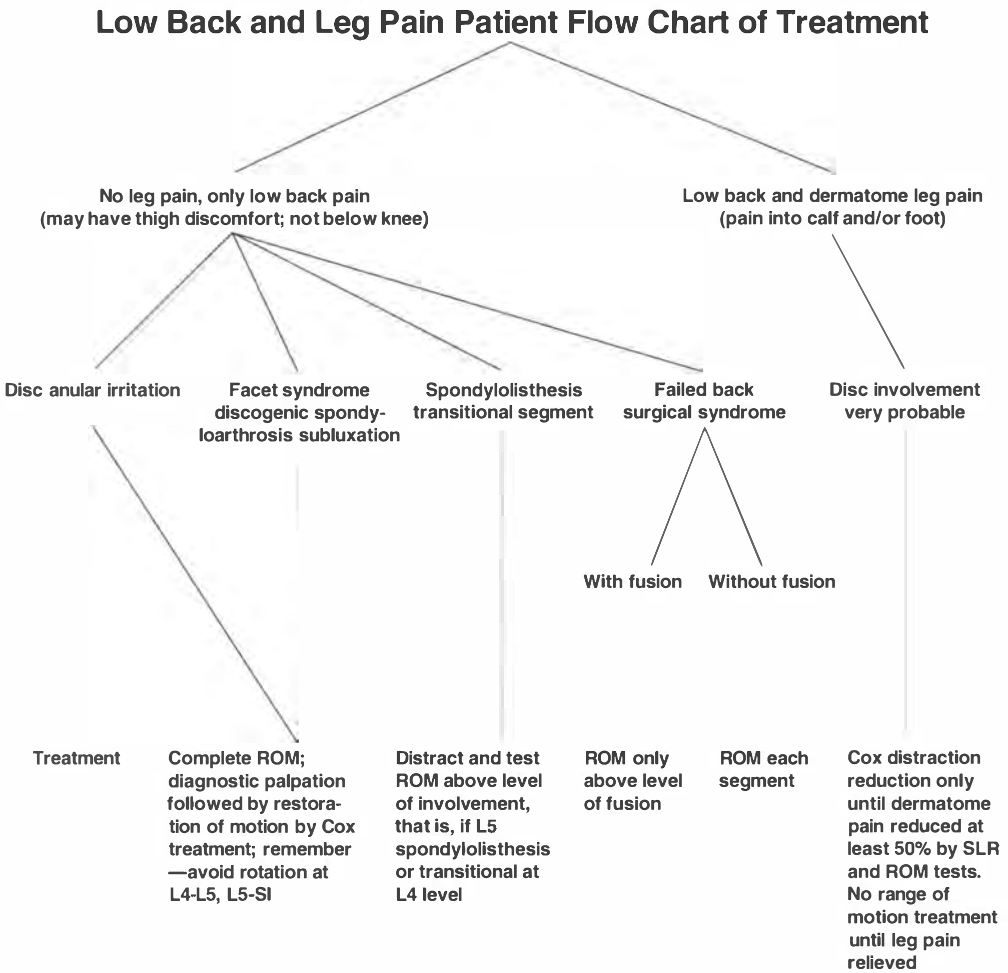
Localized or referred pain is the most frequent symptom. Brown-Sequard syndrome may be evident (293). The thoracic spinal canal is narrow and is highly susceptible to compressive and vascular insufficiency factors (284, 285, 289, 293–298). Central disc protrusion above T10–T11 is related to a high incidences of paraplegia (294). Urinary bladder dysfunction can occur later and multiple level nerve root signs can be present (295).

Obtrusive and disagreeable paraesthesia below the level of the lesion is reported in over half of cases. Motor weakness is found in all cases (293). Gait abnormalities are a key to diagnosis because of motor disturbance; sensory changes in the lower extremity, abnormal reflexes, pain and visceral referral of pain, and SLR signs are positive (295).

Drunklike staggering and difficulty walking was the chief complaint of a 27-year-old woman. Deep tendon reflex increase of the lower extremities was noted with spastic hemiparesis of the left leg. Clonus was noted. A T9 complete block on CT was found, and disc fragments and osteophytes were surgically removed. The patient developed Brown-Sequard-like syndrome postsurgically. Outcomes of surgery for tho-

Table 12.2

Flow Chart of Treatment for Low Back and Leg Pain Patients



ROM, range of motion; SLR, straight leg raising.

racic disc are not as good as for cervical or lumbar disc herniations (299).

Horner's syndrome, hand weakness, anterior chest and parascapular pain, neck and radiating upper extremity pain were the result of T1–T2 sequestration into the epidural space. Surgical removal resulted in relief. This is an unusual site for thoracic disc herniation (300).

Papilledema has been associated with thoracic disc herniation which resolved after surgical removal of the lesion. Most cases of papilledema occur with intradural spinal tumors, usually ependymomas. The increased intracranial pressure inducing the papilledema may be a hyperproteinorrachia or tumor release products effect on the arachnoid membranes. In this case of disc herniation, partial spinal block resulted in elevated protein caused by chronic epidural venous congestion because of the cord compression (290).

A patient with chronic epigastric abdominal pain attributed to chronic pancreatitis was scheduled for pancreatectomy for pain control. A herniated thoracic disc was found that was presenting as chronic pancreatitis. Chronic abdominal pain should include a suspicion of thoracic spine disc herniation in the diagnosis (301).

Multiple sclerosis coexisted in two cases of thoracic disc herniation patients, and it presents an atypical postoperative course when thoracic disc removal is done (302).

Diagnosis

As recently as 1987 myelography was reported to be the diagnostic imaging procedure of choice to diagnose thoracic spine herniated discs with only minimal documentation of CT and no reports of MRI use in diagnosis. MRI then emerged as an effective method of diagnosis and continues to be the imaging modality of choice (303).

Plain radiographs are nondiagnostic and may show degenerative disc disease (293). Discography of thoracic and thoracolumbar discs is reported to be safe and effective in evaluating dorsal pain and disc degeneration (305–307).

Two level (T6–T7 and T7–T8) herniation has been reported. This is a rare diagnosis. Trauma preceded the onset of symptoms, which were midthoracic radiating bandlike pain around the chest and occasional leg pain. Dejerine triad aggravated the pain as did deep inhalation. Enuresis, impotence, and lower extremity weakness and numbness existed. Examination showed sensory deprivation of the lower extremities, normal motor strengths, normal proprioception, negative Babinski and deep tendon reflexes. SLR was negative. Pain on palpation in the mid and lower thoracic area with range of motion restriction was noted. Plain x-rays studies were normal with enhanced CT revealing the herniations. Treatment with the posterolateral approach relieved the symptoms (293).

A 16-year-old girl became paraplegic following a headstand when severe back pain set in. MRI showed T11–T12 collapsed disc space and intraspongious disc prolapse into the T12 vertebral body. Fibrocartilaginous embolism of the spinal cord was diagnosed because of the acute vertical disc herniation into the T12 vertebral body causing increased intraosseous pressure and setting up spinal cord infarction because of the nucleus pulposus fibrocartilaginous embolism formation (308).

Calcified Discs in Children

Children have rarely been reported to show ruptured calcified thoracic discs. A 12-year-old girl presented with severe midthoracic spine pain radiating into both buttocks and anterolateral thighs. A calcified T12–L1 disc that protruded posteriorly was seen on plain x-ray study. Urinary retention developed with proprioception abnormalities, sensory deficit, and positive SLR. Myelography showed complete block at the T12–L1 disc level. A 1-cm calcified disc fragment was surgically removed and at 6 months the disc calcification had resorbed. Such central disc prolapses produce symptoms not only from compressive factors, but also possibly from thromboses of the anterior spinal artery with subsequent infarction of the spinal cord. Conservative care is urged for clinical signs of nerve root compression and surgery for cases not responding to such care unless long tract signs are present (309).

A 12-year-old boy with acute cervical and interscapular pain, torticollis, and fever showed a T3–T4 calcified disc and posterior herniation of nucleus pulposus on CT. Medication alleviated the symptoms without surgery (310).

Calcification is a rare condition in children; fewer than 130 cases have been reported (311). The nucleus pulposus is calcified and occurs most frequent between ages 5 to 10 years with male predominance (312). No cause can be found usually. Congenital abnormalities such as bilateral cataracts, infection, cardiac lesions, and bone abnormalities have been reported (313, 314).

A summary of findings of calcified disc is focal or referred pain of acute onset, painful limitation of motion, muscle spasm, tenderness and torticollis, sometimes local signs of inflammation with fever, leukocytosis and erythrocyte sedimentation rate elevation, x-ray evidence of disc calcification and protrusion. A self-limited clinical course with limitation of the calcified disc syndrome to the pediatric age patient is noted (309).

TREATMENT OF THORACIC DISC HERNIATION

Surgical approaches are not discussed in this text. Multiple and controversial surgical approaches are used only if conservative care is unsuccessful or long tract signs are present with progressive neurologic deficits. A case of conservative chiropractic distraction adjusting will be presented that was successful in alleviating the patient's symptoms.

A 40-year-old athletic woman complained of midthoracic "shawl" distribution pain in the upper and midthoracic spine area. She had fallen 2 years previously after which the pain started. She saw a physiatrist, had physical therapy, and chiropractic treatment, which did not help. MRI (Figs. 12.75 and 12.76) shows degenerative disc disease and spondylosis at the T8–T9 level with impingement of the thoracic cord to the right of the midline.

Treatment with distraction adjustment shown in Figure 12.77 as well as positive galvanic current applied to the right T8–T9 discogenic change was given. Flexibility and strengthening exercises were given to the thoracolumbar spine and the relief was excellent with pain only on prolonged use of the spine such as lifting and bending.

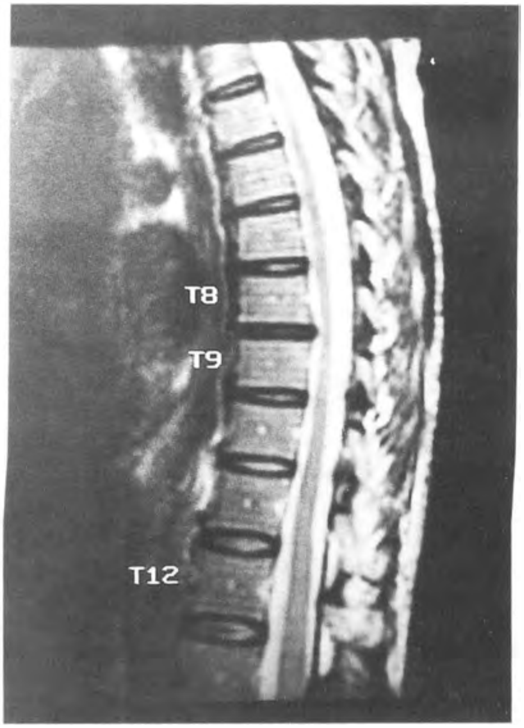


Figure 12.75. Sagittal magnetic resonance image of the thoracic spine showing the disc protrusion at the T8–T9 level.

Figure 12.76. Axial magnetic resonance imaging at the T8–T9 level shows (*arrow*) the right degenerative disc disease and spondylosis contacting the cord and creating stenosis of the lateral recess.

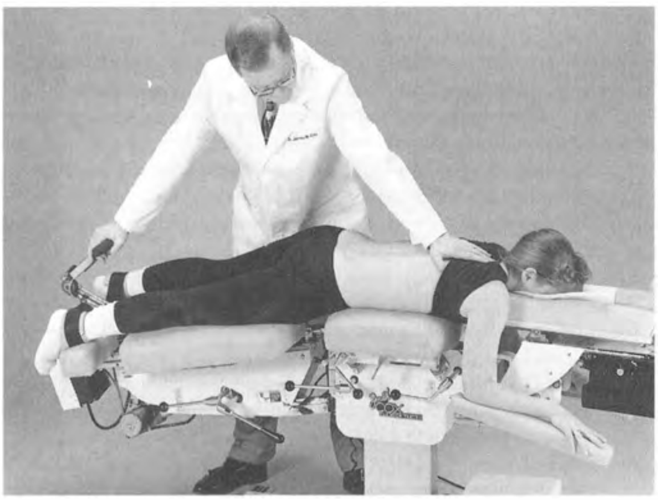


Figure 12.77. Cox distraction adjustment applied to the T8–T9 segments.

Comments on Thoracic Disc Herniation Concerning Treatment

Ninety asymptomatic patients underwent MRI of the thoracic spine with 53% of them showing disc herniations, 58% an anular tear, deformation of the spinal cord in 29%, and 38% Scheuermann's disease. Clinical decision-making must be based on clinical findings because high percentages of normal persons show thoracic disc herniations on MRI (315).

Twenty patients with 48 asymptomatic thoracic disc herniations diagnosed with MRI were followed for 26 months with 21 small herniations showing no size change and 3 increased in size. Of 20 medium-sized herniations, 16 showed small or no change and 1 a significant increase in size and 3 a decrease in size. Of 7 large herniations, 3 demonstrated no change, 4 a decrease. Five new herniations were discovered, 1 small and 4 moderate in size. Asymptomatic discs seem to be in flux as to size. Small discs tend to get larger and large ones smaller. All patients remained asymptomatic, although none of the discs resolved completely (316).

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Facet Syndrome

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chapter 13

With regard to excellence, it is not enough to know, but we must try to have and use it.

—Aristotle

This chapter, which deals with probably the single most common factor seen in chiropractic practice with low back pain patients, begins with a discussion of the biomechanics of the posterior elements of the lumbar spine.

Superimposed loads on the lumbar spine are borne by the body-disc-body anteriorly and by the two articular facets posteriorly; ligaments provide stability for the posterior elements and the intervertebral disc (IVD). It is obvious that weight distribution on these elements changes with degenerative disc disease, in which narrowing of the disc places disproportionately more weight on the articular facets.

COMPRESSIVE FORCES ACTING ON THE ARTICULAR JOINTS

The compressive force passing through the posterior column (articular facet joints) has been obtained by taking the area of the inferior articular facets. The vertebral body surface area gradually increases from T5 to L4, indicating increased weightbearing by the anterior column from above downward. The L5 vertebral body is significantly smaller than that of L4, indicating that compressive force is diverted before reaching the L5 inferior surface (1).

The mean articular facet area increases suddenly at L4 and L5 as compared with the upper lumbar levels, indicating more compressive force occurs at the articular facets in the lower rather than in the upper lumbar spine (Table 13.1). Transfer of part of the compressive force from the anterior to the posterior column is suggested. The increased transfer of weight through the pedicles at L5, which is an area of forward and downward inclination of forces as L5 sits on the sacrum at an inclined plane, has been offered as an explanation for the stress leading to fracture of the pars interarticularis (spondylolysis) and resultant spondylolisthesis.

Disc Versus Facet Compressive Weightbearing

It is important to know, under compressive loading, how much weight is borne by the articular facets versus the intervertebral disc. The percentage of weightbearing compressive load transmitted through the articular facets, in persons with normal IVDs, no evidence of degeneration, and a slightly flattened lumbar lordosis (Fig. 13.1), has been measured at 16% in two studies (1, 2) and between 3 and 25% in another (3). Morris et al. (4) state that 70% of the superimposed body weight is carried on the vertebral bodies and 30% on the articular facets. Fiorini and McCammond (5) concluded that 12% of the weightbearing was on the facets.

In degenerative disc disease, the articular weightbearing is as high as 47% (3) or up to 70% (2). Much of this abnormally high resistance is caused by extra-articular impingement of the facet tips on the adjacent lamina or pedicle, and the apophyseal joints develop gross osteoarthritic changes. It is possible that the joint capsule is nipped by such high stress placed on the tips of the articular facets. This may explain why standing for long periods can produce a dull ache in the low back that is relieved by sitting or by using some device, such as a bar rail, to rest one foot upon, to induce slight flexion of the lumbar spine (2). This contact between facet tip and lamina is labeled in chiropractic as a "facet-lamina syndrome."

It is shown that if the lumbar spine is slightly flattened (as occurs in erect sitting or heavy lifting), all the intervertebral compressive force is resisted by the disc. However, when lordotic postures, such as erect standing, are held for long periods, the facet tips do make contact with the laminae of the subjacent vertebra and bear about one sixth of the compressive force (Fig. 13.2) (2).

Table 13.1

Percentage Area of Body and Articular Facets at Various Vertebral Levels

Vertebral Levels	Total Area (Body + Facets) cm²	Total Area (Body + Facets) %	Body Area cm²	Body Area %	Area of Two Articular Facets cm²	Area of Two Articular Facets %
T5	7.00	100	5.34	76.28	1.66	23.71
T8	9.32	100	7.30	78.32	2.02	21.68
T9	10.11	100	7.91	78.23	2.20	21.76
T11	10.92	100	8.82	80.76	2.10	19.23
T12	12.04	100	10.24	85.04	1.80	14.95
L1	13.66	100	11.46	83.89	2.20	16.10
L3	16.84	100	13.82	82.06	3.02	17.93
L4	17.55	100	14.17	80.74	3.38	19.25
L5	17.93	100	14.07	78.47	3.86	21.52

Percentage Area of Body and Cross-Sectional Area of Lamina at Various Vertebral Levels

Vertebral Levels	Total Area (Body + Lamina) cm²	Total Area (Body + Lamina) %	Body Area cm²	Body Area %	Area of Lamina cm²	Area of Lamina %
T5	6.59	100	5.34	81.03	1.25	18.96
T8	8.73	100	7.30	83.61	1.43	16.38
T9	9.51	100	7.91	83.17	1.50	15.77
T11	10.10	100	8.82	87.32	1.28	12.67
T12	11.49	100	1.24	89.12	1.25	10.87
L1	12.88	100	11.46	88.97	1.42	11.02
L3	15.61	100	13.82	88.53	1.79	11.46
L4	16.43	100	14.17	86.24	2.26	13.75
L5	17.08	100	14.07	82.37	3.01	17.62

From Pal GP, Routal RV. Transmission of weight through the lower thoracic and lumbar regions of the vertebral column in man. J Anat 1987;152:98. Reprinted with the permission of Cambridge University Press.

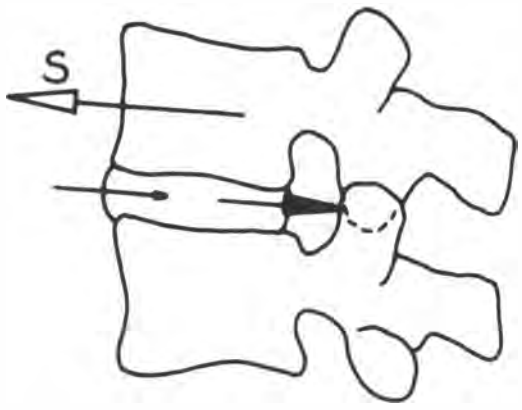

Figure 13.1. Schematic drawing of a lumbar spine having normal disc spaces and normal compressive weightbearing on the anterior and posterior columns of the spine. (Reprinted with permission from Adams MA, Hutton WC. The mechanical function of the lumbar apophyseal joints. Spine 1983;8(3):328.)

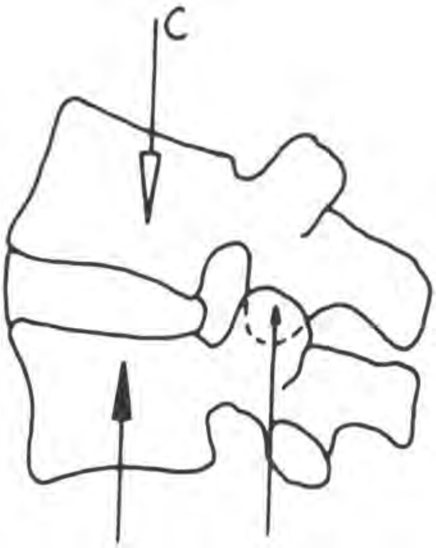

Figure 13.2. Schematic drawing of a lumbar spine with intervertebral disc space thinning and increased articular joint weightbearing. (Reprinted with permission from Adams MA, Hutton WC. The mechanical function of the lumbar apophyseal joints. Spine 1983;8(3):328.)

Simulation of Triple Joint Complex in Laboratory

A two-dimensional biomechanical model was assembled using two rigid bodies as the vertebrae and six elastic springs to represent the tissues of the disc and posterior elements. Compression loads were inflicted, and the following facts were determined (6):

1. The apophyseal joints are not loaded heavily by compression or flexion-extension loads, but they can be heavily loaded by anteroposterior shear loads.
2. Resistances developed by the apophyseal joints are not effective in relieving loads on the intervertebral disc when the motion segment is compressed, but they can be effective in relieving the disc when the segment is flexed, extended, or anteroposteriorly sheared.
3. In response to anteroposterior shear loads, the location of the facet joints relative to that of the intervertebral disc in the superior-inferior direction is a major determinant of what loads each structure will bear.

Under compressive load, the highest compressive strains were recorded near the bases of pedicles and on the superficial and deep surfaces of the partes interarticulares; the loads were increased by extension (7). It is possible that extension movement is limited by the bony contact of the facet joints (2).

Rotational Stresses on the Disc and Facet

In shear stress applied to the intervertebral joint, two thirds of the stress is borne by the disc and one third by the facets.

Normal intervertebral discs fail completely at 22.6° of rotation when studied in cadavers, whereas in real life they can tolerate only 5° of rotation without damage (8). The lumbar apophyseal joints function to allow limited movement between vertebrae and to protect the discs from shear forces, excessive flexion, and axial rotation. They are not well designed to resist compression, which is normally borne by the disc (2).

Unequal Facet Loading Leads to Unequal Degeneration

Panjabi et al. (9) present the following algorithm of the stages of injury to the functional spinal unit (FSU):

1. Asymmetric disc injury at one FSU level.
2. Disturbed kinematics of FSUs above and below this level.
3. Asymmetric movements at facet joints.
4. Unequal sharing of facet loads.
5. High load on one facet joint causing intra-articular cartilage degeneration, joint space narrowing, and facet atrophy (arthrosis).

Figures 13.3 and 13.4 are common radiographic findings in a daily chiropractic practice. The slight rotational, lateral flexion subluxation of a lumbar vertebra with unilateral disc degeneration results in greater facet loading on the concave side of the subluxation. The resultant degenerative changes are seen in the triple joint complex at the facet.

Facet Subluxation in Unequal Weight Distribution

Hadley's "S" line allows visualization of facet disrelationships, and it is especially beneficial in evaluating oblique views of the lumbar spine and, to a lesser extent, anteroposterior views. Figures 13.5 through 13.8 schematically and radiographically show how these lines are established. The Hadley "S" curve is formed by tracing a line along the undersurface of the transverse process at the superior process and bringing this down the inferior articular process to the top of the superior articular surface; this is joined by a line traced upward from the base of the superior articular process of the inferior vertebra to the lower edge of its articular surface. These lines should join to form a smooth S. If the S is broken, subluxation is present (10).

PAIN SENSITIVITY OF THE FACET SYNOVIAL-LINED JOINT

Pressure-sensitive recording paper was placed between the facet facings, and the pressure between the facets was measured. This was done on 12 pairs of facet joints, and it was

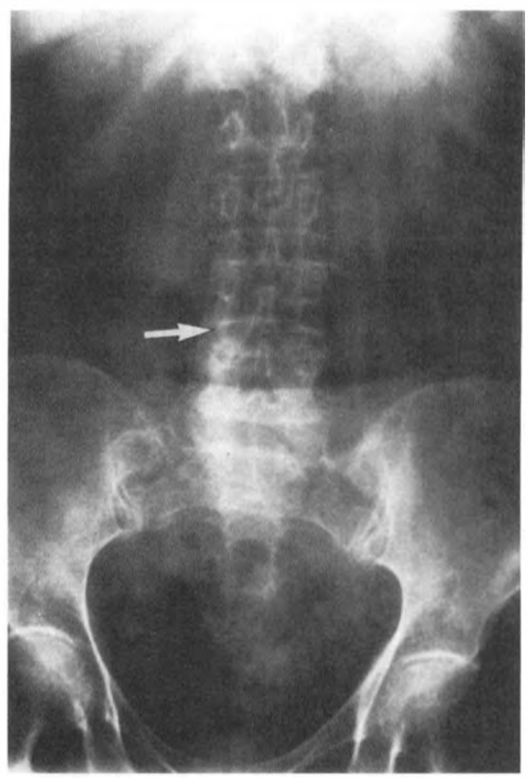

Figure 13.3. An anteroposterior projection of a lumbar spine with a slight dextrorotation and lateral flexion subluxation of the L4 vertebral body on L5 and L3 on L4, resulting in increased weightbearing on the left articular facet joints (*arrow*) compared with the right.

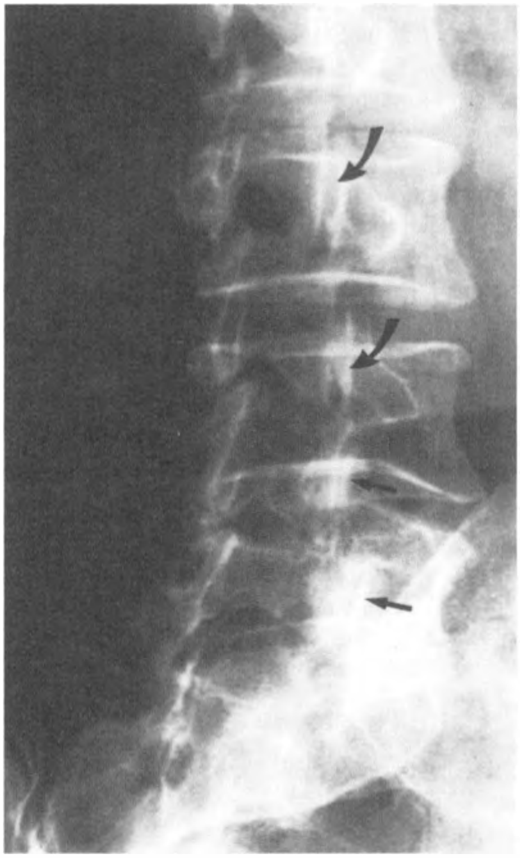

Figure 13.4. An oblique projection shows the narrowed intra-articular joint space, subchondral sclerosis, and facet imbrication at the L4 and L5 levels where increased weightbearing has taken place for a period of time (*straight arrows*). Compare these changes with the more normal joints above at L3 and L2 (*curved arrows*).

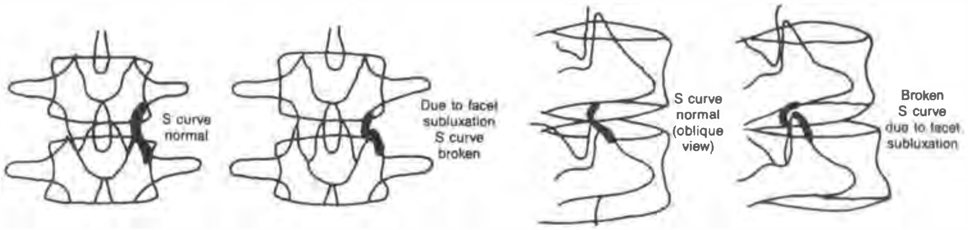

Figure 13.5. The "S" lines of Hadley are shown for determining the facet subluxation that occurs when increased weightbearing is placed on a facet joint. (Adapted from Yochum TR, Rowe L: Essentials of Skeletal Radiology. Baltimore: Williams & Wilkins, 1987:192.)

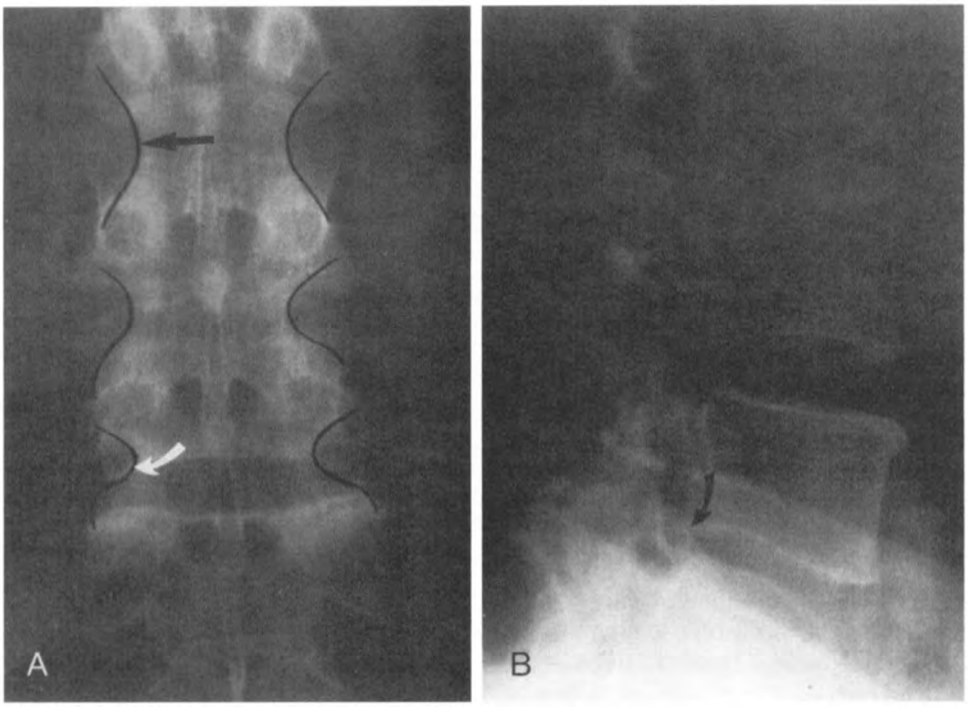

Figure 13.6. **A.** "S" lines are shown on an anteroposterior radiograph of the lumbar spine that reveals normal (*straight arrow*) and broken "S" lines (*curved arrow*). **B.** Lateral view of **A** shows the retrolisthesis (*arrow*) of L5 accompanying the Hadley "S" line changes.

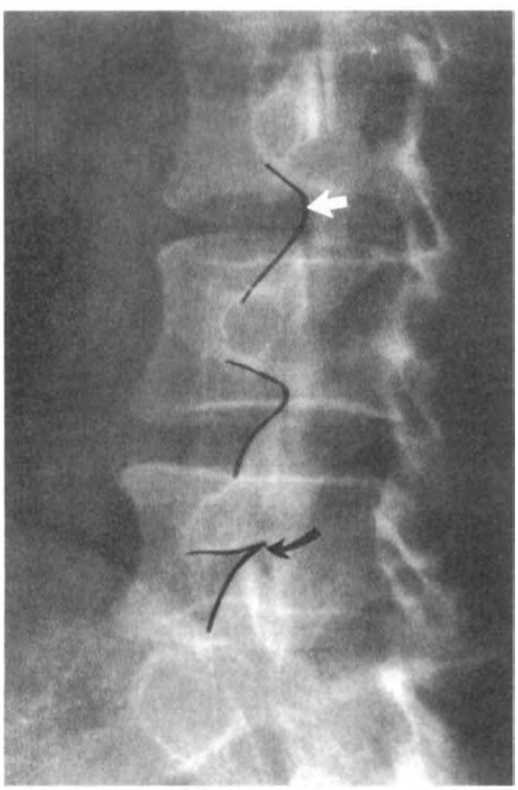

Figure 13.7. Right anterior oblique view of the lumbar spine shows normal (*straight arrow*) and abnormal (*curved arrow*) "S" lines.

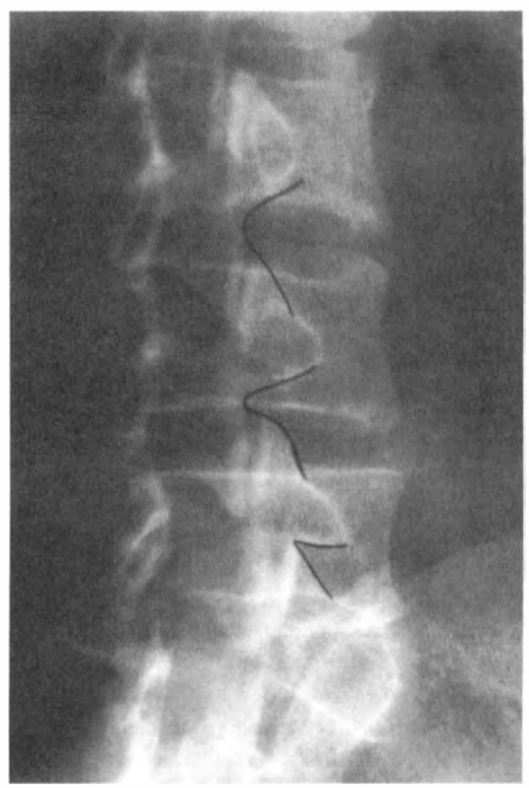

Figure 13.8. Left anterior oblique view of the lumbar spine shows normal and abnormal "S" lines.

found that narrowing the disc space and increasing the angles of extension caused an increase in pressure or impingement on the facet joint surfaces (11). Stretching of the joint capsule (or transmission of load across it) can be a source of pain because of the presence of a nociceptive type IV receptor system (12).

Histologic study of sectioned zygapophysial joints indicates the presence of an extensive vascular supply to the articular cartilage in a joint that shows minor osteoarthrosis. The anatomy of a vascular synovial inclusion of the type seen in most lumbosacral zygapophysial joints is clearly demonstrated. Because vascular structures can be related to pain, this may explain spinal pain of zygapophysial origin (13).

The capsule of the articular facets is richly innervated with sensory fibers, according to von Luschka (14). The posterior primary division of the spinal nerve and the recurrent nerve of the anterior primary division innervate the capsule. This sensory nerve supply is sufficiently developed to support the hypothesis that irritation of the capsule of the lumbar articular facets could well produce pain stimuli. In turn, this stimuli could return to the central nervous system through the posterior primary division and produce referred pain through the dermatomes of the involved nerves, which correspond exactly with the pathway of sciatic radiation, namely, the fourth and fifth nerves.

The synovial folds of the lumbar zygapophysial joints are innervated by nerves ranging from 1.6 to 12 μm in diameter, with the number of fibers ranging from 1 to 5. They run a course separate from blood vessels, indicating that they are afferent nerves that probably have a nociceptive function (15).

Ghormley (16), in his classic paper, stated ample evidence existed to regard facets as a cause of sciatic pain. He used the term "facet syndrome" to describe the sudden onset of low back pain brought on by some activity, which usually involved a twisting or rotatory strain of the lumbosacral region.

Facet joints are subject to abnormal stresses following disc degeneration. The normal pedicle-facet complex with a normal intervertebral disc carries 20% of the vertical pressure applied at the interspace, and this constitutes 10 times the weight per square inch applied to the knee joints (17).

Uneven apophyseal joint spaces, from right compared with left or vertically adjacent, indicate disc damage, instability, or possible bulge. Facet override is a finding in disc lesion (18).

FLEXION AND EXTENSION EFFECTS ON FACET LOADING

Under axial compression force, the location of the segmental mechanical balance point shifts posteriorly as the facets come into contact. In coupled flexion rotation, under axial compression, each facet carries a negligible percentage of compression that remains nearly constant as applied force increases.

The contact forces developed at the facet articulation increase considerably with extension rotation. For example, the addition of up to 5.6° of extension rotation increases the load on each facet from 10 to 30% of the compression preload. Large flexion loadings similar to those expected during heavy lifting, as well as large extension loadings, are likely to be related to facet injury and degeneration. The transfer of forces from one facet to the adjacent one occurs through different areas in flexion and in extension postures. That is, on the articular surface, the contact area shifts from the upper and central regions in flexion to the inferior tip in extension (19).

The anteromedial region of the zygapophysial joints has been shown to be the primary site of degenerative change (20).

RADIOGRAPHIC CONCEPTS OF FACET SYNDROME

Two studies done by Cox et al. (21, 22) reveal that 26% of patients with low back pain have facet syndrome either alone or in conjunction with other findings. The exact degree of low back pain caused by the facet syndrome is still unknown. A close look at the stresses imposed on the lumbosacral articulation by facet syndrome should, therefore, be of great importance to the chiropractic physician treating this condition.

Figure 13.9 is a radiograph of a patient suspected of having facet syndrome. Posterior narrowing is seen of the L5–S1 IVD space compared with the anterior disc space, and imbrication of the first sacral facet into the upper third of the intervertebral foramen at L5–S1, resulting in apparent vertical stenosis of the L5–S1 foramen as compared with the adjacent levels.

Figure 13.9. Radiograph showing a facet syndrome at L5–S1. Posterior narrowing is seen of the L5–S1 intervertebral disc space, with the first sacral facet stenosing the L5–S1 foramen by its vertical telescoping subluxation (*straight arrow*). Note also the nuclear disc invagination of the L5–S1 disc into the inferior vertebral body plate of L5. L5 shows a retrolisthesis subluxation on the sacrum (*curved arrow*).

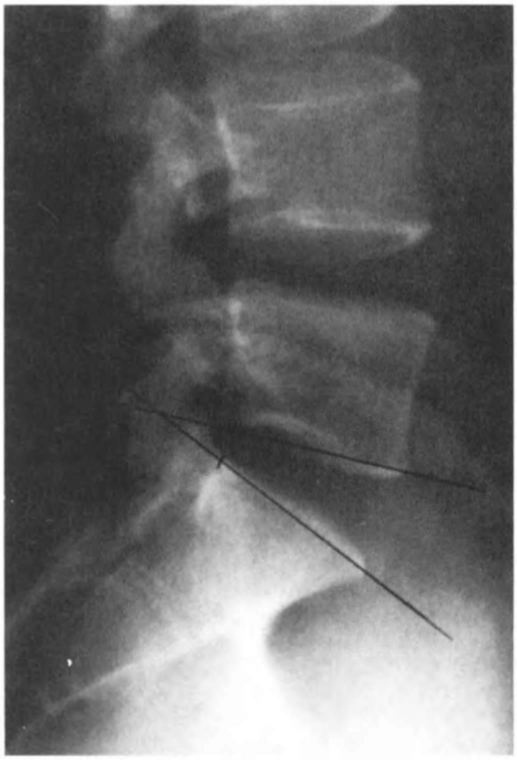

Figure 13.10. Macnab lines drawn along the superior S1 vertical plate and inferior L5 vertebral plate intersect near the zygapophysial joints instead of more posteriorly. Also, the S1 superior facet lies well above the line drawn along the inferior L5 body plate, indicating probable vertical stenosis at the L5–S1 intervertebral foramen. This is termed "facet imbrication."

Figure 13.10 shows the lines of Macnab (23) identifying the hyperextension subluxation of L5 on the sacrum, with the tip of the superior facet of the sacrum imbricating above the line drawn along the inferior plate of L5. Telescoping of the superior sacral facet into the intervertebral foramen at L5–S1 creates vertical stenosis of the foramen. Also note that the lines drawn along the inferior plate of L5 and the superior plate of the sacrum intersect at a point that is near the articular facets. The closer to the facets these lines cross, or if they are actually anterior to the articular joints, the greater will be the severity of the facet syndrome, meaning a greater posterior disc space narrowing, vertical narrowing of the foramen, and hyperextension subluxation of the facet joints.

Hellems and Keats (24) found the normal sacral base angle to be 41° (Fig. 13.11). At this degree of angulation of the sacrum, 80% of the superimposed body weight is carried on the vertebral bodies and the sacral promontory. Although only 20% of the weight is carried on the articular facets, the resulting pressure per square inch on the facets is 10 times greater than the pressure carried on the knee with the person standing in the upright posture. This example clarifies the strain produced on the articular facets in normal kinematics.

An increase of the sacral angle shifts weightbearing posteriorly onto the posterior elements and facets (Fig. 13.12). The articular facets were never created to stand this shearing stress. As the sacral base inclines further, a hyperextension subluxation of the upper motion segment or hyperflexion subluxation of the lower motion segment must take place. In Figure 13.13 is seen a 65° sacral base angle with facet syndrome. Figure 13.14 demonstrates marked structural faults with a degenerative spondylolisthesis at the L4 level and a facet syndrome and increased sacral angle at the L5–S1 level. Treatment of this last patient would involve addressing both conditions by placing a flexion pillow under L4 while contacting the spinous process of L3 in applying the distraction manipulation.

Figures 13.15 and 13.16 show two basic conditions to be dealt with in manipulation. A facet syndrome is present at L5–S1 (Fig. 13.15), but also present is a transitional segment with a unilateral pseudosacralization of the left transverse process to the sacrum (Fig. 13.16). As presented in this text in Chapter 6, *Transitional Segment,* this condition proved to be the most time-consuming and treatment-demanding condition to yield to manipulative care in a study of 576 cases (22). Couple this with a facet syndrome, and we see a very difficult case to treat. I treated the patient this case by contacting the spinous process at the L5 level and *very gently* applied flexion distraction at the L5–S1 joint. This was followed with complete range of motion manipulation of the articular facets. A belt support was used, as shown in Chapter 9, *Biomechanics, Adjustment Procedures, Ancillary Therapies, and Clinical Outcomes of Cox Distraction Technique,* to stabilize this joint while healing.

Figure 13.17 reveals a stable, normal disc space with no evidence of facet imbrication. Note that the lines drawn along the inferior L5 and superior sacral plates intersect far posterior to

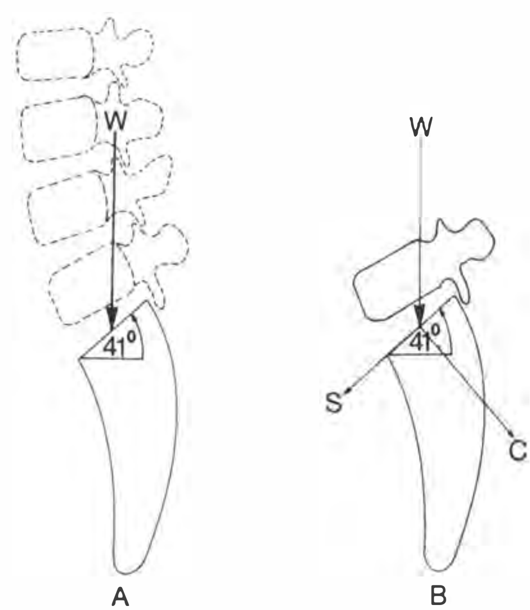

Figure 13.11. Position of the normal sacrum during erect standing. **A.** The superincumbent weight (*W*) passing through the posterior edge of the lumbosacral joint. **B.** The compression (*C*) and shearing (*S*) components of the superincumbent weight. (Reprinted with permission from LeVeau B. Biomechanics of Human Motion. Philadelphia: WB Saunders, 1977:94.)

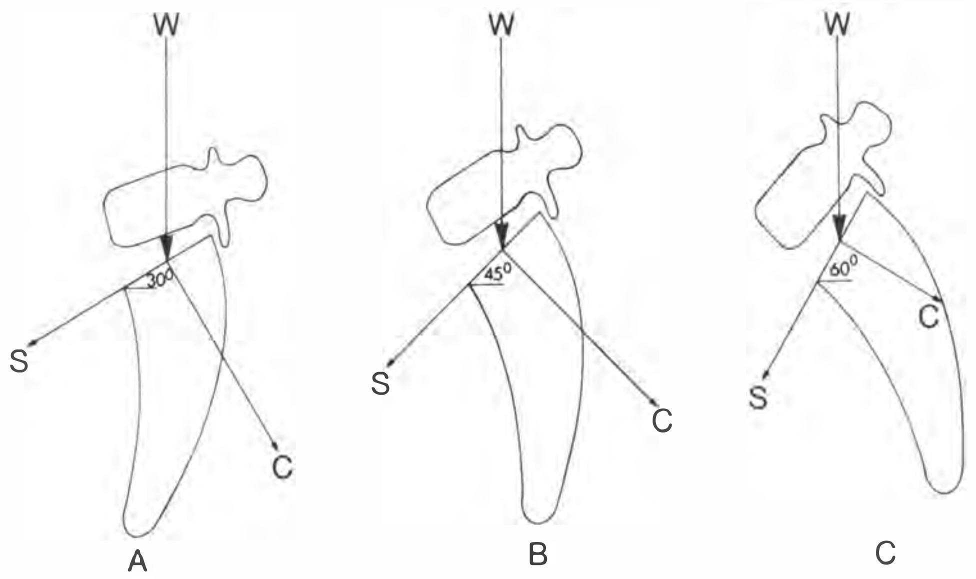

Figure 13.12. Change in the compression (*C*) and shearing (*S*) force components with change in the sacral angle. *W*, weight. (Reprinted with permission from LeVeau B. Biomechanics of Human Motion. Philadelphia: WB Saunders, 1977:95.)

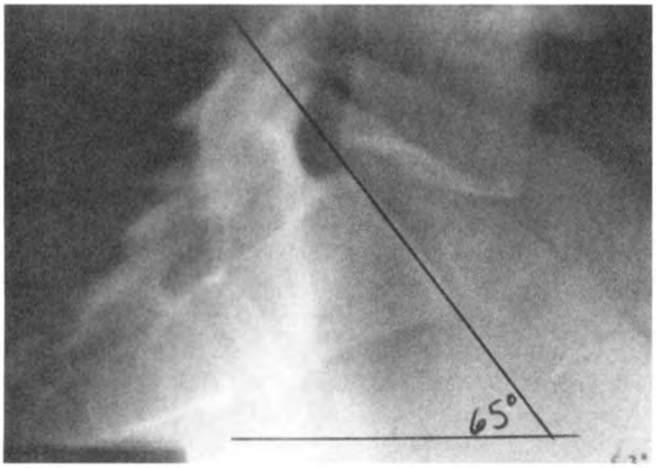

Figure 13.13. Increased sacral angle and hyperextension subluxation of L5 on the sacrum.

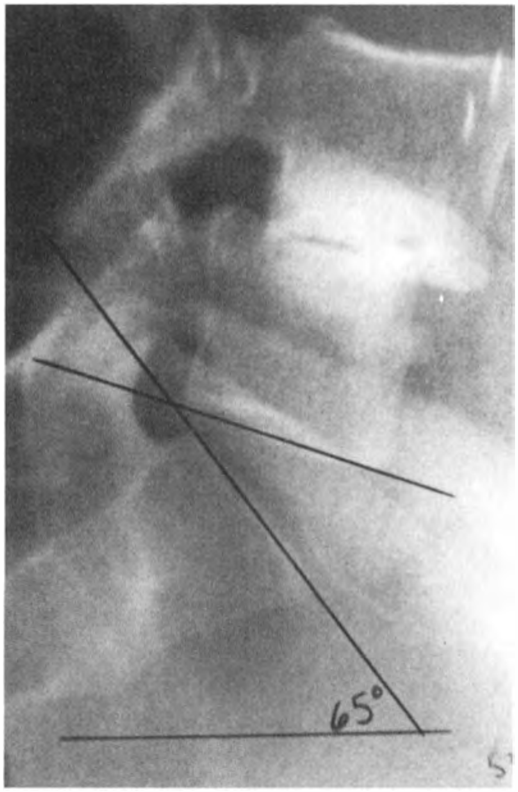

Figure 13.14. Radiograph showing a facet syndrome subluxation complex at L5–S1 and a degenerative spondylolisthesis at L4 on L5. Treatment for this combination problem is discussed in the text.

Figure 13.15. Radiograph showing facet syndrome of L5 on S1, with marked disc thinning, imbrication of the first sacral facet into the L5–S1 foramen, and probably some arthrosis of the L5–S1 facet joints.

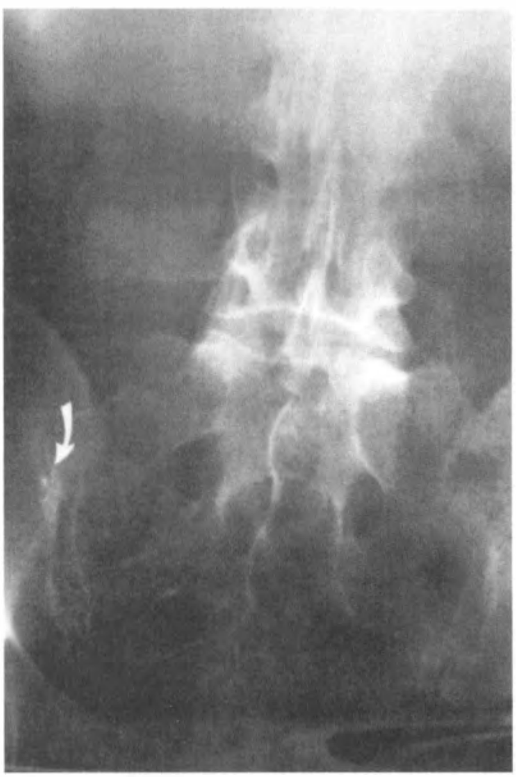

Figure 13.16. In addition to the findings on the lateral view in Figure 13.15, a unilateral pseudosacralization transitional segment is seen of the L5 transverse process with sacrum (*arrow*).

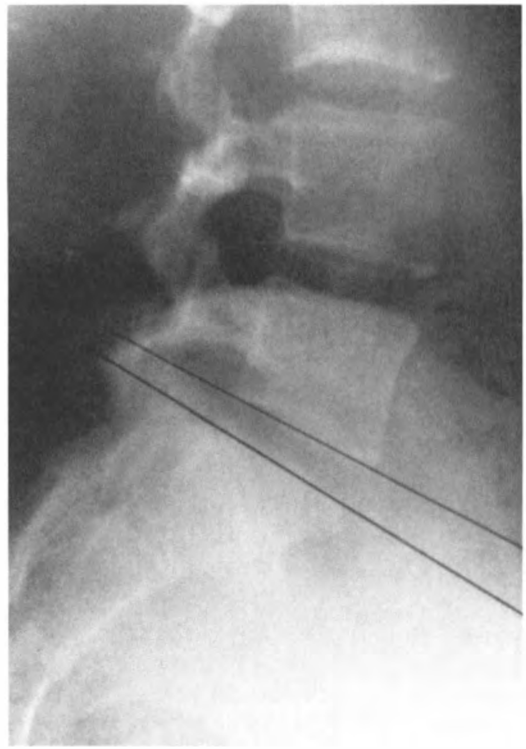

Figure 13.17. Stability is suggested in the radiograph by the parallel Macnab's lines and their intersection far posterior to the L5–S1 facet joints. Note the symmetric L5–S1 disc space indicating probable maximal weightbearing on the disc and minimal weightbearing on the articular facet joints.

the lumbosacral junction. This type of finding indicates a stable articulation and the weightbearing primarily is found on the body-disc-body, with minimal weightbearing on the articular facets at L5–S1.

STABILITY IN THE FACET SYNDROME AND AN INDICATION OF RESPONSE TO MANIPULATION

Although the articular facets are well supplied with nerve fibers from the dorsal ramus of the spinal nerve, discussion continues regarding the role the articular facet plays in the cause of low back and lower extremity pain. Van Akkerveeken determined a measurement for stability or instability of the lumbar spine from use of lateral lumbar films to determine damage to the posterior longitudinal ligament and the anulus fibrosus. This measurement is illustrated in Figure 13.18.

According to Van Akkerveeken (25), in a normal lumbar spine in full extension, with the anulus fibers and longitudinal ligaments intact, a line drawn along the posterior longitudinal ligament shows a fairly smooth arch. If the anulus fibers are cut, a definite posterior sliding of each vertebra posteriorly occurs on the vertebra below. If lines are drawn along the inferior plate of the vertebra above and along the superior plate of the vertebra below, and the intersection of these lines is called

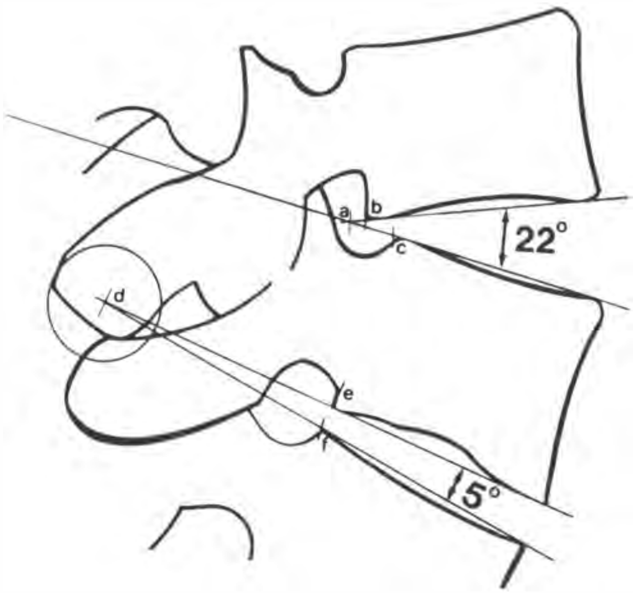

Figure 13.18. Line drawing of the lateral aspect of lumbar segment in full extension, illustrating radiologic instability and methods of measuring it (degrees of tilt and length of parallel displacement). The lower segment is stable; *de* = *df* in length. At the upper segment, radiologic instability is demonstrated; in this case, line *ab* is 3 mm shorter than line *ac* (see text for explanation). (Reprinted with permission from Van Akkerveeken PF, O'Brien JP, Park WM. Experimentally induced hypermobility in the lumbar spine. Spine 1979;4(3):238.)

point *a*, less than a 3 mm difference in length should be found between the line drawn from point *a* to the posterior margin of the superior vertebra and the line drawn from point *a* to the posterior margin of the inferior vertebra. If the difference is 3 mm or greater, instability is present, meaning damage has occurred to the anular fibers or the posterior longitudinal ligament. We use this measurement as a prognostic aid to determine the response of a patient to treatment as well as to predict future difficulty in the lumbosacral spine.

It has also been shown that the greater the discal angle, the more severe the facet syndrome. The discal angle (*edf*) shown in Figure 13.18 is 5°, a sign of stability and no facet syndrome. The other angle (*bac*) is 22°, a sign of severe facet syndrome. I believe that any discal angle greater than 15° is a sign of severe facet syndrome (Fig. 13.19).

Figure 13.20 demonstrates the use of Van Akkerveeken's line measurement to determine stability. The spines shown in Figure 13.20 are stable. Figure 13.21 demonstrates the use of this measurement in a patient with an unstable spine. A line is drawn from the point of intersection (*a*) to the posterior border of the fifth lumbar body above (*b*) and to the posterior border of the sacrum below (*c*). The distance from *a* to *b* measures 11 mm; the distance from *a* to *c* measures 16 mm. By Van Akkerveeken's measurement, therefore, the lumbosacral articulation is unstable, showing that the anulus and posterior longitudinal ligament are damaged.

The facet syndrome has been accused of causing much low back pain; a review of our present knowledge about the sensitivity of the articular bed of the facet, therefore, is in order.

Increasingly in the literature, articles are appearing concerning the innervation of the articular facets. Important anatomic relationships exist in the lumbosacral region of the adult which are traceable to embryonic development. In their discussion of the pain relationships evolving from biomechanical faults of the lumbosacral complex, Carmichael and Burkhart (26) state that the paraxial mesoderm that condenses alongside the notochord becomes segmented into somites. Each somite then differentiates into a scleratome (which contributes to vertebrae formation), a myotome (which forms axial and appendicular muscle), and a dermatome (which forms the dermis). The developing neural tube innervates each somite and its derivatives so that the nerve pattern becomes segmental.

Each scleratome divides transversely, and each hemiscleratome reaggregates with a hemiscleratome adjacent to it, becoming the centrum that forms most of the vertebral body. These divisions and reaggregations determine important anatomic relationships in the adult (*a*) spinal nerve, which originally would have run through the scleratome, now runs between the vertebrae; and (*b*) the myotome forms muscle that spans adjacent vertebral segments, thus establishing the patterns for back muscles.

The notochord, surrounded by the centrum, undergoes mu-

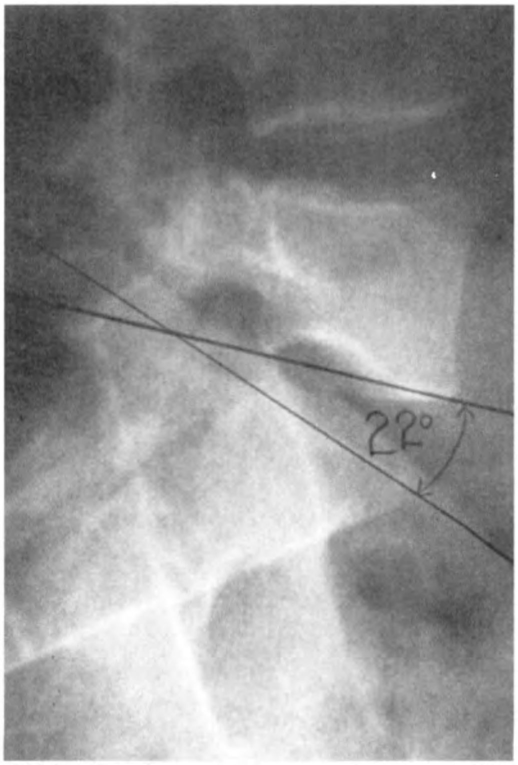

Figure 13.19. Lines are drawn to determine whether there is facet syndrome. Note that the angle is 22°. The greater this angle becomes, the greater the severity of the facet syndrome because of hyperextension of L5 and/or hyperflexion of the sacrum. The closer this angle is to 5°, the more stable the articulation.

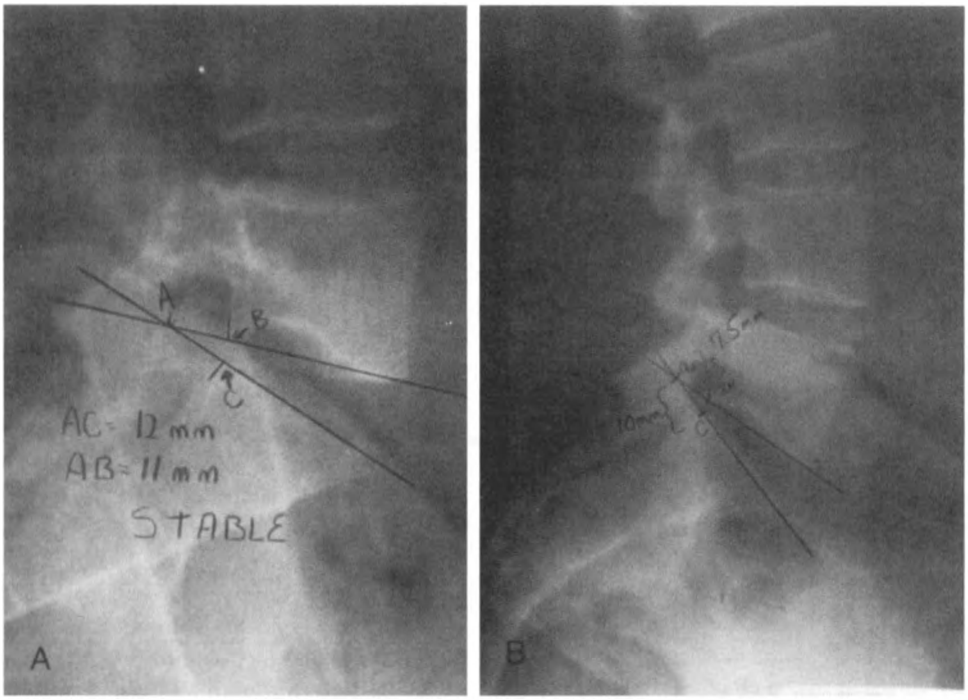

Figure 13.20. Van Akkerveeken's lines are drawn and show stability of the anulus fibrosus and posterior longitudinal ligament (**A**), where only 1 mm difference is seen between *AB* and *AC*. **B.** Also shows a stable facet syndrome, with a 2.5 mm difference between lines *ab* and *ac*.

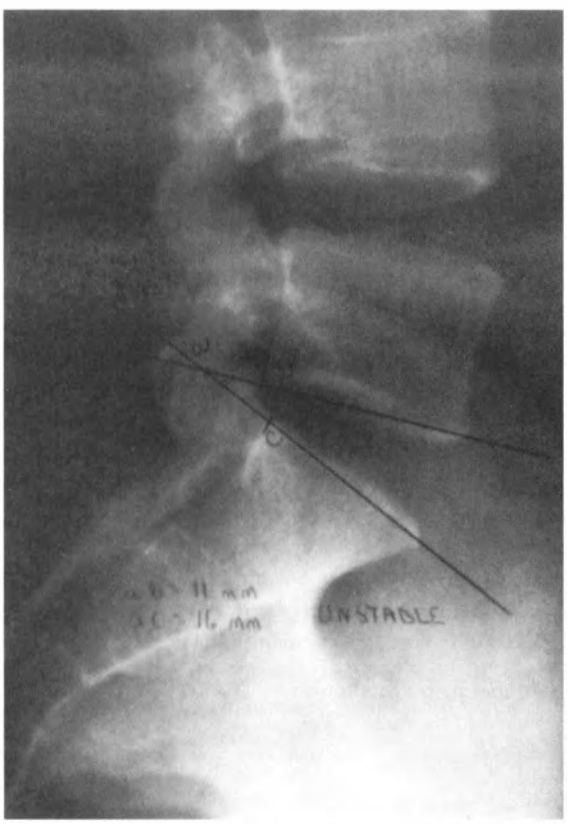

Figure 13.21. Unstable facet syndrome is shown with a 5 mm difference in lines *ab* and *ac*.

coid degeneration and usually disappears completely except for the nucleus pulposus of the intervertebral disc. The centrum eventually forms part of the membranous vertebral column. Each vertebra undergoes chondrification and ossification, a process completed several years after birth. The costal elements form a substantial part of the transverse process of the adult lumbar vertebra and the major portion of the lateral part of the sacrum.

Thus, by the process reviewed above, each vertebra is formed and the overall shape of the vertebral column is established. The five lumbar vertebrae typically are massive and show some differentiation. Generally, the vertebral foramen (which determines the shape of the spinal canal) becomes more triangular at L5 as the pedicles shorten, but the distance between the foramina shows little change.

STRUCTURAL FACTORS OF THE LUMBOSACRAL REGION

The joint between L5 and S1 is the single most common site of problems in the vertebral column because of, but not limited to, the following anatomic reasons: (*a*) this joint bears more weight than any other vertebral joint; (*b*) the center of gravity passes directly through these vertebrae; (*c*) a transition occurs here between the mobile presacral vertebrae and the relatively stable pelvic girdle; and (*d*) a change occurs in the angle that exists between these two vertebrae.

In 1976, Mooney and Robertson (27) pointed out that Ghormley had coined the phrase "facet syndrome" in 1933 and that lesions of the IVD could not explain all low back and leg

pain complaints. From his review of surgical literature, Sprangfort (28) found that only 42.6% of surgical patients obtained complete relief of back and leg pain following surgery.

Mooney and Robertson (27) also discovered that the injection of an irritant fluid into the facet joint caused referred pain patterns indistinguishable from pain complaints frequently associated with the disc syndrome. Even straight leg raising and diminished reflex signs were obliterated by precise local anesthetic injection into the facet joint. Injection of steroids and local anesthetic into the facet joint in a group of 100 consecutive patients suggested that this treatment alone achieved long-term relief in one fifth of the patients with lumbago and sciatica and partial relief in another one third of these patients. However, far fewer than half of the patients received long-term relief from pain from this technique. The point to be emphasized here is that the physician must be clinically careful to realize that a combination of therapies may be necessary to bring maximal relief of the patient's complaints.

FACET PAIN PATTERNS

Lora and Long (29) wrote that the results of stimulation in and around the facets yielded interesting pain patterns. Typical radicular radiation is not generated by stimulation of the nerves in and around the facet, but widespread referral of sensation even into the leg is possible. This referral of sensation, however, characteristically has a diffuse nonradicular character, is difficult for the patient to localize, and has not gone below the knee in any patient.

Stimulation of the L5–S1 facet characteristically produces sensation or reproduces pain in the coccyx, which is usually unilateral, or in the hip. The latter is usually described by the patient as being in the hip joint, and diffusely down the posterior thigh. Stimulation can occasionally travel circumferentially around the body along the course of the inguinal ligament into the groin.

Stimulation at the L4–L5 facets characteristically produces a local sensation that radiates diffusely into the posterior hip and thigh at the level of the electrode. Coccygeal radiation of sensation is less commonly observed with L4–L5 stimulation than with L5–S1 stimulation, but it does occur. Stimulation at L3–L4 characteristically produces radiation upward into the thoracic area. Pain or sensation radiates around the flank and into the groin and anterior thigh much more diffusely with L3–L4 stimulation than with L5–S1 stimulation. Coccygeal sensations in the perineum are produced more commonly with L3–L4 stimulation than with L4–L5 stimulation, but less commonly with L3–L4 stimulation than with

pain radiation, at least as judged by stimulation of the posterior ramus by use of this technique, may be much more diffuse than is generally supposed. Although hip, thigh, and groin radiations are well known from studies of patients with disc protrusion, the observation that stimulation characteristically reproduces pain in the coccygeal area or produces sensation in this region is not as well known. *It certainly seems possible that coccydynia is, in fact, another manifestation of lumbar degenerative disc disease* (Table 13.2).

Stimulation at the T12, L1, L2, and L3 levels does not produce leg or coccygeal sensations. Radiation of sensations is limited to the upper back, thoracic and cervical regions, and around the course of the T12, L1, and L2 nerve roots in a diffuse fashion on the anterior abdominal wall.

I would note that these are scleratogenous pains that do not cause any sensory or motor deficits in the lower extremity. These pains never radiate below the knee and are usually isolated to the buttock and upper thigh. When motor and sensory changes are noted down the lower extremity, a disc lesion should be suspected. Figure 13.22 shows the distribution of sensations from L4–L5 and L5–S1 facet irritation.

McCall et al. (30) studied the referral of induced pain from the posterior lumbar elements to (*a*) trace the exact area of pain referral from the L1–L2 and the L4–L5 levels and (*b*) compare the distribution and intensity of the pain produced by intra-articular versus pericapsular provocation. In their study, normal subjects were given injections of 0.4 mL of 6% saline. Pain started within 25 seconds of each injection, with the episode usually lasting 5 minutes. At both the L1–L2 and the L4–L5 levels, injection into the joint interior (intra-articular provocation) produced less intense pain than did pericapsular injection.

The upper lumbar level was more sensitive than was the lower lumbar level. The distribution of referred pain from either intra-articular or pericapsular injection was the same, but the intensity was worse with the pericapsular injection than with intra-articular injection.

In general, injection into the upper lumbar level referred pain to the flank region, whereas injection at the L4–L5 level referred pain to the buttocks. Thigh pain never extended beyond the knee. No contralateral pain was noted. *No demonstration of significant leg pain was produced in these normal subjects.*

Although no nerve endings are found in the articular cartilage and synovium, the fibrous capsule of the synovial joint is innervated.

Table 13.2

Facet Joint Pain Patterns Described by Lora and Long

L5–S1 facet pain distribution Coccyx Hip Posterior thigh Groin Flank	L4–L5 facet pain distribution Posterior hip and thigh Coccyx
L3–L4 facet pain distribution Upward to thoracic spine Diffuse flank and groin pain Coccyx	T12, L1, L2, L3 facet pain distribution No leg or coccygeal pain Radiating pain to thoracic and cervical spines

Based on Lora J, Long D. So-called facet denervation in the management of intractable back pain. Spine 1976;1(2):121–126.

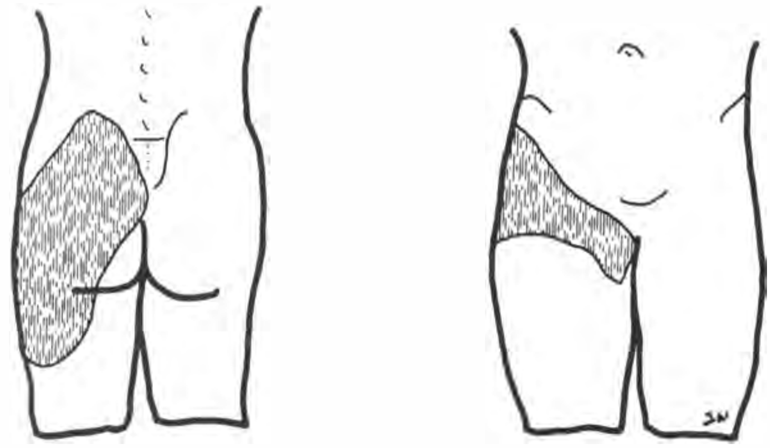

Figure 13.22. Distribution of sensations from L4-L5 and L5-S1 facet irritation.

Table 13.3

Facet Joint Pain Patterns Described by Schofferman and Zucherman

L4–L5, L5–S1 pain distribution:
Posterior thigh, calf, rarely to foot
Back pain greater than leg pain

Based on Schofferman J, Zucherman J. History and physical examination. Spine: State of the Art Reviews 1986;1(1):14.

McCall et al. (30) question the existence of scleratomes because of the considerable overlap of pain patterns between upper and lower lumbar spine facets.

Schofferman and Zucherman (31) feel that leg pain may prove more useful diagnostically. The distribution and quality of the pain are used to separate referred pain from radicular pain. Pain in the absence of neurologic deficit is referred pain, whereas pain in the presence of neurologic change is radicular. It must be borne in mind, however, that no neurologic signs may be present in the early stages of radicular problems. *Referred pain shares the same distribution as the innervation of the affected zygapophysial articulations. Pain arising from the L4–L5 and L5–S1 articulations will be felt in the posterior thigh, and occasionally in the medial or lateral calf, and back pain is usually greater than leg pain. Numbness or tingling can accompany this pain. Posterior joint complex pain (facet, ligament, anulus) rarely, but occasionally, extends beyond the calf and into the foot.*

Contrast this pain distribution with that of radicular pain caused by nerve root compression, for example, by a disc protrusion in which the predominant and more severe pain is usually felt in the thigh and in the posterior lateral calf, extending to the toes. Although dermatomal pain may not be exact, certain patterns are characteristic. L3 pain involves the groin and anterior medial thigh; L4 pain involves the anterior thigh and medial calf and gluteal area; L5 pain involves the lateral thigh, lateral and possibly medial calf, and great toe; and S1 nerve pain involves the posterior thigh, posterior calf, and lateral aspect of the foot and heel (Table 13.3).

The results of the above study (31), show that irritation of the articular facets at L4–L5 and L5–S1 can result in pain in the coccyx, perineum, groin, buttock, and flank and into the posterior thigh, radiating as far as the knee. Therapeutic interest in the facet is to maintain its ability to continue its normal ranges of motion and thereby render it as free of subluxation as possible.

LUMBAR FACET INJECTIONS WITH CORTICOSTEROIDS: ARE THEY OF ANY BENEFIT IN CHRONIC LOW BACK PAIN?

Local corticosteroid injections into facet joints proved to have little efficacy in patients with chronic low back pain. Forty-nine patients with chronic low back pain had their facets between the lumbar or sacral vertebrae injected with methyl prednisone acetate and 48 were injected with placebo. After 1 month, 42% of 49 patients injected with prednisone and 33% of the placebo group had marked or very marked improvement in pain level, functional status, or back flexion. Only 1 of 5 patients in the corticosteroid group, compared to 1 of 19 of the placebo group, had sustained improvement from months 1 to 6 (32).

In 22 of 40 patients who received lidocaine (2%) injections into their lumbar facet joints followed by 2 mg of cortivazol near the joint the pain was relieved; 17 of the 22 patients who received relief stated it was 90% relief (33).

Two groups (n = 86) of patients with chronic low back pain were randomly assigned to receive either facet joint injection or facet nerve block. Relief was short lived, and by 3 months only 2 patients continued to report complete relief of pain. Patients with pain 7 years or longer were more likely to report good to excellent pain relief than those with a shorter history. Neither facet joint injection nor facet nerve blocks are satisfactory treatment for chronic back pain (34).

Facet joint injections are routinely and safely performed throughout the United States, despite their expense and unproved efficacy (35). Correlation between facet block and clinical outcome is not possible (36).

Radiofrequency Lumbar Facet Denervation Shows Clinical Relief

Of 82 patients who underwent diagnostic medial branch posterior primary ramus blocks, 42 reported at least 50% relief of pain and proceeded to radiofrequency denervation. Of the 42 patients undergoing denervation, 45% reported at least 50% relief of pain 2 years after the procedure or at last follow-up (37).

Facet joint injection was performed in 245 patients who presented with chronic symptoms of low back pain, with or without nondermatomal lower limb pain referral (38). No previous back surgery had been performed, and each patient underwent both facet studies and provocative lumbar discography at the lower three lumbar levels. To localize accurately any level of symptom relief, only one level per day was studied in each patient. Lumbar discography was performed, and the presence or absence of symptom reproduction on injection of contrast medium was recorded at each level.

Among these patients, intervertebral discs were a more frequent source of symptoms than the facet joints. In 45 patients, complete symptom relief followed injection of local anesthetic into the facet joints. Following facet injection, no significant difference was apparent in the incidence of complete symptom relief between the three groups of patients: the incidence was 19% for those with symptomatic disc disease, 25% for those with nonsymptomatic disc disease, and 17% for those with total disc resorption. By contrast, of 45 patients with normal three-level lumbar discography, only 2 (5%) had complete symptom relief following facet injection. *The study indicates that the facet joint and capsule are infrequent pain sources in patients with severe chronic low back pain, particularly when the discs are normal, but also in the presence of significant disc degeneration* (38).

This highlights a controversy with respect to the relief possible through facet injection. I have been negatively influenced by facet and epidural injection attempts to relieve low back pain.

Manipulative Care of the Facet Syndrome

The manipulation used in treating facet syndrome is Cox fl ion-distraction procedures as performed on the Zenith-Cox instrument.

Patients are divided into two types for purposes of manipulative care: *patients having low back pain only, and those with low back pain and sciatica.* The flow chart shown in Figure 13.23 describes our treatment outline. Note that we do not place zygapophysial joints through their physiologic ranges of motion when the patient has sciatic radiculopathy until the leg pain shows at least 50% relief as noted by subjective patient evaluation and objective signs of straight leg raising, range of thoracolumbar motion, Déjérine's triad, and Kemp's sign. Any patient who has only low back pain, with leg pain not extending below the knee, is treated with full physiologic range of motion applied to the facet articulations. Flexion distraction is the first manipulative movement administered, followed by the remaining four normal ranges of motion, which will be discussed next.

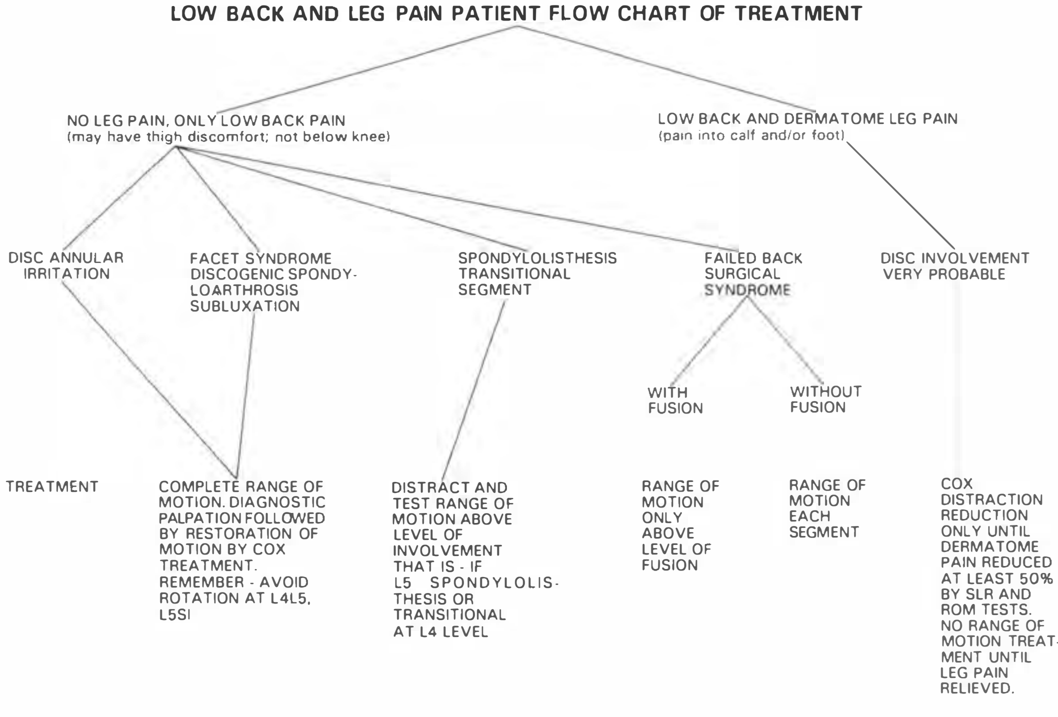

Figure 13.23. Flow chart of treatment for patients having low back pain alone or low back pain and sciatica. This chart outlines the treatment approach followed for manipulative care based on patients' findings.

Normal Joint Movements

The lumbar articular joints are capable of five movements: flexion, extension, lateral flexion, rotation, and circumduction. Pearcy (39) measured the ranges of active flexion and extension, axial rotation, and lateral bending in the lumbar spines of normal volunteers in vivo, to assess the relation between the primary and accompanying movements in the other planes. He stated that L5–S1 revealed larger movements of flexion and extension than did other levels of the lumbar spine, although L5–S1 did not demonstrate consistent patterns of equal movement of flexion and extension as seen at other levels of the lumbar spine. Lateral bending at L4–L5 and L5–S1 showed significantly less mobility than in the upper three levels.

In voluntary flexion and extension, Pearcy (39) found little accompanying axial rotation or lateral bending. During both axial rotation and lateral bending, large accompanying rotations occurred in the other planes. Axial rotation had a consistent pattern of accompanying lateral bending.

Pearcy (39) found lateral bending of approximately 10° occurring at the upper three lumbar levels, whereas significantly less lateral bending was evident at 6° and 3°, at L4–L5 and L5–S1, respectively. In flexion and extension, accompanying axial rotation of 2° or more, and lateral bending of 3° or more, occurred rarely, and larger accompanying rotation at an intervertebral joint should be considered abnormal. During twisting and side bending, axial rotation to the right is accompanied by lateral bending to the left and vice versa at the upper three levels. At L5–S1, axial rotation and lateral bending generally accompany each other in the same direction, whereas L4–L5 is a transitional level. During lateral bending, generally extension occurs at the upper levels and flexion at L5–S1.

Range of Motion Variation in Painful Versus Nonpainful Spines

Mayer et al. (40) studied the range of motion in the lumbar spine in painful versus nonpainful low back patients. He concluded that low back pain patients exhibit lower gross motion than normal subjects (54%), with the ratio of lumbar flexion to gross flexion decreased (63 to 43%). Range-of-motion exercising can significantly increase functional pain-free range both in lumbar (71%) and pelvic motion (39%) over a 3-week period.

Yang and King (3) state that normal, nonarthritic facet joints carry 3 to 25% of the superimposed body weight. If a facet joint is arthritic, the load could be as high as 47%. Transmission of the compressive facet load occurs through contact of the tip of the inferior facet with the pars interarticularis of the vertebra below. Further, facet overload causes rearward rotation of the inferior facet, which stretched the facet capsule.

Nociceptor Origin of Low Back Pain

Wyke (41) states that the cause of low back pain is irritation of nociceptors. The term "nociceptive" means "sensitive to tissue abnormality." Two abnormalities causing pain are mechanical and chemical. Three morphologic types of nociceptors are found:

1. Unmyelinated fibers in interstitial tissues.
2. Free naked nerve endings.
3. Paravascular nociceptive system in the adventitial layers of blood vessels, which are also unmyelinated.

Wyke points out that the apophyseal capsule contains unmyelinated nerve fibers. They are sensitive to both chemical and mechanical irritation, and high tensions develop in the facets following disc degeneration and the carrying of more weight.

SUMMARY OF MANIPULATIVE PRINCIPLES

Conclusions, based on the references cited above, indicate that manipulation can be beneficial by increasing spinal range of motion, relieving nociceptor irritation, perhaps equalizing the weightbearing between the anterior weightbearing column of the lumbar spine (made up of the vertebral body-disc-vertebral body) and the posterior column of the spine (namely, the articular facet), and finally relieving the compressive forces against the nerve root within the vertebral canal and intervertebral foramen.

Distraction Adjustment and Ancillary Care of Facet Syndrome Subluxation

Refer to Chapter 9, *Distraction Adjustment Procedures, Ancillary Therapies, and Clinical Outcomes of Cox Distraction Technique,* for the full protocol of treating facet syndrome. This includes patient placement on the table, tolerance testing, and application of all physiologic ranges of motion to the facet joints. Physiologic therapeutics, including positive galvanism, tetanizing current, hot and cold treatment, bracing, nutrition, low back wellness school, exercises, acupressure therapy, and treatment response to distraction adjusting are covered in Chapter 9 for facet syndrome condition.

Ancillary Care for Facet Syndrome

Those patients with hyperlordosis, facet syndrome, or anterior weightbearing stress on the lumbar spine who have ankle pronation or pes planus arch defects are treated with foot manipulation and arch orthotics. Figures 13.24–13.26 show this condition and the orthotic used to correct it.

All facet syndrome patients attend low back wellness school, especially those with unstable or severe pain type. This class is held every 2 weeks, and it becomes a routine part of patient management.

Cox exercises 1–6 and 8–10, shown in Chapter 9, Figure 9.83, are recommended.

Figure 13.24. Pes planus of both feet.

Figure 13.25. Medial view of marked arch planus in a patient with facet syndrome of the lumbar spine.

Figure 13.26. Orthotics used to correct pes planus and additional care of the patient with facet syndrome.

RETROLISTHESIS SUBLUXATION

The treatment of retrolisthesis subluxation is discussed at this time because of its seemingly increased incidence with facet syndrome; it is often a dual subluxation with the facet subluxation.

Figure 13.27 shows an unstable facet syndrome of L5 on S1, with L5 being 5 mm posterior on the sacrum. This creates an apparent facet imbrication of the L5–S1 intervertebral foramen by the superior facet of S1 entering the upper third of the foramen. This subluxation is far from being totally accepted or explained. Let us consider some opinions on this subluxation prior to studying its manipulative care under our type of manipulation.

I feel that retrolisthesis can be caused by three primary factors:

1. Congenital underdevelopment of the pedicles of the lumbar vertebra, so-called "pedicogenic stenosis." This underdevelopment certainly creates alteration of motion capacity and can be relieved only by the best of treatment.
2. Multifidus and rotatores muscle spasm.
3. Subluxation of a primary traumatic cause, such as hyperflexion.

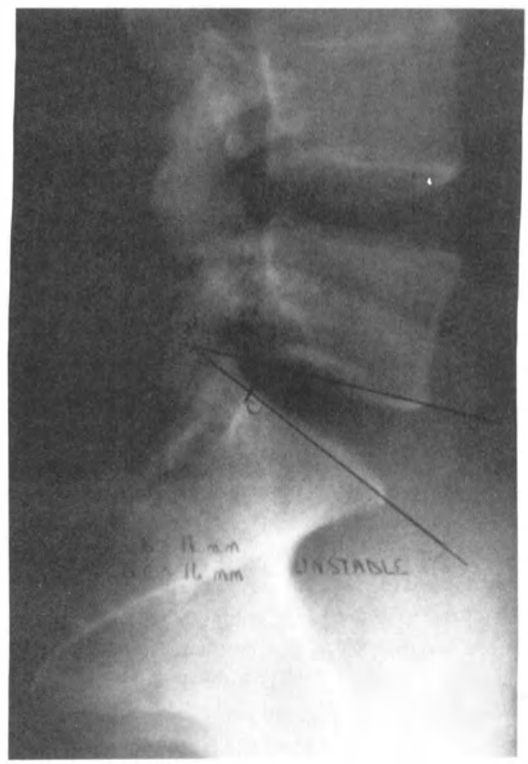

Figure 13.27. Radiograph showing an unstable facet syndrome of L5 on the sacrum with L5 being 5 mm posterior on the sacrum, a retrolisthesis subluxation of L5. Note the apparent stenosis of the intervertebral foramen caused by the facet imbrication of the first sacral facet and the posteriority of L5 on the sacrum.

Willis (42) found the depth of the last lumbar vertebra to be greater than the first sacral segment, which he felt gave rise to an optical illusion on x-ray film of backward displacement of the fifth lumbar vertebra on the sacrum. He stated that, in measuring 50 skeletons, the depth of the L5 and S1 bodies were found to be equal in 34% of the cases; in the other 66%, lumbar depth exceeded sacral depth or was less in a few cases.

I feel that in facet syndrome in which discal degeneration occurs, the increased facetal weightbearing will force the inferior fifth articular facet to impact the first sacral facet and cause a posterior displacement of the fifth body as the two segments approximate one another. In turn, the only means of returning some degree of alignment is to open the disc space and relieve the impaction hyperextension subluxation of L5 on the sacrum. In the end, the clinical result and relief of patient symptoms will depend on effective clinical application of manipulative principles based on anatomic abnormality.

Examination should reveal the following in retrolisthesis:

1. Underdevelopment of the pedicles resulting in probable sagittal stenosis as measured by Eisenstein's procedure, described in Chapter 4, *Spinal Stenosis.* With this shortened pedicle and the resultant posterior placement of L5 on the sacrum, George's line will show a posteriority of L5 on the sacrum. George's line is shown in Figure 13.27 as the line behind the L5 body. In the figure, it is not a smooth line continuing behind the sacrum; rather, it breaks as it shifts anterior to the posterior sacral position. George's line can be continued behind all the lumbar bodies. It normally is a smooth, uninterrupted line behind the normal lumbar lordosis.
2. Flattening of the lumbar lordosis on physical examination.
3. In some cases, spasm over the paravertebral musculature, which is tender when touched; gluteus maximus spasm and tenderness, and/or an adductor muscle that is spastic and tender to touch.
4. Radiation of pain into the groin, buttock, posterior thigh, and flank, as described previously.

Treatment

Treatment is shown in Figures 13.28 through 13.31. Figure 13.28 shows extension manipulation being applied gently on the manipulative instrument. We avoid thrusting into this segment, as it can be painful to the patient. By using extension manipulation we can also use lateral flexion with extension, as shown in Figure 13.29 to place the articular facets through their physiologic ranges of motion.

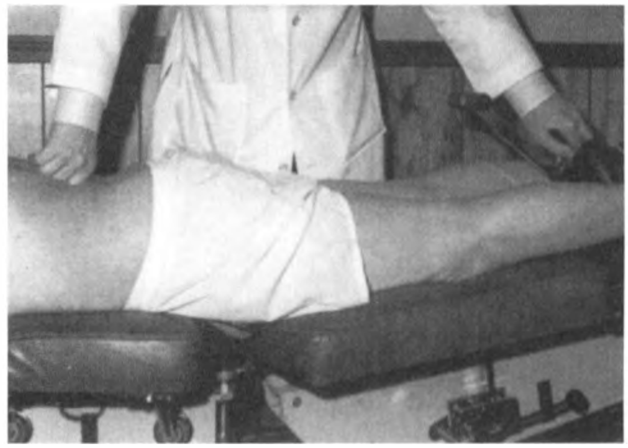

Figure 13.29. Following extension tolerance by the patient, the facets can be placed through lateral flexion and circumduction. We use this motion only on patients who have regained full range of mobility without pain in the flexion posture with the table. The doctor must be sensitive to the infliction of stenosis by such extension motion at L5–S1 and must test the patient's ability to take this type of manipulation prior to its application. We use it only on those patients who feel marked relief from extension position manipulation, which negates its use in elderly persons with intermittent neurogenic claudication caused by stenosis.

Figure 13.28. Shown is extension manipulation being applied to a retrolisthesis subluxation of L5. The table is gently brought into extension as a downward pressure is applied to the spinous process of L5.

Figure 13.30. Placing the patient on his or her side for the application of extension manipulation is an excellent method. It allows complete control of the depth of extension while allowing the contact hand to detect and control the extension forces being applied.

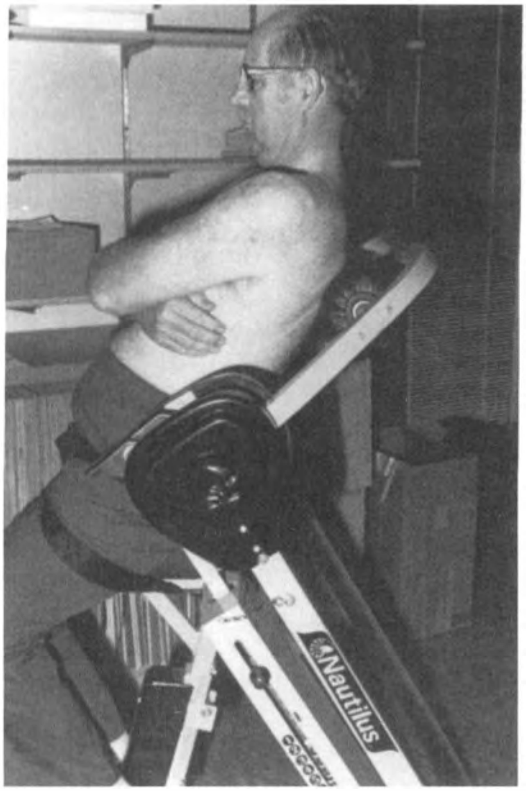

Figure 13.31. Following relief of pain in facet syndrome and retrolisthesis, we use extension Nautilus conditioning for the paravertebral muscles. We prefer using a maximum of 130 pounds of extension force. We start the patients, even elderly little ladies, on 20 to 30 pounds of resistance and build them up.

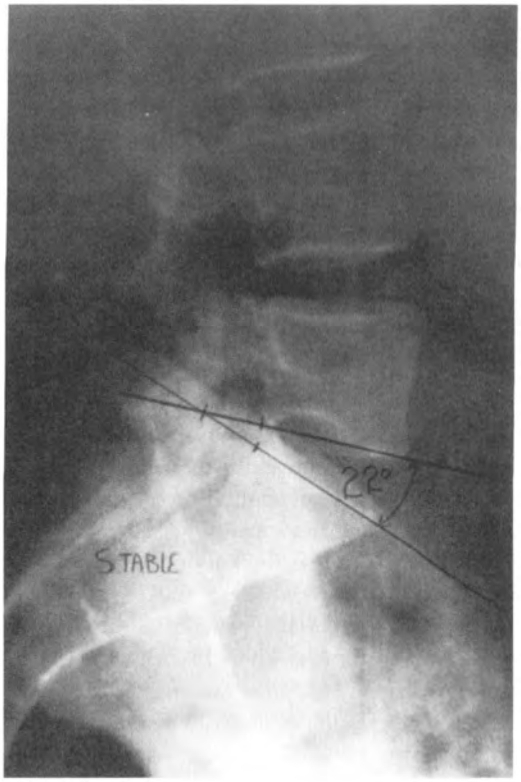

Figure 13.32. Spot lateral view shows the 22° discal angle and a stable facet syndrome. The superior S1 facet is telescoped into the L5–S1 foramen, and nuclear invagination of the L5–S1 disc into the inferior end plate of L5 is seen.

Figure 13.30 shows treatment being applied with the patient on his side. Two purposes are found for this technique. First, it is excellent for the patient who has too much pain to lie on the abdomen for care. Also, it is an excellent modality for placing the patient into extension while controlling the motion of the vertebrae with the contact hand on the spine.

In addition to manipulation, other modalities used in facet syndrome include goading of acupressure points B22 to B49, alternating hot and cold packs, massage, electrical stimulation, belt support in severe pain, exercises, low back wellness school, and Nautilus exercise, as shown in Figure 13.31.

CONCLUSION

This concludes the discussion of the mechanics and treatment of probably the most common condition encountered in low back pain patients—facet subluxation syndromes. This chapter concludes with a case presentation of facet syndrome.

Case Presentation

A 51-year-old woman was seen for the chief complaint of low back pain and no leg pain. It had worsened this time following yard work, but she had low back pain off and on for most of her life.

Examination revealed +2 deep reflexes bilaterally, no motor weakness, and no sensory abnormalities. The ranges of motion of the thoracolumbar spine were normal, and straight leg raises were negative.

Figure 13.32 reveals a stable facet syndrome at the L5-S1 level. Note that the discal angle is 22°. The greater the discal angle, the greater the severity of the facet syndrome, as indicated by the thinning of the posterior L5–S1 disc space. In this case, we do have nuclear invagination of the inferior vertebral plate of L5 by the intervertebral disc. The posterior disc space is markedly thin compared with the anterior.

Treatment consisted of distraction manipulation with a small flexion pillow under the L5 vertebral body. Deep goading of the paravertebral muscles over the acupressure points B22 through B49 was used in preparation for distraction manipulation. This patient was given knee-chest exercises, abdominal strengthening exercises, and hamstring stretching. Three visits resulted in almost total relief of the low back pain.

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Spondylolisthesis

James M. Cox, DC, DACBR

chapter 14

For of those to whom much is given, much is required.

—John F. Kennedy

HISTORICAL DATA

Herbinaux (1) in 1782 was the first to recognize spondylolisthesis as a cause of obstruction in his obstetric cases, but Killian (2) was the first to describe and name it, calling it a slow subluxation of the posterior facets. Robert (3) believed that some defect in the neural arch must be present, and Neugebauer (4) recognized that the slip could occur with or without a neural defect.

Figure 14.1 is an illustration of the normal L5–S1 locking mechanism of the intact intervertebral disc (IVD) stabilizing the L5 vertebral body to the sacrum, of the neural arch solid bone stabilizing the anterior body to the arch, and of the articular facets locking the entire functional spinal units of L5 and the sacrum. Figure 14.2 is an illustration of the progressive slippage that occurs in a person from birth through development.

CLASSIFICATION

In 1963, Newman (5) classified spondylolisthesis into five types. His classification, which follows, is still valid and useful today.

1. Dysplastic (congenital). Congenital abnormalities of the upper sacrum or the arch of L5 permit the "olisthesis" to occur.
2. Isthmic, in which the lesion is in the pars interarticularis. Three kinds can be delineated:
   - Lytic, which is a fatigue fracture of the pars
   - Elongated but intact pars
   - Acute fracture of the pars (not to be confused with "traumatic," see 4)
3. Degenerative, caused by a longstanding intersegmental instability.
4. Post-traumatic, caused by fractures in areas of the bony hook other than the pars.
5. Pathologic (i.e., generalized or localized bone disease).

Dysplastic Spondylolisthesis

Congenital or dysplastic spondylolisthesis occurs at L5–S1, with defects of fusion of the neural arch occurring in the upper sacral vertebrae as well as at L5. Hypoplastic facets of the sacrum develop, which fail to provide sufficient resistance to the forward shear force of L5 on S1 (6). The L5 arch may reveal spina bifida, which occurs in girls twice as frequently as it occurs in boys. During the growth spurt between ages 12 and 16, the condition commonly manifests itself, probably because of increased weightbearing and stress. The pars interarticularis either elongates or separates (6). The dysplastic type of spondylolisthesis can be difficult to differentiate from the isthmic type on radiography. A strong genetic association is found in dysplastic spondylolisthesis (7), and a study by Wynne-Davies and Scott (8) showed that one of three (33%) relatives of patients with dysplastic spondylolisthesis will be affected.

Isthmic Spondylolisthesis

Isthmic spondylolisthesis is the most common type of spondylolisthesis, and it is caused by a defect in the ossification of the pars interarticularis. Three subdivisions of isthmic spondylolisthesis have been delineated: the lytic (subtype A), an elongated pars without separation (subtype A), an elongated pars without separation (subtype B), and an acute pars fracture (subtype C). Subtype A can be seen in Figure 14.3.

Spondylolysis

"Spondylolysis" is a term applied to the mechanical failure of an apparently normal isthmus. This occurs most frequently at the L5 level, less frequently at the L4 level, and rarely at levels above L4. It is no longer questioned that spondylolysis is a fracture that may or may not heal. These fractures are postulated to occur because of the assumption of the upright pos-

ture by the infant, allowing a fatigue type of fracture to occur when stress beyond the strength of bone occurs. Rosenberg et al. (9) obtained radiographs of the lumbosacral spines of 143 patients who had never walked. The frequency of spondylolysis and spondylolisthesis as well as of other spinal abnormalities was determined. The average age of the patients was 27 years, with an age range from 11 to 93 years. The underlying diagnosis responsible for the nonambulatory status varied, but cerebral palsy predominated. No case of spondylolysis or spondylolisthesis was detected, and this is significant when it is compared with the 5.8% incidence in the general population. The incidences of spina bifida (8.4%) and of transitional vertebra (10.9%) were similar to those found in the general population. Scoliosis was found in 49%, and vertebral body height was increased in 33%. Degenerative changes occurred in only 2.8%. These results support the theory that spondylolysis and isthmic spondylolisthesis represent fatigue fractures resulting from activities associated with ambulation.

According to Scoville and Corkill (10), King studied 500 normal school children on whom he conducted x-ray studies at the ages of 6, 12, and 18. He did the same with 25 children with back problems. He found almost no progression or development of spondylolisthesis after the age of 6 years in any of these children. True spondylolisthesis rarely if ever progressed after the patient reached maturity. Pfeil (11) showed that the infant spine is susceptible to fatigue fracture in the isthmus.

The isthmus can be seen in Figure 14.4. Two layers of cortical bone are found here, the anterolateral and the posteromedial, which are joined by parallel thick trabeculae directed inferolaterally and anteriorly from the base of the superior ar-

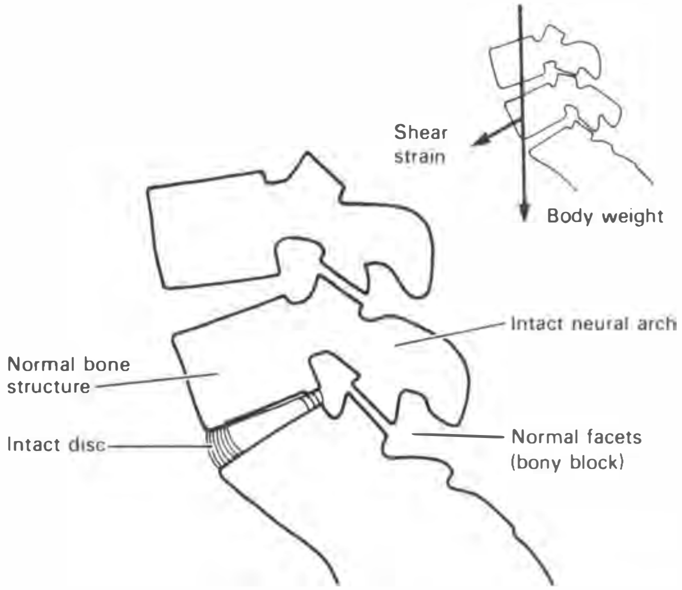

Figure 14.1. Illustration of normal locking mechanisms resisting forward displacement of the fifth lumbar vertebral body. (Reprinted with permission from Macnab I. Backache. Baltimore: Williams & Wilkins, 1977:45.)

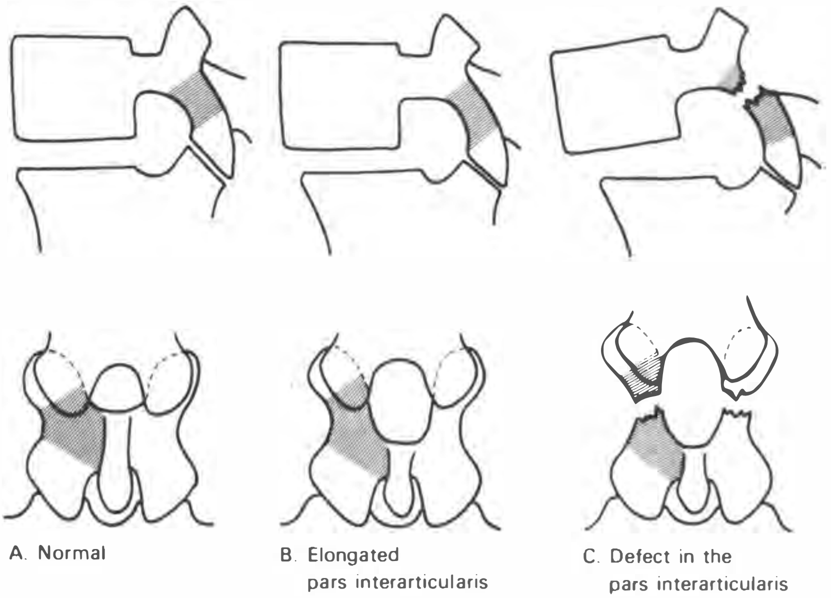

Figure 14.2. Illustration of isthmic spondylolisthesis. The pars interarticularis, which was normal at birth (**A**), becomes attenuated and elongated, allowing the vertebral body to slip forward in relation to the vertebral body below (**B**). Eventually, the elongated pars interarticularis may break (**C**). This defect in the pars interarticularis is, however, secondary to the slip and is not the cause of the forward displacement of the vertebral body. (Reprinted with permission from Macnab I. Backache. Baltimore: Williams & Wilkins, 1977:46.)

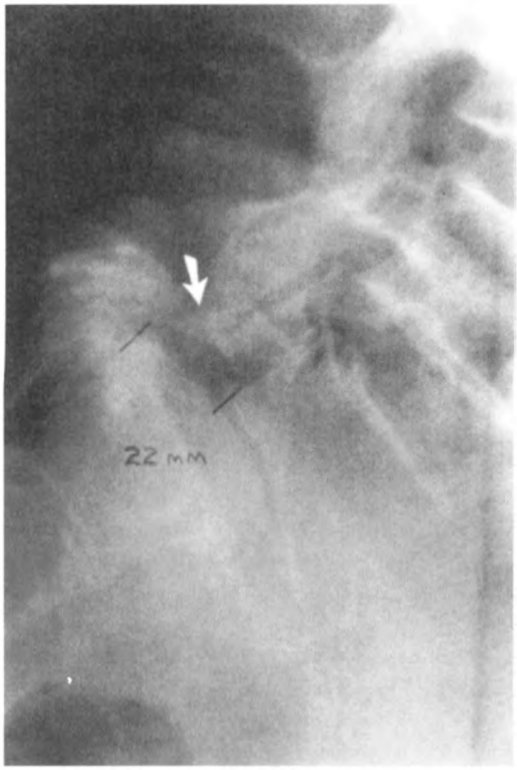

Figure 14.3. A lytic fatigue fracture defect of the pars interarticularis of L5 (*arrow*) is shown as the cause of this 22-mm slippage of L5 on the sacrum.

ticular process (12). The anterolateral layer is the thicker of the two, and it appears to be capable of resisting forces that tend to bend the inferior articular processes posteriorly or posteromedially, which are induced whenever the effect of gravity is transmitted to a vertebra inclined below the horizontal anteriorly, and are induced when the vertebra is exposed to axial torque. Sullivan and Farfan (13) studied the effect of axial torque, which tends to disrupt the inferior articular processes; they believe that such damage predisposes to spondylolysis.

INCIDENCE OF SPONDYLOLYSIS AND SPONDYLOLISTHESIS

A study (14) reported by Wiltse stated that if 100 children aged 5 were to be studied radiographically, probably not one would be found with a defect of the pars. If the same children were examined toward the end of the first grade (age 7), however, the incidence would be approximately 4.4%, which is just slightly below the national average. Baker [as reported in Finneson (15)] found that as these children reached age 18, only 1.4% more showed spondylolisthesis, with most of the increase occurring between ages 11 and 16, the time of participation in the most strenuous athletics, which produce fatigue fractures.

One reason that forward slippage occurs most often in children aged 5 to 7 years may be because of the increased activity or to the increased sitting in the lordotic posture done by children. It is known that fracture never occurs in animals other than humans, and only humans have lordosis (16–18). Average age at onset of symptoms of spondylolisthesis is 14 in girls and 16 in boys (19).

The severity of symptoms and the treatment of spondylolisthesis in the child vary greatly from that in the adult. Surgery may be more imperative in the child than in the adult because further slippage occurs more often in the child. Furthermore, the outcome of fusion is better in the child than in the adult, with the adult being more willing to curtail activities, to prevent further aggravation of the condition. It is also known that, following surgery, greater relief from pain is seen in the child than in the adult. For the adult, the prime reason for surgical treatment is to relieve pain, not to prevent progression of slippage. Slippage rarely increases in the adult (15).

Semon and Spengler (20) found that in a large group of college football players, spondylolysis was not a predisposing factor to low back pain. Furthermore, the mere indication of spondylolysis or spondylolisthesis on x-ray film did not mean that spondylolysis or spondylolisthesis caused the person's low back pain. Newman (1) observed that, despite the obvious displacement at the L5–S1 intervertebral joint, the symptomatology seems to derive from the L4–L5 joint. This would be logical, because the forward slippage of L5 does allow the superior facet of L5 to enter the intervertebral foramen in a telescoping effect at the L4–L5 level. Furthermore, at the time of the slippage, either or both discs (i.e., the L4–L5 or L5–S1) must break down, allowing anular stretching and tearing. Without this phenomenon, no forward slippage of the vertebra could occur. This would be true even if growth defects were seen within the arch, namely, pars interarticularis fracture. The disc, being a very pain-sensitive structure, certainly creates symptomatology as the slippage occurs. Perhaps it is understandable why in the adult, after this slippage occurs and the anular fibers heal, the pain lessens or disappears.

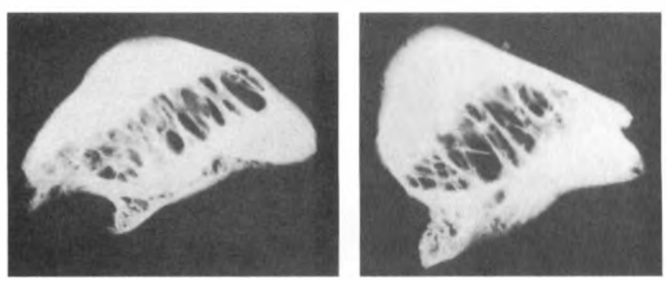

Figure 14.4. Photograph of two slices through the isthmus from the fifth lumbar vertebra of a 66-year-old man, which were cut parallel to the plane of the narrowest perimeter of the isthmus (i.e., the plane of a spondylolytic defect). This is typical of the normal appearance of the isthmus. The anterolateral layer of cortical bone can be seen in the **upper left** region of the slices. (Reprinted with permission from Krenz J, Troup JDG. The structure of the pars interarticularis of the lower lumbar vertebrae and its relation to the etiology of spondylolysis. J Bone Joint Surg 1973;473 (55B):735.)

Anatomy of the Partes Interarticularis Defect

A pars defect is visible on the x-ray film and in a cadaver specimen (Fig. 14.5); the actual specimen dissected out at necropsy can be seen in Figure 14.6.

In Figure 14.7, a discogram of the L4–L5 level, disruption of the anular fibers is certainly seen, which allowed dye to escape from the nucleus into the perimeter of the disc. This demonstrates the tearing that would occur in the anulus at the time of slippage.

In a study of facet joints with the use of arthrography, an abnormal communication between the two facet joints bordering the separated pars interarticularis was observed in 9 of 11 patients. This communication occurred in the area of the defect. In one patient with bilateral spondylolysis of the L5 vertebra, both left adjacent apophyseal joints were observed to communicate not only with one another but also with the contralateral facet joints through a transverse channel joining the isthmic areas of L5 (21). Furthermore, it was found that spondylolysis considerably altered the soft tissues of the adjacent facet joints. Irritation of these structures might explain certain complaints such as low back and scleratogenous pain in patients with spondylolysis.

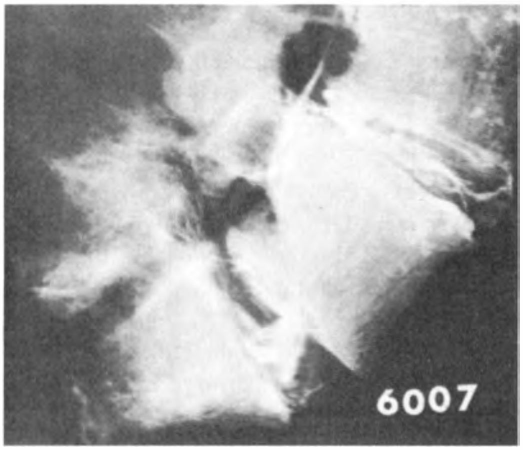

Figure 14.5. Radiograph showing spondylolysis in a cadaver specimen. A defect of the inferior articular process is clearly visible. The lumbosacral disc shows degeneration, but this does not appear to be as advanced as that at the L4–L5 level. (Reprinted with permission from Farfan HF. Mechanical Disorders of the Low Back. Baltimore: Williams & Wilkins, 1973;7:164.)

Among the causes of spondylolisthesis, the fifth lumbar vertebra, placed at the apex of the lumbar curve, is probably the recipient of the highest stress on flexion and rotation movement. If L5 is well anchored to the pelvis by enlarged transverse processes, the same findings may well be seen at the L4 level. According to Farfan (16), during forced rotation the neural arch is placed under such stress that a permanent sprain can occur to it. This sprain could take two forms:

1. The interarticular distance between the inferior facet articulations is reduced, which may allow the sprained neural arch to slip through the other.
2. The angle of these processes to the axis of the pedicle would be increased from a normal angle of about 90° to an abnormal angle of about 130°.

This stress produces an apparent lengthening of the pedicles, which in turn could allow the forward slip of the affected vertebra. Farfan further believes that the defect in the lamina is probably a fracture at the junction between the laminae and the pedicle, as the angle between these structures is opened. Furthermore, the injury at the disc is an epiphyseal separation of the superior epiphysis of the sacrum.

Figure 14.6. Photograph of L5 isolated from the same specimen as in Figure 14.5. (Reprinted with permission from Farfan HF. Mechanical Disorders of the Low Back. Baltimore: Williams & Wilkins, 1973;7:165.)

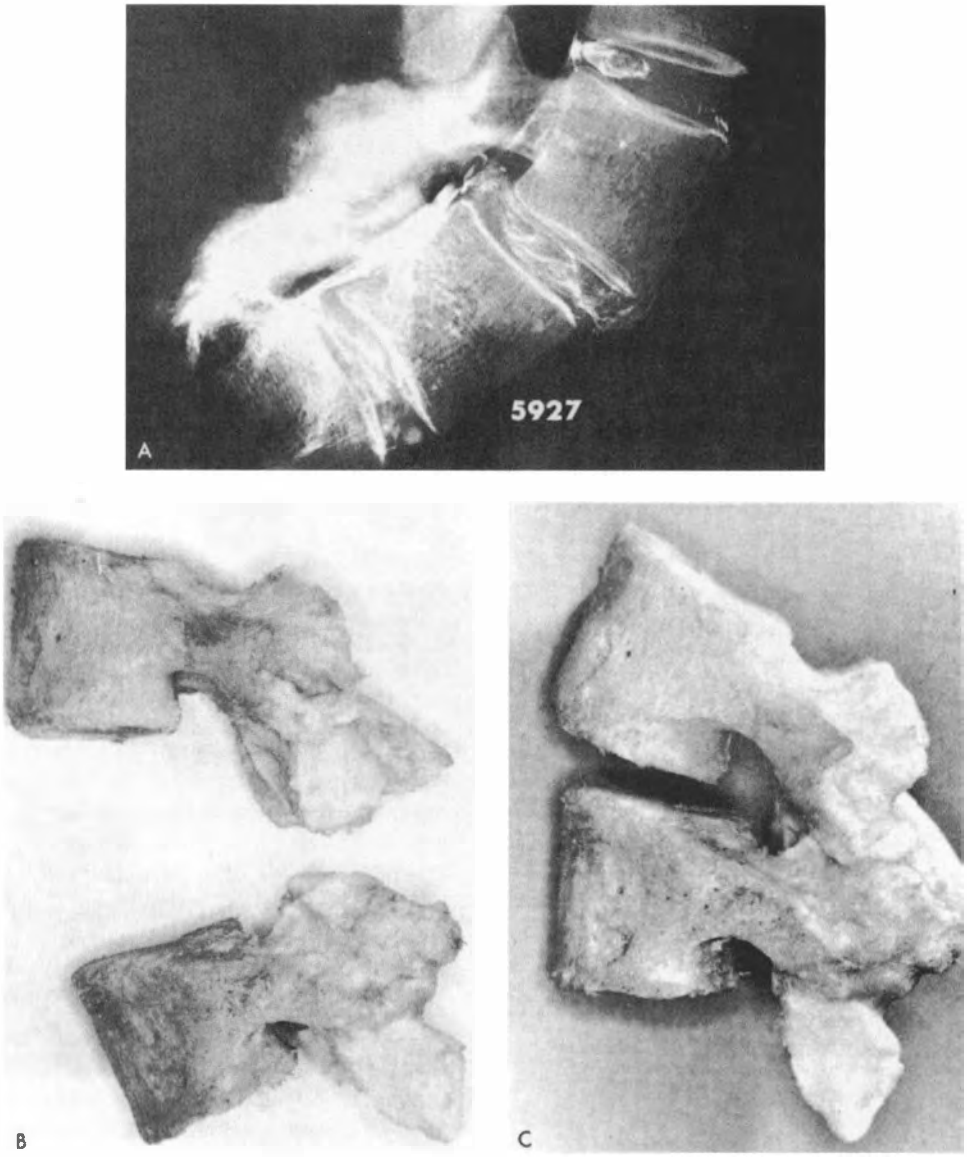

Figure 14.7. **A.** Discogram showing spondylolisthesis of L4 on L5 in a cadaver specimen. No defect is seen in the pars interarticularis; however, there appears to be a prolonged inferior articular process. The disc is degenerated. **B.** Skeletal arrangement. The specimen does not show a true elongation. **C.** The apparent elongation is caused by superimposition of subluxated superior and inferior articular facets and to the widening of the angle between the lamina and pedicle. (Reprinted with permission from Farfan HF. Mechanical Disorders of the Low Back. Baltimore: William & Wilkins, 1973:167.)

PAIN ORIGIN IN SPONDYLOLISTHESIS

Pars Interarticularis Defect

Free nerve endings within the pars defect tissue can be a source of back pain in some patients with symptomatic spondylolysis (22), with activities of daily living stimulating these neural structures to levels of nociception as a result of peripheral or central sensitization (23).

Tissue from the spondylolysis defect shows delayed union or pseudoarthrosis with fibroblasts and macrophages in a pseudosynovial lining membrane and occasional perivascular infiltrates containing mainly CD2 lymphocytes and CD11b monocytes or macrophages. Pain in spondylolysis or spondylolisthesis might derive from the spondylolytic defect itself, probably from stretching of the local neural elements rather than from their sensitization or stimulation by local inflammatory mediators (24).

Sacral Base and Protruding Disc

Forward slippage of L5 and the posterior displacement of S1 posteriorly and cranially into the superior recess of the L5–S1 neural foramen produces encroachment of the neural canal. This process takes place without a true herniation of the IVD

and with only relatively minor degrees of spondylolisthesis (25). Figures 14.8 and 14.9 demonstrate the pseudoherniation of the L5–S1 disc into the vertebral canal as a broad-based nonfocal bulging disc. Note that the disc bulging does not materially contact the thecal sac, and this patient has relatively benign low back pain that healed well with conservative chiropractic distraction adjusting.

Spondylolytic spondylolisthesis can and does occur without symptoms. It is known that Eskimos have a 40 to 50% occurrence of spondylolisthesis but not that high an incidence of pain with it. Forward slippage of the body will not occur without degenerative changes in the underlying disc (i.e., forward slippage is not possible without anular tearing or breakdown). The disc is not capable of withstanding the shearing stresses of the body above on the one below.

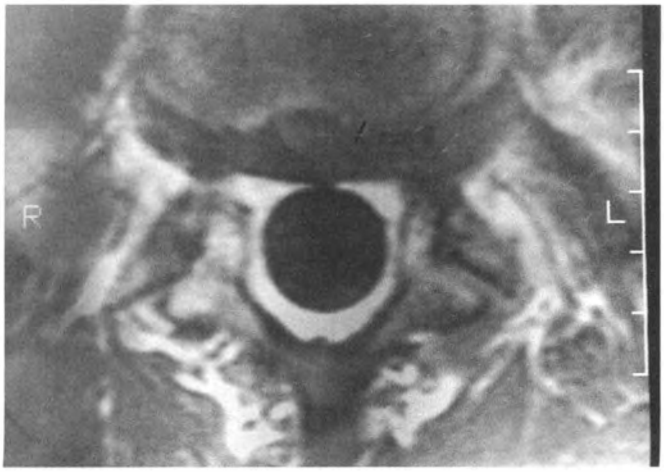

Figure 14.8. The *arrow* in this axial view demonstrates the broad-based, nonfocal pseudobulge of the disc in spondylolisthesis, which narrows the sagittal vertebral canal diameter without thecal sac compression but with lateral recess narrowing.

Figure 14.9. Sagittal view of the pseudodisc bulge seen in Figure 14.8.

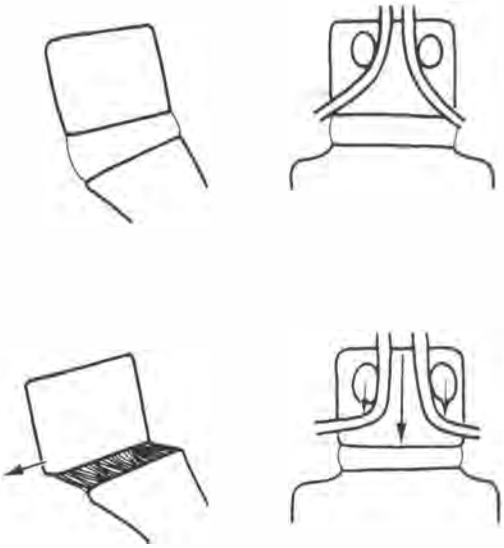

Figure 14.10. Illustration of kinking of the nerve roots by the pedicles as the body of L5 slips downward and forward. (Reprinted with permission from Macnab I. Backache. Baltimore: Williams & Wilkins, 1977:54.)

In a study comparing the incidence of pain in patients with spondylolisthesis by age, Macnab (19) divided patients into three age groups (under 26, 26 to 39, and 40 and older). In the 40 and older group, the incidence of spondylolisthesis in patients with back pain was approximately the same as it was in the general population, whereas in the under 26 group, nearly 19% of back pain patients exhibited spondylolisthesis. Thus, spondylotic spondylolisthesis found in a patient under 26 years of age who does have back pain probably is the cause of the symptoms; if spondylotic spondylolisthesis is found in patients 26 to 39 years of age, it is a possible cause; and if it is found in patients 40 years of age or older, it rarely, if ever, is the sole cause of symptoms.

Figure 14.10 shows how L5 spondylolisthesis kinks the L5 nerve root passing under the L5 pedicles. This can be confused with root symptoms caused by L4–L5 disc protrusion. An L4 spondylolisthesis could kink the L4 nerve root and cause femoral nerve paresthesia.

MECHANISMS OF NERVE ROOT COMPRESSION IN SPONDYLOLISTHESIS

Macnab (19) described at least six mechanisms of compression of the L5 root in isthmic spondylolysis:

1. Disc herniation of L4–L5.
2. The free fragment of the L5 posterior neural arch rotating

anteriorly and pivoting on the sacrum, with compression of the L5 root between the distal pars remnant and the sacrum.
3. Occasional kinking of the L5 root around the L5 pedicle in spondylolysis.
4. Encroachment by a degenerative, bulging anulus fibrosus at L5–S1.
5. Neuroforaminal stenosis.
6. Extraforaminal entrapment between the L5 corpotransverse ligament and the sacral ala.

Clinically, pseudospondylolisthesis results in stenosis of the lumbar spinal canal, and it can impinge on the nerve roots of the cauda equina and induce neurogenic claudication (26).

Vibrational Effects in Spondylolisthesis

Helicopter pilots, because of vibrational forces, have been found to have a significantly higher incidence of spondylolisthesis than transport pilots or cadets (27). In a study of 21 pilots with spondylolisthesis followed for 12 to 131 months, 16 had follow-up examination. Only one was found to have significant progression of the displacement. Of the 12 pilots with spondylolisthesis who had back pain, all continued to fly. The other nine pilots did not develop pain. It was concluded that pilots with spondylolisthesis could continue to fly with minimal risk of morbidity and loss of flight time (28).

Spondylolysis Is Questionable As Cause of Back Pain

Hall (29) interestingly points out that the incidence of spondylolysis was higher in asymptomatic persons (9.8%) than in those with low back pain (9.2%). He further concluded that pre-employment x-ray examination does not have a high predictive value for future back problems and is not worth the radiation risk.

SPONDYLOLISTHESIS IS RISK FOR RECURRENT BACK PAIN WITH HIGH PHYSICAL DEMANDS

Spondylolysis is essentially a stress fracture, which usually occurs in early adolescence and heals with fibrous tissue to a point of significant stability. The radiographic incidence of this abnormality is about 4.5%. Most persons remain asymptomatic indefinitely. Thus, unless pre-existing complaints of pain under severe physical demand exist, no justification is seen for limiting sports or strenuous physical labor.

Spondylolisthesis is a different story, however. The greater the displacement, the greater the risk of recurrent back problems associated with high physical demand. Limits on demanding physical or recreational activity are therefore appropriate (30).

The incidence of spondylolysis is cited at 5% of persons with bilateral defects, whereas 1% have unilateral defects (31).

Spondylolisthesis Disability

Mild to moderate spondylolisthesis detected by chance in a middle-aged population does not predispose to more disabling back pain than back problems experienced by those without spondylolisthesis. However, women with spondylolisthesis have mild back symptoms more often than control subjects (32).

Patients with defects of the L5 arch suggest that a low-grade spondylolisthesis does not invariably lead to severe physical impairment or frequent permanent disability, with the possible exception of patients with defects at the L4 level (33).

In a prospective study of college football players, the incidence of asymptomatic spondylolisthesis was 4%. The incidence of back pain did not differ in persons with or those without a pars defect (34).

Slippage and Ongoing Pain

Only patients with a spondylolisthesis greater than 25% were found to have ongoing low back pain. Therefore, work restrictions are unwarranted (34).

CONDITIONS INFLUENCING SYMPTOMS

Genetics

Radiographs of the lumbar spine in 130 close relatives of 45 patients with lumbar spondylolisthesis showed spondylolysis or spondylolisthesis in 37 (28.5%). The occurrence of spondylolisthesis is more than *four times higher* than the incidence (6%) in the general population (35).

Diabetics

A decreased prevalence of lower back pain among diabetic patients is reported; it might be attributable to increased nonenzymatic glycosylation of connective tissue proteins in juxta-articular tissues, which may make these tissues stiffer and less liable to small pain-inducing subluxations. It is possible that diabetic patients have an even higher prevalence of spondylolysis than nondiabetics (36).

Oophorectomy

Oophorectomy has been shown to provoke an abrupt decrease in serum estrogen level and a deficit of testosterone and androstanedione, which are also secreted by the ovary. It appears that the loss of elasticity in the paraspinal ligamentous system produced by hormonal changes caused by oophorectomy can contribute to degeneration and to the development of the vertebral slip (37).

Age, Hamstring Length, Pregnancy, Slippage

Patients with hamstring muscle contracture showed a higher degree of spondylolisthesis and greater disc degeneration than patients without hamstring contracture (38).

Age at symptom onset in the patients with L5 spondylolisthesis was 19 years, and the age at radiographic diagnosis was 23, whereas the L4 spondylolysis patients showed symptoms at 20 years and were radiographed for diagnosis at 30 years. Occasional low back pain occurred in 91% and chronic pain was found in 73%; 60% found their pain constant, with 79% finding loading of the lumbar spine to be pain-producing.

Of the 255 patients studied 55% reported having had sciatica (38); 70% had received treatment for low back pain. A comparison of surgically treated versus nonsurgically treated patients showed no statistically significant differences in frequency of symptoms and functional impairment, degree of spondylolysis at diagnosis, or progression of slippage.

Pregnancy showed no statistically significant differences in frequency of symptoms, functional impairment, or degree of progression of slippage when 63 pregnant women were compared with 21 women who had never been pregnant, and to 171 men.

STABILITY OF SPONDYLOLISTHESIS AND ITS THERAPEUTIC IMPLICATIONS

Every case of spondylolysis or listhesis I see on radiography raises the question: How much of this patient's pain is caused by the defect and slip and how much is from other causes? Disc instability and facet hypoplasia determine the degree of forward slip of the spondylolisthesis segment (39). Iliolumbar ligament weakness accompanies L5 transverse process lack of thickness and results in instability at the lumbosacral junction (40).

To present the implications of translational instability and its determining factors on pain and treatment response, I will next print a research study on 10 cases of true spondylolisthesis I published using Friberg's diagnostic work and distraction adjusting. This paper covers the how and why in treating spondylolisthesis with distraction adjusting.

CHIROPRACTIC ADJUSTMENT RESULTS CORRELATED WITH SPONDYLOLISTHESIS INSTABILITY[1]

Summary

Ten true spondylolisthesis patients, nine with the lesion at L5 and one at L3, were tested by vertical suspension radiography compared to neutral lateral weight-bearing X-ray to determine translational segmental instability. Cases were classed as unstable (high instability) if over 3 mm of translation of the spondylolisthetic segment occurred and as stable (low instability) if less than 3 mm of motion was seen. Chiropractic distraction adjustment was applied in each case, and the response to care was evaluated by subjective rating of pain relief. Results found that all five patients with stable spondylolisthesis cases obtained 75% or greater relief from chiropractic adjustment of the type used by the author, whereas one with the unstable variety experienced over 75% relief while the other four had less than 50% relief of pain. As defined in this paper, stable true spondylolisthesis seems to respond better than the unstable variety.

Fifty percent of patients with spondylolysis develop spondylolisthesis. Pain is the most common symptom, with the peak age for the onset of symptoms occurring during the adolescent growth spurt. Spondylolisthesis is the most common cause of low back pain and sciatica in children and adolescents, but most adolescents with spondylolysis are asymptomatic (1).

The severity and frequency of low back pain symptoms does not show correlation with the degree of spondylolisthesis segment (1).

The treatment result of stable (low instability) vs. unstable (high instability) spondylolisthesis cases under chiropractic adjustment has not been addressed. The study reported in this paper was designed to answer this question. Stability was determined by radiographic measurement of translatory motion of the spondylolisthetic segment during movement from neutral lateral standing to axial loading of the spine (4). The relief obtained from the chiropractic adjustment was then documented.

Methods

Ten patients, seven men and three women, age 24–61 years, were radiographed in neutral lateral(Fig. 1) and axial traction by hanging suspension (Fig. 2) as described by Friberg (4).

Translational motion of one vertebral functional motor unit upon its adjacent segment by 3 mm as the spine is flexed and extended has been considered physiological motion; greater than 3 mm movement is felt to represent abnormal translational motion (3, 10, 11). One group felt their study indicated that up to 4 mm of translational motion was physiological (6).

Stability of the spondylolisthesis segment was defined as less than 3 mm of translational movement seen between the vertical suspension X-ray and the neutral lateral standing X-ray. Instability was defined as translational movement of the spondylolisthesis segment on vertical traction greater than 3 mm from the slippage seen on neutral lateral X-ray view.

X-ray examination of a 33-year-old man in neutral lateral, flexion, extension, and axial traction is illustrated to demonstrate the method used in this study (Figs. 3–6). Figure 3 reveals neutral lateral slippage of L5 on the sacrum by 10.5 mm. Figure 4 shows that in flexion the slippage actually reduces by 0.5 mm, while extension (Fig. 5) shows a 0.5 mm reduction of the slippage from the neutral lateral projection. Figure 6 is the

[1] J Manual Medicine 1991;6:67.
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References for this paper are at the end of the paper on page 623.

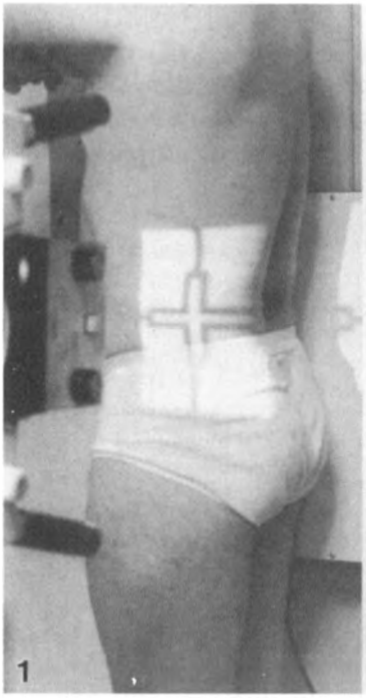


Figure 1. Neutral lateral standing posture for radiography of the lumbosacral spine

Figure 2. Hanging suspension study by Friberg [4] for study of translatory instability of the spondylolisthesis segment

axial hanging suspension traction study as shown in Fig. 2, which shows a reduction to 5.5 mm, representing translatory instability of 5 mm for L5 on S1.

Figure 7 is the neutral lateral X-ray of an L3 true spondylolisthesis slip of 8 mm, which was reduced by 3 mm to a 5-mm slip on the vertical suspension study (Fig. 8). Figure 9 demonstrates a 10-mm L5 slippage on the sacrum in neutral lateral projection and no slippage on axial distraction suspension (Fig. 10).

Treatment in the ten cases consisted in distraction manipulation as demonstrated in Fig. 11. A small flexion roll is placed under the spondylolisthesis segment, and the doctor's hand is placed with thenar contact for manipulation on the spinous process above the spondylolisthesis segment. The upward migration of the superior facet of the spondylolisthetic segment into the osseoligamentous canal (intervertebral foramen) at the level directly above can induce stenosis and possible nerve root compression as well as facet irritation. The treatment goal is to lever the spondylolisthesis body posteriorly by flexing and distracting the lumbar spine into hypolordosis or slight kyphosis, thus increasing the vertical and sagittal diameter of the intervertebral foramen directly above the spondylolisthesis segment. With a cephalic lift to the spinous process by the doctor's thenar contact, the segment is lifted gently as caudal distraction is applied with the caudal section of the table at a rate tolerable to the patient. That is, three 20-second distraction sessions are applied and during each session, the spinous process is lifted cephalad five or six times as the caudal table section is placed to apply distraction to the lumbar spine. No more than 1 to 2 inches of downward table motion is allowed under distraction. This limits the amount of distraction to safe parameters, especially since the flexion roll is placing the lumbar spine into slight kyphosis. This distraction adjustment opens the posterior arch and disc space while relieving segmental facet dysfunction at the level above the spondylolisthesis slippage. Attention must also be paid to possible sacroiliac joint dysfunction and appropriate correction made.

Specific exercises are given to the patient to do at home. These are shown in Figs. 12 and 13 and consist of knee-chest flexion and hamstring stretching.

The patients attended the Low Back Wellness School to learn how to bend, lift, and twist the spine in daily living to prevent pain and disability.

Patient relief was subjectively graded on a scale of 1 to 4. One was 75% or greater relief of pain; two, 50% or greater relief; three 25% or greater relief, and four, nil relief. Data was then analyzed using correlation and regression to determine the stability of the patient's spondylolisthesis and the amount of relief obtained from the treatment. Potential effects of gender, age, and the number of treatments on patient relief were also analyzed (9).

Results

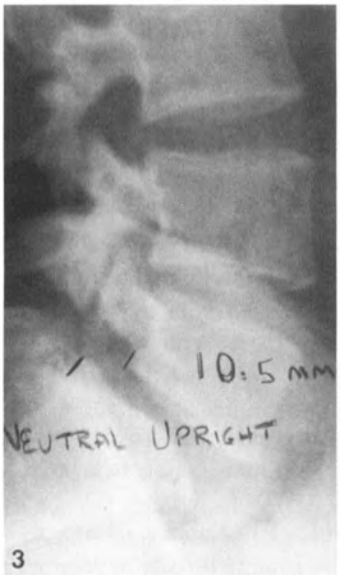
Table 1 shows the patient characteristics of age, sex, weight, height, level of involvement, neutral lateral standing slippage, vertical standing suspension slippage, the amount of movement from neutral to vertical suspension, the percentage of total reduction of the slippage from neutral to vertical suspension, the degree of stability as measured by being stable (low instability) if there was less than 3 mm of motion from neutral upright X-ray to vertical suspension or unstable (high instability) if there was greater than 3 mm of motion, the response to treatment, and the number of adjustments given to the patient.

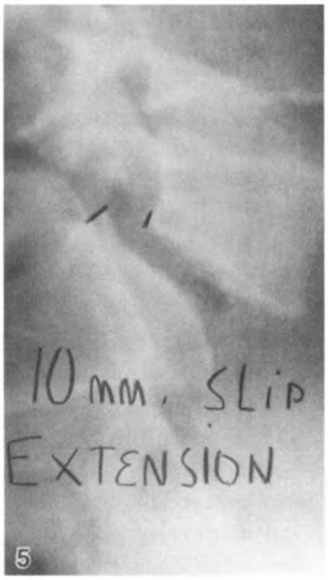
Of the five patients who had stable spondylolisthesis, all reported 75% or greater relief from treatment, while four of the five patients with instability reported less than 50% relief from treatment. At first appearance, patients with stability thus seem to respond better to treatment.

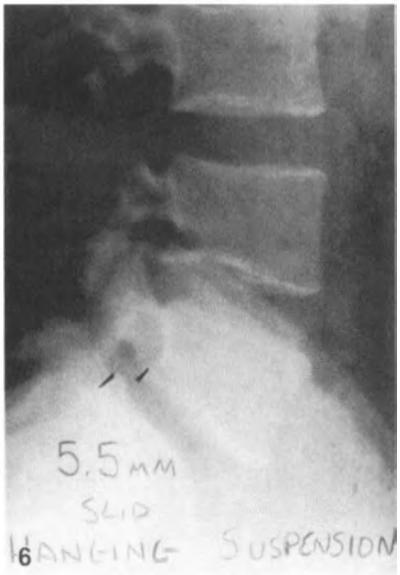
However, Table 1 demonstrates differences across patients in gender, age, and number of treatments. Did men have less pain relief than women? Does a patient's age contribute to his or her potential for pain relief? Do patients who receive many treatments have more pain relief than those with few treatments?

These questions are addressed with the results reported in Tables 2 and 3. Calculation of the bivariate Pearson correlation coefficients shows that only stability is a significant predictor of pain relief. Stability has a correlation coefficient of -0.72, which suggests that patients with instability report less marked pain relief.

Multiple regression shows that with stability entered into the equation, the R^2 is 0.72, with $F = 8.62$, which is signifi-







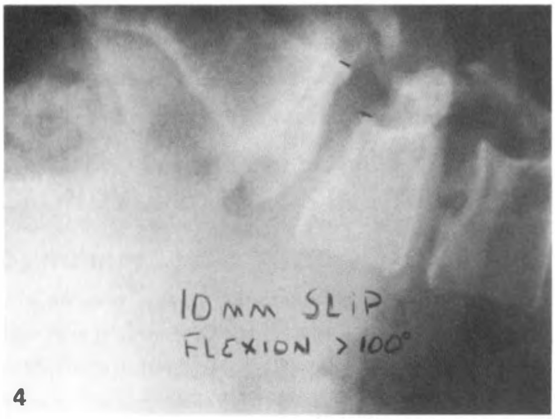


Figure 3. Neutral upright radiograph shows a 10.5-mm slippage of the L5 vertebral body on sacrum

Figure 4. Flexion study of the patient in Fig. 3 shows a 10-mm slippage

Figure 5. Extension study of the patient in Fig. 3 shows 10-mm of slippage of the L5 body on the sacrum

Figure 6. Suspension study reveals translational posterior movement of the L5 body on the sacrum by 5 mm from the neutral lateral study

cant. Examination of the Beta shows that instability patients report 0.7 unit, or almost 1 unit less pain relief than stable patients — i.e., while stable spondylolisthesis patients on average reported 75% or greater relief (scale value 1), then unstable spondylolisthesis patients on average reported 50 to 74% pain relief (scale value 2). Furthermore, when a patient's stability is known, the R^2 demonstrated that pain relief could be predicted 52% of the time.

Discussion

Flexion and extension studies can fail to demonstrate instability of the spondylolisthesis segment, whereas traction radiography, as shown in this paper based on the work of Friberg (4) is successful in showing the abnormal movement. In 117 patients with a known spondylolisthesis, lateral spot radiography showed an anteroposterior translatory movement of 5 mm or more in 24 of 45 patients with lytic spondylolisthesis of L5, in all 7 patents with degenerative spondylolisthesis of L4, and in 37 of 65 patients with a retrolisthetic displacement of L3, L4, or L5. Such instability was seen on axial traction studies in symptomatic spondylolisthesis patients even when flexion-extension studies failed to show any instability. In chronic low back pain of unknown etiology, axial traction of the spine produced abnormal posterior movement at segments that appeared quite normal on a static radiograph (4).

Advancement of spondylolisthesis slip has no value for the evaluation of severity of this condition, since the adult incidence level of slippage of 6 to 7% is reached by the age of 5–7 years. If increased slippage occurs, it is usually noted between 9–15 and seldom after age 20. One study showed that only 7 of 500 spondylolisthesis subjects followed up showed any progressive slip (5).

Treatment of 54 unstable degenerative spondylolisthesis cases with medial facetectomies and posterolateral fusion with combined distraction and compression rod instrumentation resulted in reduction of preoperative low back pain in 87% and of sciatica in 67% to 7.5% and in 5.6% postoperatively (8). Preoperative neurogenic intermittent claudication in 63% and neurogenic bladder in 11% had disappeared completely in all patients by the time of the follow-up examination.

Boston brace treatment of 67 persons with symptomatic spondylolysis and spondylolisthesis yielded an excellent or good result with no pain and return to full activities in 52 (78%)

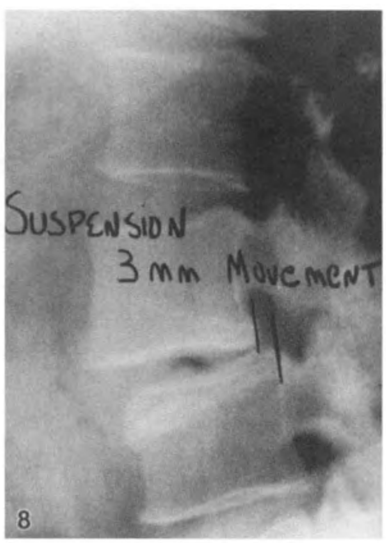
(14). Progressive adolescent spondylolisthesis during the growth spurt in 28 patients with grade I or II slippage was treated with antilordotic braces. The brace was worn for 25 months of mean duration, with the result that all patients were pain-free and none demonstrated a significant increase in percentage slip (2).

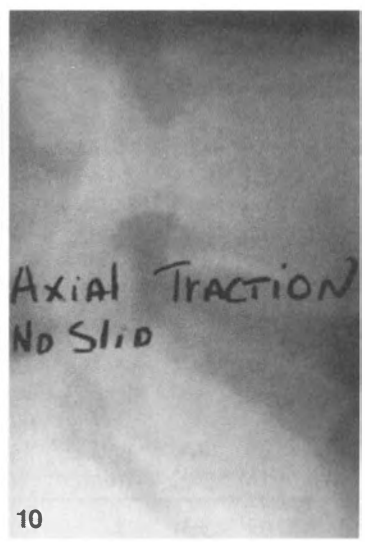
One study showed that in two thirds of nonoperative cases of grade II or less symptomatic spondylolisthesis, pain relief was obtained (12). Forty-eight patients with symptomatic back pain secondary to spondylolisthesis were treated with flexion and extension exercises with a 3-year follow-up. After 3 months of exercise, 27% of the flexion-treated patients were found to have moderate or severe pain and 32% were unable to work or had limited work duties. Among those treated with extension, at 3 months 67% had moderate to severe pain and 61% were unable to work. After 3 years, 19% of the flexion-treated patients had moderate or severe pain and 24% were unable to work, whereas 67% of the extension-treated patients were found to have moderate or severe pain and 61% to be unable to work. The overall recovery rate for flexion exercise patients was 62% and that for extension-treated patients was zero. The conclusion of this conservative treatment study was that flexion and isometric back-strengthening exercises should be used (13).

Tight hamstring muscles are a common presenting complaint or finding in symptomatic spondylolisthesis, and postural deformity or abnormal gait resulting from hamstring tightness leads to clinical evaluation (2). Eighty percent of symptomatic spondylolisthesis patients have tight hamstring muscles which tilt the pelvis backward and do not permit the hip to flex sufficiently for a normal stride. The patient then walks with a stiff-legged, short-stride gait resulting in a pelvic waddle as the pelvis rotates with each step (7).

Scoliosis occurs in 23 to 48% of patients who have symptomatic spondylolisthesis, usually due to lumbar muscle spasm and not to structural changes. The incidence of spondylolysis or spondylolisthesis is slightly higher (6.2%) in children who have idiopathic scoliosis than in the general population (7).

This study shows that of ten true spondylolisthesis patients, five showed less than 3 mm translation of the spondylolisthetic segment (stable spondylolisthesis) on vertical suspension compared with neutral upright radiography. All five of these stable spondylolisthesis patients received 75% or greater subjective relief from chiropractic adjustment.





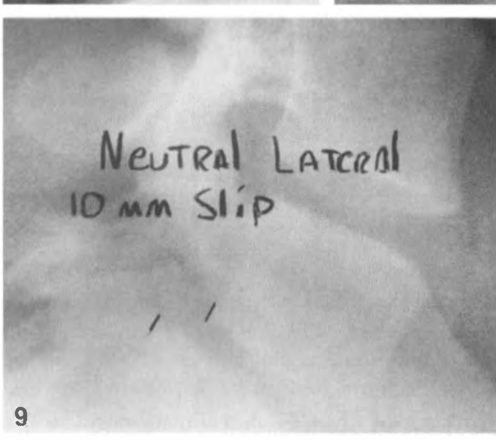


Figure 7. The L3 vertebral body is 8 mm anterolisthesed on the L4 body in neutral lateral radiograph

Figure 8. Hanging suspension study shows posterior translation of the L3 body on the L4 body by 3 mm, to give a 5-mm slip

Figure 9. 10 mm of anterior spondylolisthesis of L5 on sacrum is seen on neutral lateral projection

Figure 10. Suspension of the patient in Fig. 9 shows complete reduction of the 10-mm slippage of L5 on sacrum

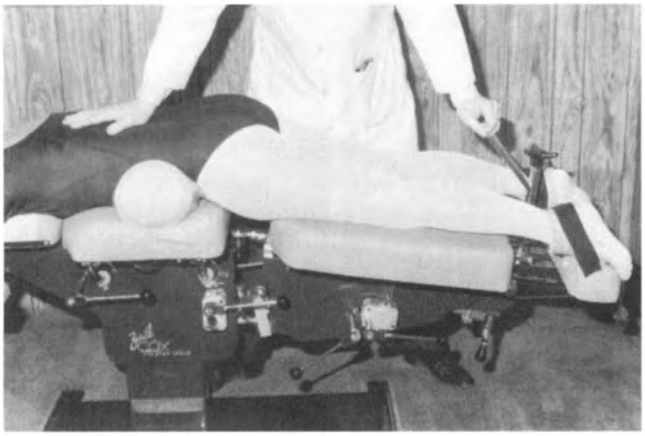


Figure 11. The distraction adjustment technic used in treatment

Figure 12. Knee-chest exercises were performed twice daily, six repetitions at a time, each held for a slow count of four

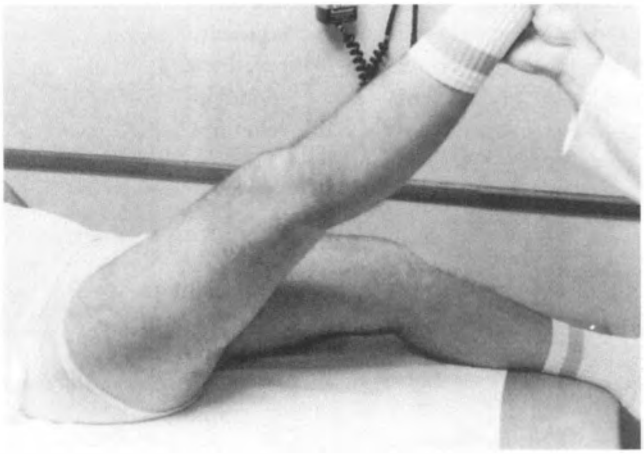


Figure 13. Hamstring stretching utilizing proprioceptive neuromuscular facilitation was done daily, three times per extremity

Table 1

Data for Each Patient

Case	Age	Sex	Ht (cm)	Wt (kg)	Level Affected	Neutral Slip[a] (mm)	Vertical Slip[a] (mm)	Movement[a] (mm)	Reduction[a] (%)	Stability[a]	No. of Treatment[a]	Response to Treatment[a]
1	31	F	165	63	L5	10	1	−9	90	1	23	4
2	24	F	111	66	L5	10	4	−6	60	1	?	3
3	61	M	205	71	L5	8	4	−4	50	1	3	2
4	15	F	160	66	L5	8	6	−2	25	2	11	1
5	54	M	190	70	L5	12	10	−2	8	2	6	1
6	42	M	?	?	L5	21	22	+1	0	2	6	1
7	25	M	185	73	L5	5	0	−5	100	1	24	1
8	37	M	165	70	L5	10	8	−2	20	2	3	1
9	33	M	152	66	L5	10.5	5.5	−5	48	1	6	3
10	59	M	210	71	L3	8	5	−3	38	2	6	1

[a]Neutral slip, spondylolisthesis slippage on neutral upright lateral view; vertical slip, slippage seen on vertical suspension study; movement, slip from neutral to vertical suspension (mm); reduction, percentage of movement from neutral to vertical suspension; stability, high instability = 1, and low instability = 2; relief, pain relief as a result of treatment; subjective response on a scale of 1 to 4, where 1 = 75% or greater relief, 2 = 50% to 74% relief, 3 = 25 to 49% relief, and 4 = less than 25% relief.

Table 2

Correlation Coefficients of Study Variables[a]

	Age	No. of Treatment	Gender	Stability
Relief	−0.23	0.35	−0.53	**−0.72**
	(0.52)	(0.36)	(0.12)	**(0.01)**

[a]Pearson correlation coefficients are reported with their probability levels in parentheses; only the significant coefficient (stability) is printed in boldface type

Table 3

Regression Analysis of Stability on Pain Relief[a]

	b	Beta	R	R^2	F	p
Stability	−1.58	−0.72	0.72	0.52	8.62	0.02
Intercept	4.33					

[a]Both the unstandardized (b) and the standardized regression coefficients (Beta's) are reported but only the standardized will be used for interpretation

The other five patients had greater than 3 mm of translational motion according to a comparison of the slippage seen in neutral standing and in vertical suspension studies. These five patients with unstable spondylolisthesis experienced less than 50% relief as a result of chiropractic adjustment in four of the five cases.

Conclusion

This study suggests that spondylolisthesis patients showing translational instability greater than 3 mm have a less favorable outcome of manipulation than those patients with 3 mm or less movement. Comparison of the vertical suspension studies and neutral upright radiography may be helpful in predicting the degree of success that can be achieved by treating spondylolisthesis with chiropractic adjustments.

There may be other valid predictors of response of spondylolisthesis cases to manipulative treatment, namely that if the vertebral body translates less than 50% of the slippage amount in going from neutral lateral standing radiograph to vertical suspension study, the response is more favorable than if the slippage amount translates by greater than 50% of the slippage amount. Such a criterion could be explored in future studies. The amount of translational movement appears to be predictive for the patient response to chiropractic adjustment.

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Friberg (41) concluded that *the degree of patient pain does not depend on the slippage in spondylolisthesis or retrolisthesis, but rather, correlates significantly with the amount of translatory movement.*

Low Back Symptoms Increase with Motion Instability

Lateral spot radiography showed an anteroposterior translational movement of 5 mm or more in 24 of 45 patients with lytic spondylolisthesis of L5, in all of 7 patients with degenerative spondylolisthesis of L4, and in 37 of 65 patients with a retrolisthesis displacement of L3, L4, or L5. In cases of spondylolisthesis or retrolisthetic instability, the upper vertebra moved posteriorly during traction and anteriorly during compression (41).

Specifically, the degree of translatory movement at the spondylolisthetic level differed significantly among groups complaining of different degrees of pain. The asymptomatic

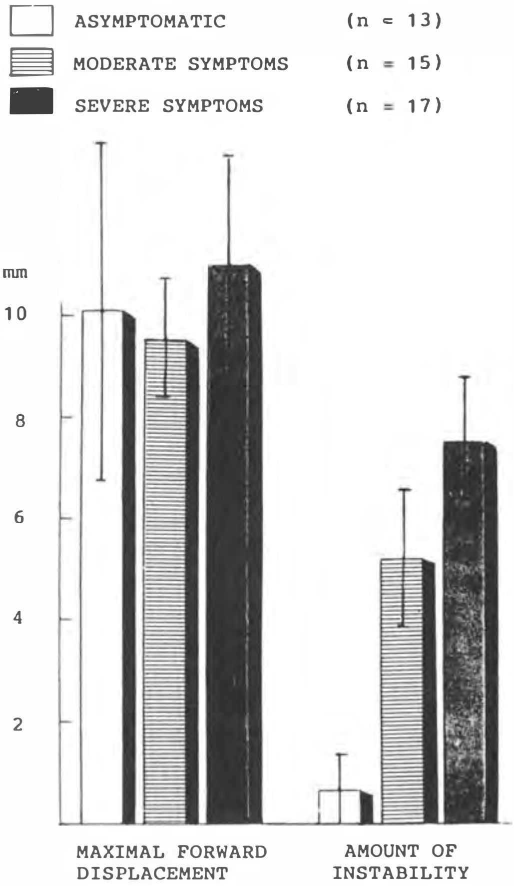

Figure 14.11. Means and standard deviations of the maximal anterior slip and of the degree of translatory instability provoked by axial traction and compression in 45 patients with lytic spondylolisthesis of L5. The patients were classified in three categories according to the severity and frequency of low back pain symptoms. (Reprinted with permission from Friberg O. Lumbar instability: a dynamic approach by traction-compression radiography. Spine 1987;12(2):123.)

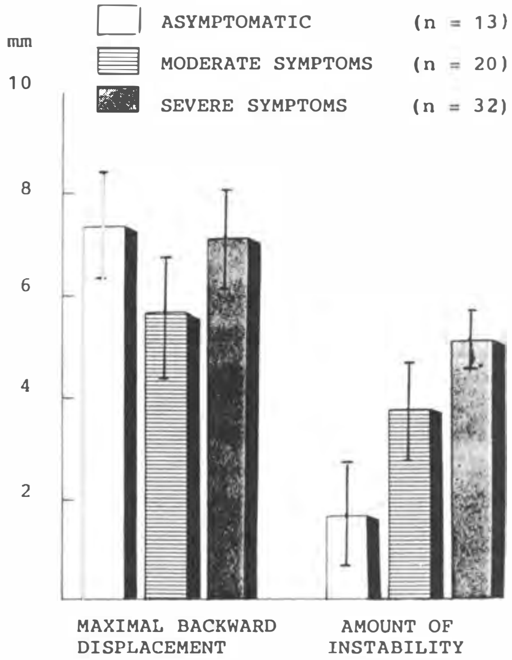

Figure 14.12. Means of the maximal posterior slip and of the translatory instability provoked by axial traction and compression of the lumbar spine in 65 patients with retro-olisthetic malalignment. The patients were classified in three categories according to the severity and frequency of low back pain symptoms as in Figure 13.4. (Reprinted with permission from Friberg O. Lumbar instability: a dynamic approach by traction-compression radiography. Spine 1987;12(2):125.)

group of patients showed a mean amount of movement of 0.7 mm, whereas the group with moderate pain symptoms showed 5.2 mm of movement, and the group with severe pain symptoms showed 7.5 mm of movement (Fig. 14.11). Therefore, the frequency and severity of low back pain symptoms correlated significantly with the amount of translational movement.

The same is true in retrolisthesis (Fig. 14.12). As in the case of lytic spondylolisthesis, a correlation was found between the amount of translatory movement and the degree of low back pain symptoms in retrolisthesis. The difference between the asymptomatic group and the symptomatic group with respect to the amount of instability was statistically significant (41).

Complete Reduction of Spondylolisthesis on Suspension

Case 1

A 25-year-old man was involved in a motorcycle accident and developed low back pain. Figure 14.13 shows a 5-mm anterior true spondylolisthetic slip of L5 on the sacrum, which reduces to 0 mm on vertical distraction, as shown in Figure 14.14. This patient was difficult to stabilize and required 6 weeks of manipulative care and the use of a stabilizing orthosis, as shown later in this chapter. This type of memory foam belt is used to stabilize our unstable spondylolisthesis patients, with some wearing the belt to bed at night until the pain is at least 50% reduced. This stability hastens healing.

Unilateral Pars Interarticularis Defect: Slippage Occurs on Extension

Case 2

A 35-year-old man had low back pain following a pushing incident. Left L4–L5-S1 pain on palpation was noted with a positive Kemp's sign and positive straight leg raising (SLR) at 45°. Range of motion was markedly limited.

Figure 14.15 is a neutral lateral upright lumbosacral radiograph showing a pars defect and anterior slippage of L5 on the sacrum. Figure 14.16 shows the pars defect to be unilateral. Flexion study (Fig. 14.17) indicates no further slippage of L5, whereas Figure 14.18, extension motion, reveals 6 mm of anterior slippage of L5 on the sacrum.

Figure 14.19 is an upright suspension radiograph showing 100% reduction of the slip.

This is an excellent study showing unilateral spondylolysis allowing translational instability. Without motion and suspension studies, this instability would not be appreciated. Treatment of this type of instability requires a lumbosacral brace for 2 months

Figure 14.13. A 25-year-old man is shown with a 5-mm anterior true spondylolisthetic slip of L5 on the sacrum. The pain followed a motorcycle accident.

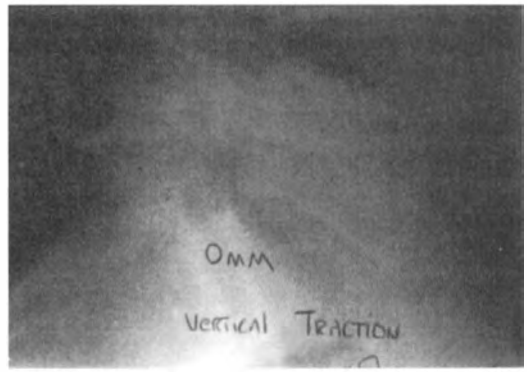

Figure 14.14. On vertical tractions, the 5-mm anterior subluxation of L5 reduces to 0 mm.

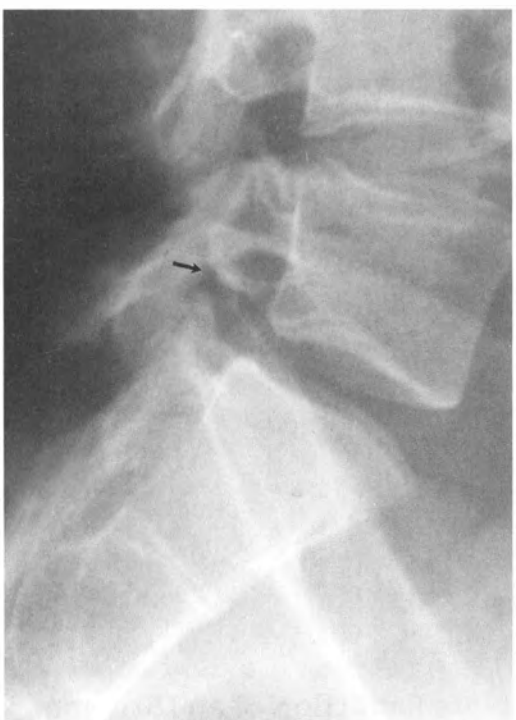

Figure 14.15. Lateral view reveals the pars interarticularis defect (*arrow*) with forward slip of L5 on sacrum. Oblique views proved this to be unilateral spondylolysis.

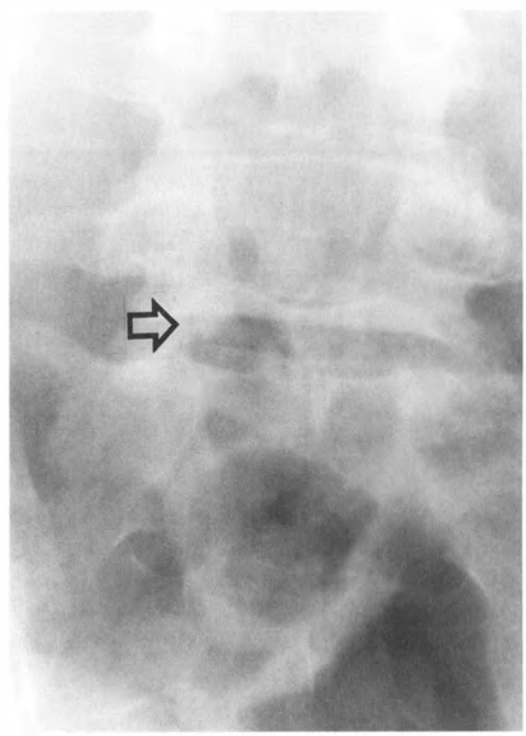

Figure 14.16. Tilt anteroposterior view at L5–S1 shows a unilateral pars defect (*arrow*), which was confirmed by oblique views.

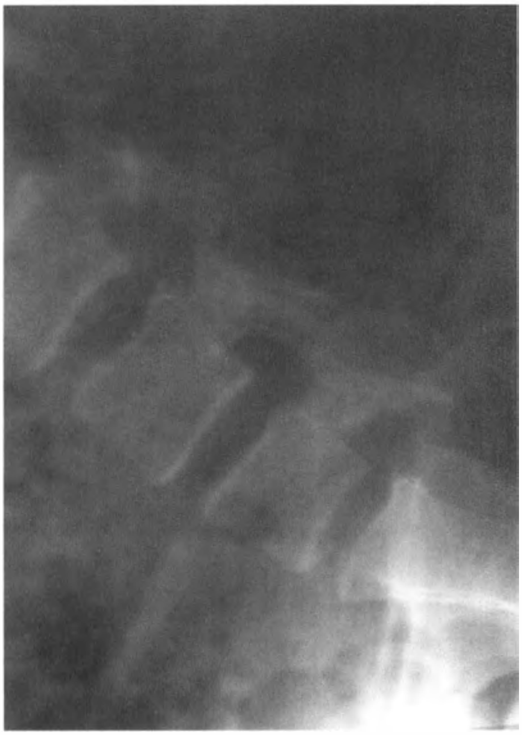

Figure 14.17. Flexion study of Figure 14.15 shows no increased slip of L5 on sacrum, with perhaps reduction of the slip.

Figure 14.19. Vertical suspension shows complete reduction of the 6-mm slip of L5 seen on extension.

Figure 14.18. Extension shows 6-mm anterior translation of L5 on the sacrum representing instability.

while stabilizing exercises of the abdomen and low back along with hamstring stretching are performed. The spinal distractive adjustment treatment is shown in Figures 11–13 of the reproduced paper entitled "Chiropractic Adjustment Results Correlated With Spondylolisthesis Instability" as well as in Chapter 9, *Biomechanics, Adjustment Procedures, Ancillary Therapies, and Clinical Outcomes of Cox Distraction Technique*. Galvanic electrical current into the involved pars interarticularis defect was given. The patient attended low back wellness school to learn ergonomics for lifting, bending, and twisting at work and home to prevent future pain. Excellent relief was obtained.

L3 Spondylolisthesis with Vacuum Instability

An L3 20% slippage with vacuum change within the nucleus pulposus (*arrow*) is shown in Figure 14.20. Note the pars interarticularis interruption and hyperostosis around the defect (*arrowhead*). L4 retrolisthesis subluxation is also seen.

Complete Reduction of an L5 10-mm Spondylolisthesis Slippage

Figure 14.21 shows a neutrolateral standing radiograph revealing a 10-mm spondylolisthesis slippage of L5 on the sacrum in a 31-year-old overweight woman. Figure 14.22 shows a vertical suspension lateral radiograph taken of this same patient that reveals total reduction of the spondylolisthetic slippage. This represents a 10-mm translational movement of the spondy-

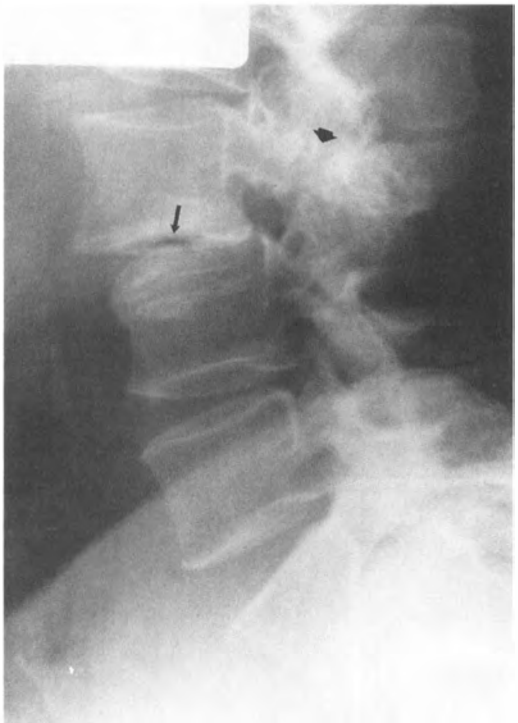

Figure 14.20. L3 is 20% anterolisthesed on L4 with vacuum change of the nucleus pulposus (*arrow*). The *arrowhead* shows the hyperostosis around the pars interarticularis defect.

Figure 14.21. In the upright neutral posture of this 31-year-old woman, the L5 is 10 mm anterior on the sacrum.

lolisthetic slip under vertical distraction. This patient's symptoms consisted of severe low back and radiating thigh pain, which interfered with her ability to sit, bend, lift, or twist at the waist. This case required considerably more days and manipulative treatments to attain relief than do less unstable cases.

Figure 14.22. On vertical traction, the 10-mm slippage shown in Figure 14.21 is totally reduced to no slippage.

MULTIPLANAR COMPUTED TOMOGRAPHY, MAGNETIC RESONANCE IMAGING, AND DISCOGRAPHIC EXAMINATION

Patients (700) with various types of spondylolisthesis were evaluated with reformatted computed tomography (CT) (42). Of these, 450 had pars interarticularis defects, 225 had degenerative spondylolisthesis, and 25 had iatrogenic subluxation. Of the 450 isthmic defects, 92% were at L5 and 7% were at L4. Unilateral clefts were demonstrated in 68 patients. The defect seen in the pars interarticularis is shown in Figure 14.23 from CT reformation.

Spondylolysis is seen to occur with two types of congenital clefts (43):

1. Retroisthmic defect (Fig. 14.24*A*), which occurs within the neural arch, behind the pars interarticularis and medial to the spinous process. It is probably of no consequence.
2. Retrosomatic cleft (Fig. 14.24*B*), which occurs anterior to the pedicle, in the fusion plane of the pedicle with the vertebral body. This defect is associated with degeneration of the disc and spondylolisthesis.

Magnetic resonance imaging (MRI) and conventional radiographic discography were used to study 101 levels in 36 patients with low back pain to detect early disc degeneration

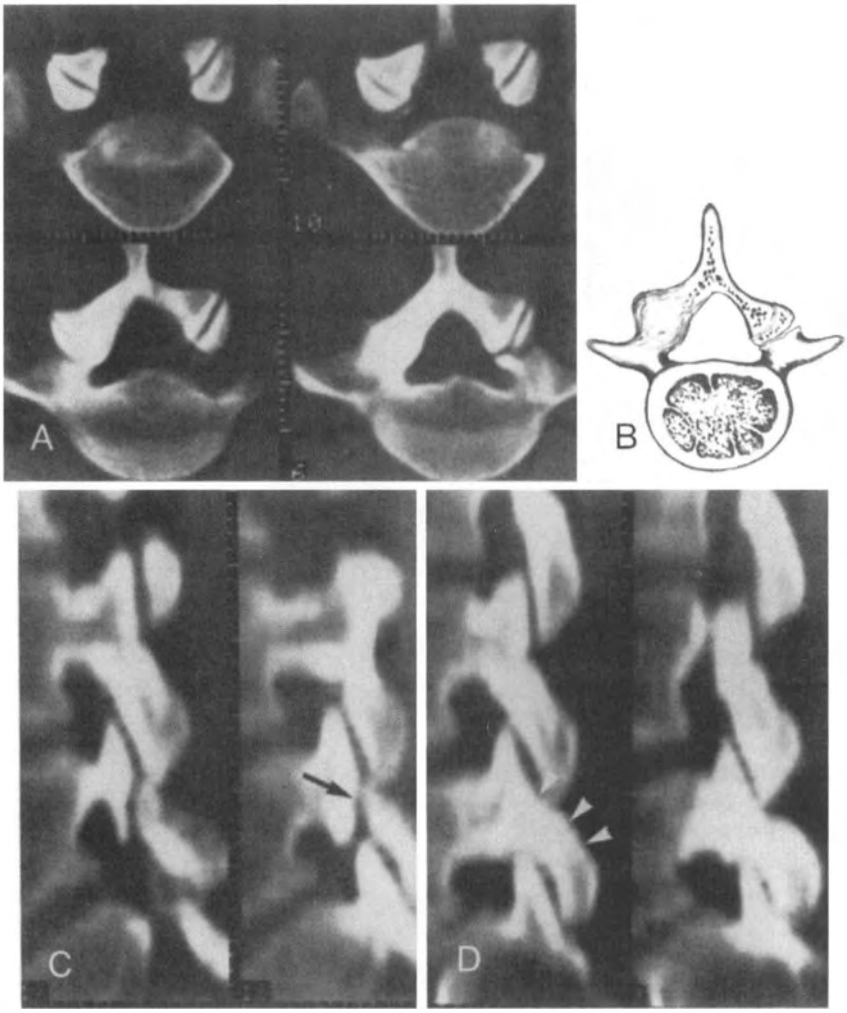

Figure 14.23. Unilateral pars interarticularis defect. **A.** Axial scan on a patient with unilateral right pars interarticularis defect. **B.** Diagram of **A. C.** Sagittal reformation through the right pars defect (*arrow*). **D.** Sagittal reformation through the thickened pars (*arrowheads*). (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville MD: Aspen, 1985:220.)

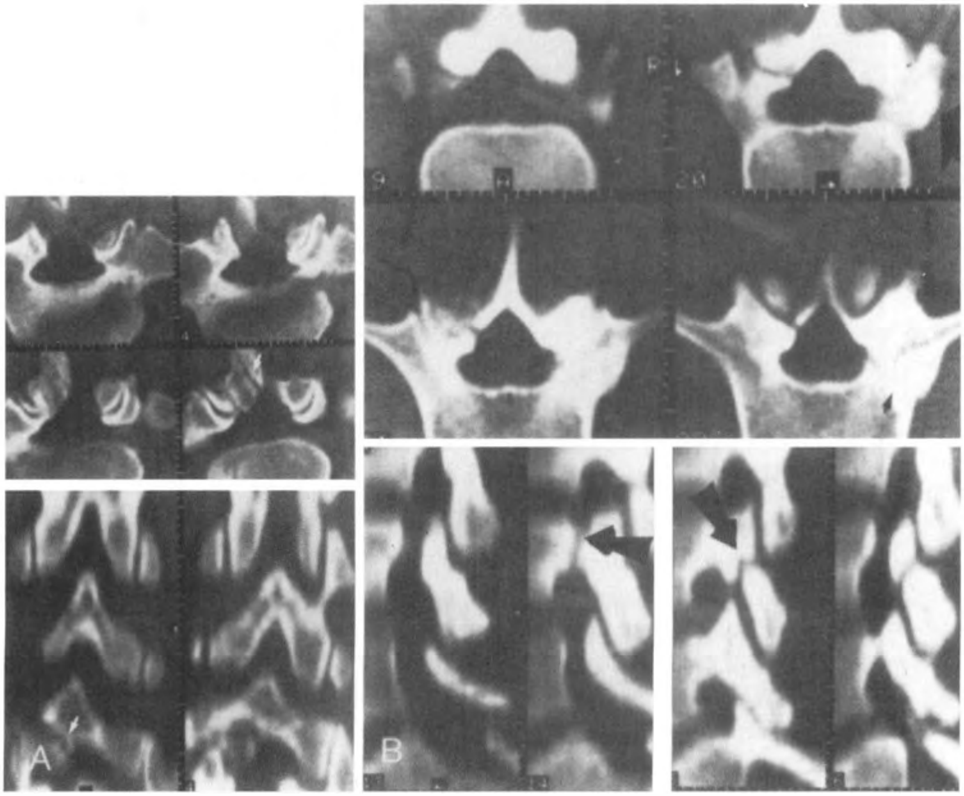

Figure 14.24. **A.** Retroisthmic cleft. Axial (*Top*) and coronal (*Bottom*) views reveal clefts within the left lamina (*arrows*). **A.** (*Top*) Axial scan with pars defect on the left (*arrow*) and a retrosomatic cleft on the right. **B.** Retrosomatic clefts. **B.** (*Bottom left*) Sagittal view through the retrosomatic cleft, which lies anterior to the pedicle (*arrow*). (*Bottom right*) Sagittal view of the opposite side through a typical pars defect (*arrow*). (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985: 230, 231.)

Table 14.1

MRI Signal and Discographic Pattern of the Affected and Adjacent Levels in Patients with Spondylolisthesis

Patient No.	Grade (1–4)	Level	MRI	Disco	Adjacent Levels	MRI	Disco
			Signal			Signal	
1	Lysis only	L5–S1	nl	nl	L3–L4	nl	nl
					L4–L5	nl	nl
2	1	L5–S1	⬇	hern	L3–L4	nl	nl
					L4–L5	nl	nl
3	2	L5–S1	⬇	deg	L2–L3	⬇	deg
					L3–L4		deg
					L4–L5	↓	deg
4	1	L5–S1	⬇	deg-hern	L3–L4	nl	nl
					L4–L5		deg-hern
5	1	L3–L4	⬇	deg	L4–L5	⬇	deg
					L5–S1	nl	nl

Reproduced with permission from Schneiderman G, Flannigan B, Kingston S, et al. Magnetic resonance imaging in the diagnosis of disc degeneration: correlation with discograpy. Spine 1987;12(3):280.
nl, normal; ⬇, marked loss or no signal; ↓, intermediate signal loss; deg, degenerated; hern, herniated; deg hern, degenerated herniated.

(44). Table 14.1 shows the MRI signal and discographic patterns found at the level of spondylolisthesis and adjacent levels.

Figure 14.25 shows the degenerative change of the L3–L4 disc, which is the level of degenerative spondylolisthesis of L3 on L4. Note how the nuclear material has dissipated throughout the entire anulus fibrosus of the disc and the signal intensity of the disc is decreased. Figure 14.26 reveals the marked degenerative internal derangement of the nuclear material at L5–S1, with protrusion of the nuclear material posteriorly.

Plain Radiographs Necessary

A plain radiograph should be used for primary examination to detect displacement, lysis, and degenerative changes. MRI has additional advantages in the evaluation of the intervertebral neural foramina in spondylolisthesis. MRI is recommended as the second imaging modality after plan radiographs if surgery is contemplated (45).

Recumbent Radiographic Study Superior to Upright Study

Upright, recumbent flexion and extension studies of 50 consecutive adult patients with spondylolisthesis showed 31 to display abnormal translation. Of these, *18 had abnormal motion only when they were examined in the lateral decubitus position and not when standing.* Nine displayed excessive motion in both positions. Only four displayed more translation while standing.

When spondylolisthesis is being analyzed to maximize motion, flexion and extension radiographs should be obtained in the lateral decubitus position. Instability denotes surgical need in symptomatic spondylolisthesis (46).

MRI Diagnosis for Pars Interarticularis Defects

A hypointense area in the pars interarticularis on T1-weighted images before the appearance of spondylolysis on plain radiography or CT has been documented. This hypointense area may be caused by hemorrhage in the pars interarticularis or edema in adjacent tissues. Changes in MRI signal intensity in the pars interarticularis are useful in the early diagnosis of spondylolysis (47).

MRI Changes of the Lumbar Pedicles

Type I changes are characterized by decreased signal on T1-weighted images and increased signal on T2-weighted images. Type II changes are characterized by increased signal on T1-weighted images and isointense or slightly hyperintense signal on T2-weighted images. Type III changes are characterized by low signal intensity on both T1- and T2-weighted images.

Progressive reactive marrow changes are seen in 40% of teenage and adult patients with spondylolysis (48).

Single Photon Emission Computed Tomography

Fifty patients with spondylolysis and back pain were evaluated by single-photon emission computed tomography (SPECT bone scanning). In acute spondylolysis, the SPECT scan tends to revert toward normal even though healing of the spondylolysis has not occurred. As spondylolisthesis develops and progresses, the SPECT scan again becomes positive. SPECT scanning in spondylolysis is not a positive or negative process, but

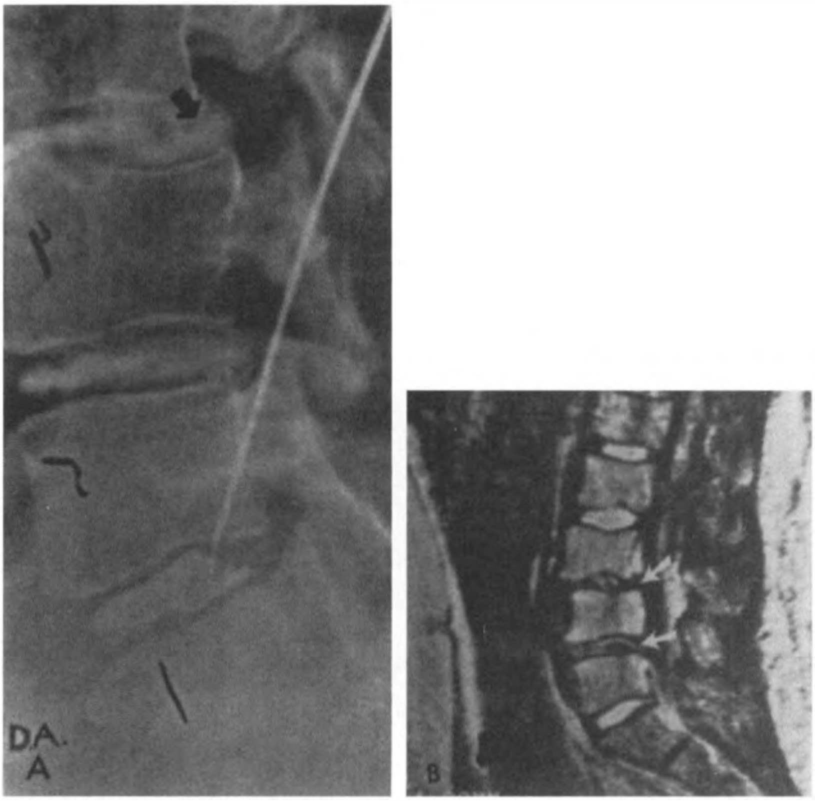

Figure 14.25. **A.** Lateral discogram examination demonstrates degenerated herniated discs at the level of L3–L4 and L4–L5. A normal disc is identified at L5–S1. A grade 1 spondylolisthesis L3 on L4 is seen (*arrow*). **B.** Sagittal MRI (SE 2000/56) demonstrates grade 1 spondylolisthesis L3 on L4 and marked loss of signal intensity at the levels of L3–L4 and L4–L5 (*arrows*). Note normal intensity at L2–L3 disc and L5–S1 disc. (Reprinted with permission from Schneiderman G, Flannigan B, Kingston S, et al. Magnetic resonance imaging in the diagnosis of disc degeneration: correlation with discography. 1987;Spine 12(3):280.)

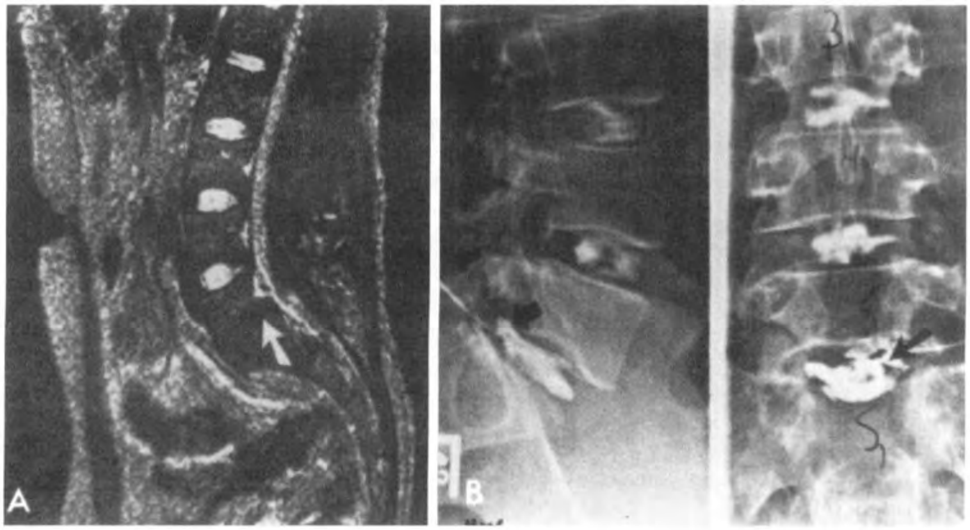

Figure 14.26. **A.** Sagittal MRI (SE 2000/70) demonstrates grade 1 spondylolisthesis and disc degeneration at the L5–S1 level (*arrow*). Normal disc intensities are noted at the L4–L5 level. **B.** Anteroposterior and lateral discography demonstrates normal disc levels at L3–L4, L4–L5, and a degenerated herniated disc at L5–S1 (*arrows*). (Reprinted with permission from Schneiderman G, Flannigan B, Kingston S, Thomas J, et al. Magnetic resonance imaging in the diagnosis of disc degeneration: correlation with discography. Spine 1987;12(3):281.)

rather varies with the time and stability of the spondylolytic spine (49).

When a given patient develops spondylolysis, it is likely the patient will have a positive SPECT scan showing activity in the area of the pars. If the spondylolisthesis does not occur, then the SPECT scan tends to gradually become negative.

As slippage occurs, the pars interarticularis is disrupted and a microfracture of it occurs. The result is remodeling and development of degenerative changes about the disc space. The SPECT scan reflects these changes by showing increased activity in the involved area. The SPECT scan of the lumbar spine should not be viewed as either a positive or negative screening test, but rather as a means of evaluating mechanical stresses that are occurring at any given level and time at the site of the spondylolysis (49).

Surgical Success Depends on Positive SPECT Scan

Surgery is found to relieve pain if a positive SPECT scan is present and the patient obtains relief by immobilization with a lumbar brace prior to surgery. Negative SPECT scan patients have had pain after surgery (50).

DISC HERNIATION WITH SPONDYLOLISTHESIS IS UNUSUAL

Painful lumbar disc herniation in spondylolytic spondylolisthesis is rare. Most people with spondylolisthesis are asymptomatic. When low back symptoms are present, these are thought to originate from segmental degeneration, instability, or facet joint osteoarthrosis. Radicular symptoms are considered to be caused by nerve root compression or impingement at the pars interarticularis defect (45).

TREATMENT OF SYMPTOMATIC SPONDYLOLISTHESIS

Surgical Stabilization

Although most people with spondylolysis are asymptomatic, and those with back and/or leg pain usually respond to conservative treatment, a small percentage of patients with intractable back and/or leg pain may require operative treatment. Anterior or posterior fusion gives good results in about 75% of cases (51). An instrumented posterolateral arthrodesis in combination with a Gill procedure and a L5 nerve root decompression results in a high rate of fusion, satisfactory clinical success, and a high rate of return to work (52).

Fusion Results

Longstanding intractable lumbar and/or radiating pain with spondylolysis-olisthesis ($n = 31$), degenerative disc disease and/or facet joint arthrosis ($n = 23$), and pain after laminectomy/decompression ($n = 9$) showed 28 of 49 preoperatively employed patients returned to work. No correlation was found between relief of pain and return to work. The clinical results were best in the spondylolysis-olisthesis group. Posterolateral lumbosacral fusion with transpedicular fixation provides a satisfactory clinical outcome in patients with spondylolysis-olisthesis, but the high incidence of complications related to the fixation device in the other indications studied was a serious drawback of the method (53).

Unremitting symptoms after 6 months of nonoperative care or progression of slippage and neurologic signs indicate surgical care (47).

L4 Spondylolisthesis

Spondylolytic lesions at the L4–L5 level are more unstable than those at L5–S1 level, and the iliolumbar ligament is not implicated as the cause of this difference, although its presence did effect the range of motion seen. Surgical stabilization should be considered an option with an L4–L5 lesion sooner in the course of treatment, whereas the L5 lesion has an anatomic advantage that allows more conservative treatments to be successful (54).

DIAGNOSIS AND TREATMENT OF CHILDREN WITH SPONDYLOLISTHESIS

Twenty-three percent of patients experiencing back pain secondary to spondylolisthesis have an onset of pain before the age of 20, with age 2 years being the youngest child specifically recorded to present with back pain as the chief complaint that led to the diagnosis of spondylolisthesis (55).

Spondylolisthesis is the most important cause of back pain in children and adolescents, and fusion is recommended even in asymptomatic patients if the slip exceeds 40%. In many cases the slip seems to progress in a short time, leading to low back pain, hamstring tightness, or radiating pain (56). Surgical stabilization of the slipped segment should be limited to the slip seen on the preoperative hyperextension x-ray film (57).

Source of Pain in Children with Spondylolisthesis

Pseudoarthrosis is the source of pain in children with spondylolisthesis, with secondary changes occurring in the adjacent discs, resulting in discogenic back pain. In an attempt to prevent disc degeneration at adjacent levels, the spondylolisthesis segment should be stabilized with the Scott wiring technique (58).

Clinical Findings in Children with Acute Spondylolysis Causing Low Back and Leg Pain

Three children with low back pain radiating to the leg and with spasm of the hamstring and paravertebral muscles were reported (59). All three had x-ray findings of unilateral or bilateral spondylolysis, and localized positive bone scan pointing to spondylolysis as the cause of the pain. The ages of the three children were 10, 7.5, and 14 years.

The authors felt that the symptoms of these three cases were caused by referred pain from noxious stimuli affecting a branch of the posterior primary ramus in the facet joints, and the diagnosis of the cases was facet syndrome. The facet joint involvement was explained by a communication between the defective area of the pars interarticularis and the facets above and below it, as demonstrated by Ghelman and Doherty (60), and Maldague et al. (21). Irritation of these communicating joints and of the richly innervated periarticular tissues may account for the low back radiating pain of patients with spondylolysis.

Clinical Correlation with Severity of Spondylolisthesis Slippage

Saraste (38) did a 20-year follow-up study of 255 spondylolysis or spondylolisthesis patients to correlate the clinical and radiographic findings for their condition. Mean value of progressive slippage of all cases was 4 mm; mean slippage in adolescents was 2.5 mm and in adults 5 mm. L4 showed a greater mean value of slippage, 7 mm, compared with 4 mm at L5.

Flexion-extension radiographs in the standing position as compared with recumbent films showed negligible positional changes. I would contrast the use of flexion and extension failure to demonstrate motion at the spondylolisthetic segment to the Friberg work on vertical distraction translatory motion, showing marked motion. I further feel that the sacral motion under the spondylolisthetic segment may be a far greater cause of the apparent motion on vertical traction than the movement of L5 on the sacrum or L4 on L5.

Saraste (38) found 20% of the spondylolytic patients had severe disc degeneration at the L4 and L5 spondylolysis levels when originally seen, but at follow-up, 50% of the L5 and 70% of the L4 spondylolysis groups had progressed to severe disc degeneration. Interestingly, over half the cases showing more than 25% slippage at the time of diagnosis showed severe disc degeneration.

Pain is the most common symptom of spondylolysis and spondylolisthesis, with the peak onset of symptoms at the adolescent growth spurt (61–67). Most adolescents with spondylolysis are symptomless, although spondylolisthesis is the most common cause of low back pain and sciatica in children and adolescents (64).

In a study of 500 first grade students, Fredrickson et al. and Wiltse and Jackson (68, 69) described the natural progression of spondylolytic patients as follows: at age 6, the incidence of spondylolysis and spondylolisthesis was 4.4 and 2.6%, respectively; the incidence at adulthood was 5.4 and 4.0%. None of these children were found to be symptomatic, whereas Wiltse and Jackson (69) found few symptomatic children between ages 10 and 15 years with spondylolisthesis, although they felt that most slippage occurred between these ages. Wiltse also found a 5% incidence of spondylolysis in children aged 5 to 7 years, with an increase to 5.8% by age 18. Most of the slippage occurred between ages 11 and 15, the time of growth spurt and vigorous exercise (65, 68, 70).

Healing with Bracing and Electrical Stimulation

Healing of an acute spondylolysis with intermittent bracing and daily external electrical stimulation is reported using a molded plastic thoracolumbar sacral orthosis (TLSO) while out of bed for 6 months and during athletic activities for an additional 5 months. Three months after starting the external electrical stimulation, a CT scan demonstrated progressive osseous healing and the patient had minimal symptoms (71).

Pars Interarticularis Fractures Heal

Canadian Eskimos show adolescent and young adult spondylolysis stress fractures to heal by middle adulthood; even after 45 years of age, the overall frequency of spondylolysis declined, indicating that even complete defects occasionally healed (72).

PSEUDOSPONDYLOLISTHESIS (NO PARS FRACTURE PRESENT)

Pseudospondylolisthesis is caused by degeneration, sagittal facets, or elongated pars interarticularis.

DEGENERATIVE SPONDYLOLISTHESIS

Lumbar Degenerative Spondylolisthesis

Degenerative spondylolisthesis (DS) is the slipping of one vertebral segment on the one below in the presence of an intact neural arch. It occurs secondary to facet joint arthritis and disc degeneration (5, 73–76). DS usually affects people older than 50, being more common in blacks; women are more often affected than men. The L4–L5 level is most often involved (5, 74, 76–78), with L3 next in order of frequency (78). Approximately 10 to 15% of patients with DS require surgery for relief of pain (15, 76–78). The severe pain is radicular, not relieved by conservative therapy, and usually associated with cauda equina symptoms secondary to stenosis of the canal by the hypertrophic subluxating facet joints (5, 75–78). Twenty-five percent of spondylolisthesis is caused by degenerative spondylolisthesis (5).

The cause (5, 16, 75, 76, 79), pathology (16, 77, 78, 80–82), symptoms (75–78), and diagnosis (5, 16, 76–78) of DS have been discussed in the literature.

The degenerative lesion is caused by longstanding intersegmental instability (75, 83, 84). Farfan believes that multiple small compressive fractures occur in the inferior articular processes of the vertebra that slips forward (16).

In the patients who come to the doctor with clinical symptoms, degenerative spondylolisthesis occurs six times as frequently in females than males, six to nine times more frequently at the L4 interspace than the adjoining levels, and four times more frequently when L5 is sacralized (78). When the lesion is at L4, the L5 vertebra is more stable and in less lordosis than average. Finneson states that he has never seen DS in a patient under age 40 (15).

Slipping in DS never seems to exceed 33% unless surgical intervention has occurred. The predisposing factor is thought to be a straight, stable lumbosacral joint that sits high between the ilia. This arrangement puts increased stress on the joints between L4 and L5, leading to decompensation of the ligaments, hypermobility and degeneration at the articular processes, and multiple microfractures of the inferior articular processes of L4, allowing forward slipping (78).

Finneson (15) states that the appropriate treatment is symptomatic therapy, with surgery used only for patients with severe pain. Most patients have little or no neurologic deficit, but a few have severe changes. The myelogram is characteristically dramatically abnormal. Circulatory change in the legs is not part of the syndrome. It is the L5 nerve root that is compressed in an L4–L5 olisthesis. The nerve is compressed between the inferior articular process of L4 and the upper margin of the body of L5.

Many structures can be stressed and irritated in DS. In discussing pain mechanisms in DS, it is well to remember the innervation of the facet joints (85–91), intervertebral disc (16, 87, 90–103), posterior longitudinal ligament (16, 87, 93, 96, 99, 101, 104), anterior longitudinal ligament (87, 91, 93, 96, 99, 104), dura mater (91, 93, 96, 104, 105), and vertebral periosteum and bone (87, 91, 96, 104). It is not well understood just how the pain in spinal stenosis caused by DS is produced. Probably the best explanation is that the nerves are denied adequate nourishment because of pressure on the tiny blood vessels that supply them (106, 107). The perineurium of the spinal nerves themselves is richly supplied with tiny nerve fibers. Perhaps ischemia of these causes the pain (108).

Naked endings of the sinuvertebral nerve have been identified in the granulation tissue ingrowth of reparative healing in the anulus fibrosus (102). Pain receptors may be there, which would explain discogenic pain in the absence of herniation. Certainly tearing of disc fibers occurs in DS. Souter and Taylor (109) state that branches of the sinuvertebral nerve supply the outer layer of the anulus fibrosus, most of the terminations being naked nerve endings, probably mediating pain sensation. They also found fine nerve fibers in the granulation tissue in the deeper layers of the anulus fibrosus of a degenerative disc.

Bogduk (93) states that lumbar IVDs are innervated posteriorly by the sinuvertebral nerve, and both lateral and anterior aspects of the anulus fibrosus and the anterior longitudinal ligament are innervated by a series of nerves derived from the ventral rami and the sympathetic nervous system. The posterior lateral aspect of each IVD receives branches from the ventral ramus at each level and/or the terminal portion of the gray ramus communicans. The lateral aspects of the IVD receive ascending or descending branches from the gray rami communicantes, which reach the anuli fibrosi by passing between and then deep to the attachments of the psoas major. Three recent studies (93, 102, 103) corroborated earlier reports of nerve fibers as far as a third of the way into cadaveric anuli fibrosi, and nerve endings as deeply as halfway into anuli fibrosi obtained during posterior and anterior fusion operations.

Degenerative spondylolisthesis causes stenosis at the vertebral canal because of compensatory hypertrophy and sclerosis of the superior facets, which consequently encroach on the lateral recesses, causing an hourglass deformity seen on myelography (105, 110). Anterior slippage of this superior vertebra compresses the dural sac between its anteriorly migrated inferior facets and the superior border of the lower vertebra (Figs. 14.27 and 14.28). The slippage has a natural tendency to increase (111), but the severity of symptoms cannot always be correlated with the severity of the slip because a severe slip may occur without marked degenerative change, and vice versa. Backache of several years duration, most commonly increased

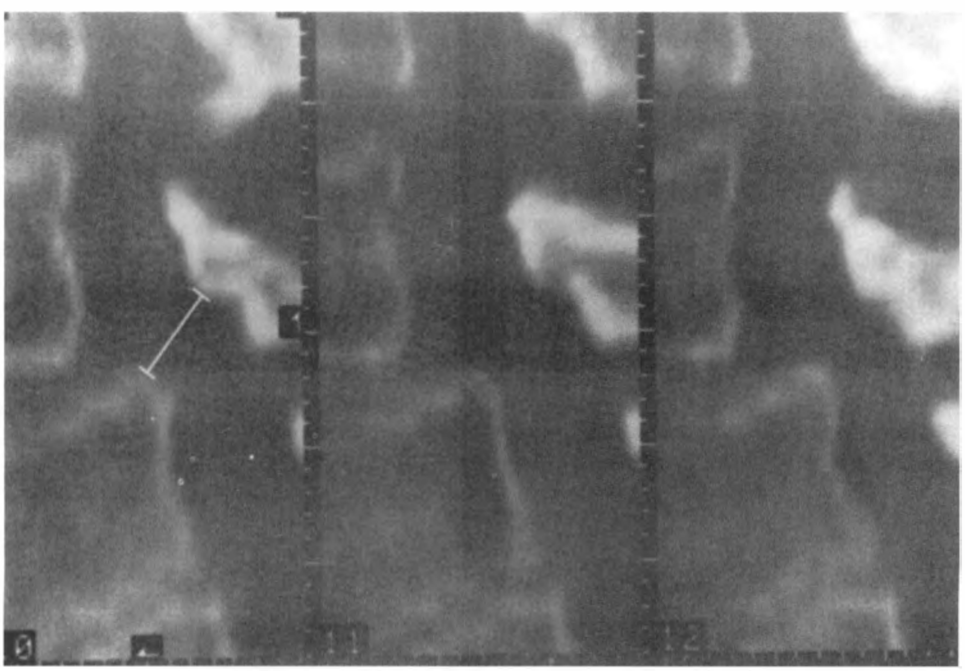

Figure 14.27. Degenerative spondylolisthesis, L4–L5. Sagittal reformations reveal 8 mm forward subluxation of L4 on L5. The diameter of the spinal canal is reduced to 8 mm. This is measured from the posterior lip of the superior end plate of L5 to the undersurface of the L4 lamina. (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:235.)

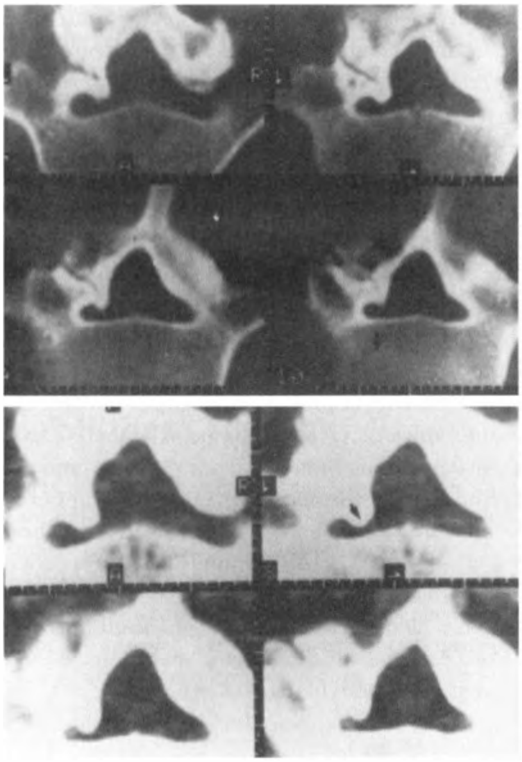

Figure 14.28. Lateral recess stenosis. **Top four panels,** bone window axial scans demonstrate lateral recess stenosis at the level of a pars interarticularis defect (*arrow*). **Bottom four panels,** soft-tissue axial view on the same patient. (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:234.)

by exercise or by getting up from bed rest, is common. Sciatic pain usually follows months or years or back pain. Weakness and numbness of the legs as well as absent ankle reflexes may be seen in DS.

Nerve entrapment, the most important feature of DS, can occur in any of four ways (104): (*a*) pressure on the L4 nerve at the foramen by osteophytes arising from the posteroinferior surface of the vertebral body of L4; (*b*) pressure on the L5 nerve from posterior displacement of L5 on L4, forming a bony ridge in the region of the lateral recess; (*c*) pressure on the L5 nerve root in a narrow lateral recess at the lower border of the L5 vertebra; or (*d*) pressure on the L5 nerve by the anteriorly inferior articular process of L4.

Treatment of DS should be conservative as long as the pain is tolerable (108, 112), as only rarely do patients with lumbar spine stenosis have neurologic changes that in themselves warrant surgery.

FACET ROLE IN DEGENERATIVE SPONDYLOLISTHESIS

Facet orientation plays a significant role in the advancing slippage in DS. Sagittally oriented facets offer less bony resistance to the forward and downward force of the L5 vertebral body than do oblique or coronally faced facet joints (Fig. 14.29). Facet tropism is extremely common in these patients, and it is likely an important predisposing factor leading to dislocation (75).

Facet joint arthrosis (severe erosion and degeneration) is a hallmark of spondylolisthesis with intact neural arches (DS) (43). These changes are seen in Figure 14.30. The joint space seems unusually widened because of severe erosion of the articular surfaces.

SUBLUXATION AT THE LEVEL OF SPONDYLOLISTHESIS

The most severe clinical symptoms of spondylolisthesis can occur when unrestricted anterior dislocation of the inferior facet of the upper vertebral body occurs beyond the confines of the anterior limb of the superior facet. This can occur bilaterally, causing forward dislocation, or unilaterally, causing rotatory or lateral subluxation (Fig. 14.31).

REVERSE SPONDYLOLISTHESIS (RETROLISTHESIS)

Reverse spondylolisthesis (retrolisthesis) is an instability occurring at usually the L3–L4 and L4–L5 levels because of disc degeneration (disc narrowing, spur formation, sclerosis, end plate erosion, and facet joint laxity). Foraminal stenosis is an important feature because of upward displacement of the superior facet of the lower vertebra into the neural foramen. Figure 14.32 reveals retrolisthesis above a spondylolisthesis subluxation.

CASE PRESENTATIONS OF DEGENERATIVE SPONDYLOLISTHESIS FROM THE AUTHOR'S CLINIC

Advancing Degenerative Spondylolisthesis

Case 3

Figures 14.33 through 14.37 are studies of a 52-year-old woman who developed low back pain and ulcerative colitis in 1982. She required a colon resection in 1983. The progressive nature of her degenerative spondylolisthesis is unusually revealed by progressive x-ray studies. Figure 14.33 is a neutral lateral radiograph taken in 1982, which does not show disc degeneration or spondylolisthesis at L4. Figure 14.34, taken in 1984, does show disc degeneration of the L4–L5 disc, with anterior subluxation of L4 on L5 by about 8 mm. Figure 14.35, made in 1987, shows advanced degenerative changes of the L4–L5 disc, with total loss of disc space and permanent stabilization of L4 on L5. Figures 14.36 and 14.37 are the axial and sagittal reformation showing the extensive L4–L5 discal degeneration and the L4 pseudospondylolisthesis. Note the rotatory subluxation of the inferior facets with narrowing of the lateral recesses and sagittal diameter of the vertebral canal.

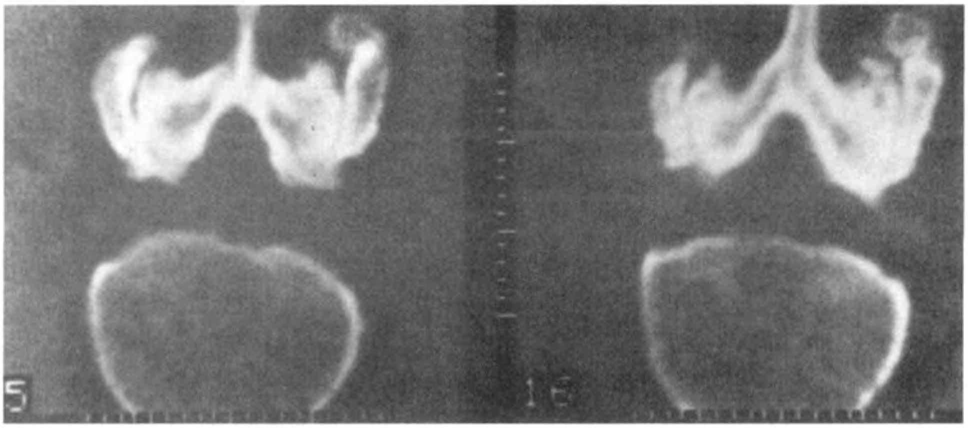

Figure 14.29. Degenerative spondylolisthesis, L4–L5. Axial views demonstrate sagittally oriented facets that have dislocated. Cartilaginous surfaces are irregular and eroded. (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:235.)

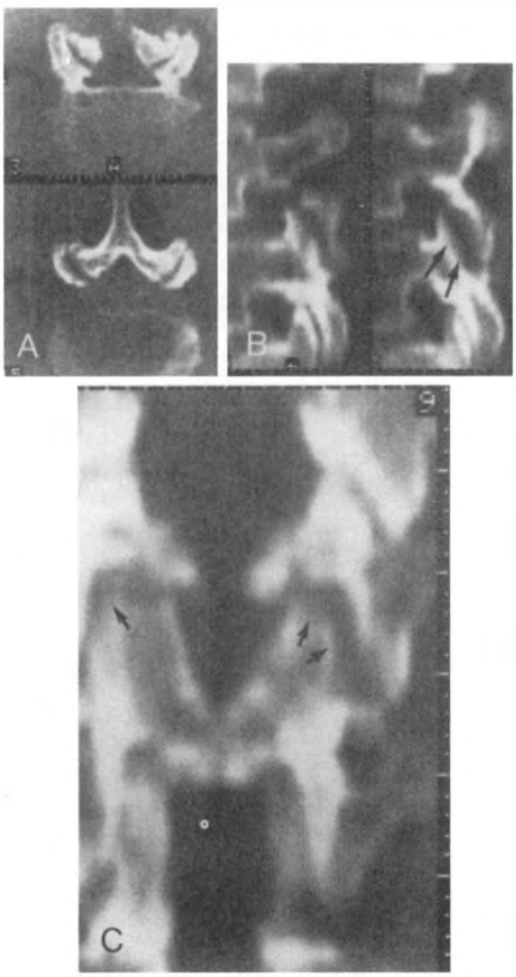

Figure 14.30. Arthropathy in degenerative spondylolisthesis. **A.** Axial scan demonstrates severe erosive arthritis of the facet joints, especially on the left. Cartilage erosion is present, and the joint space is widened. **B, C.** Sagittal and coronal views similarly show a widening of the joint, with destruction of the articular surfaces (*arrows*). (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:241.)

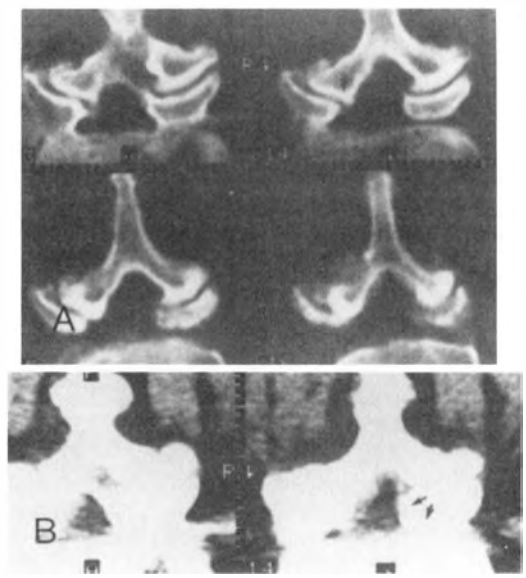

Figure 14.31. Lateral subluxation. **A.** Axial scans windowed for bone. **B.** Soft tissue scan reveals coronally oriented facets. Considerable lateral subluxation is seen of the facets, causing prominent compression of the left lateral recess. The space available for the theca and cauda equina is remarkably reduced. (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:247.)

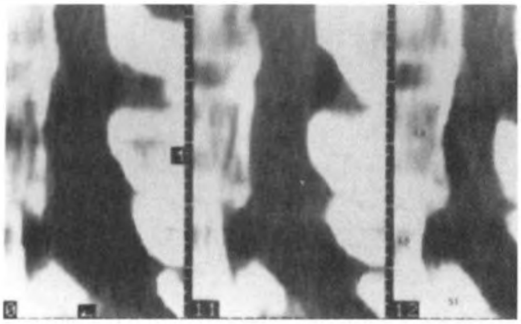

Figure 14.32. Retrolisthesis above spondylolisthesis. A series of sagittal reformations reveals a 9-mm forward spondylolisthesis of L5 on the sacrum and 9-mm retrolisthesis of L4 on L5. Note only minimal compression of the spinal canal is evident in this patient. (Reprinted with permission of Steven Rothman, MD. Rothman SLG, Glenn WV. Multiplanar CT of the Spine. Rockville, MD: Aspen, 1985:251.)

Figure 14.33. A neutral lateral lumbar spine radiograph taken in 1982 shows normal bone, disc, and soft tissue at all levels.

Figure 14.34. Repeat lateral radiograph taken in 1984 reveals degenerative disc disease at L4–L5, with degenerative spondylolisthesis of L4 on L5.

Figure 14.35. Repeat lateral radiograph of the spine seen in Figures 14.33 and 14.34 shows advanced degenerative disc disease at L5–S1, with total loss of the disc space and extreme hyperostosis of the opposing vertebral body plates of L4 and L5.

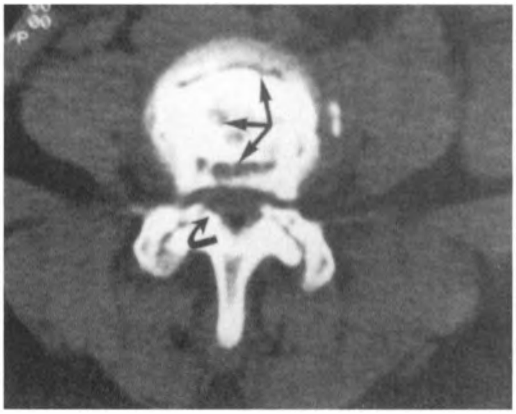

Figure 14.36. Axial computed tomography scan at the L4–L5 disc level reveals osteochondrosis vacuum phenomenon of the disc (*straight arrows*) as well as facet degeneration. The lateral recesses are narrowed, with rotosubluxation of the vertebral arch and the anterior rotation of the right inferior facet (*curved arrow*).

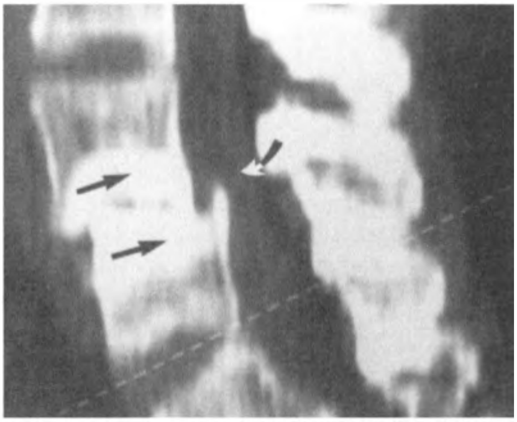

Figure 14.37. Sagittal reformatting shows the degenerative spondylolisthesis of L4 on L5 with the marked bone hyperostosis of the opposing bone plates (*straight arrows*). Note the stenosis at the vertebral canal between the posterior superior L4 vertebral body arch (*curved arrow*).

Figure 14.38. Lateral projection shows anterior displacement of L4 on L5, with traction spurring of the anterior lateral body plates of L3, L4, and L5.

Stable Pseudospondylolisthesis of L4 on L5

Case 4

A 60-year-old man was seen complaining of low back and bilateral leg pain, which was worse following walking. No pain was experienced on sleeping or sitting, except that when he stood after sitting he again felt the discomfort in the legs. Doppler examination of the lower extremities revealed no evidence of vascular claudication.

Figure 14.38 is the lateral radiograph of this patient, which reveals an anterolisthesis of L4 on L5. This is a degenerative spondylolisthesis of L4 on L5. Note the marked loss of the L5–S1 disc space with nuclear invagination of the L5 disc into the inferior plate of L5. Marked anterolateral hypertrophic changes are seen at the L3–L4, L4–L5, and L5–S1 levels. Seen is a left lean of the lumbar spine with a levorotation subluxation of the L3–L5 segments.

Figure 14.39, a lateral projection with Eisentein's measurements made, does show that this patient has stenosis at the L5 level. Remember that any time the sagittal diameter of the vertebral canal is less than 12 mm, stenosis is present, and 12 to 15 mm is an impending stenosis. Also note the 4:1 ratio of the 41-mm vertebral body sagittal diameter to the 10-mm vertebral canal sagittal diameter.

Figures 14.40 and 14.41 are the flexion and extension studies in this case. Note that flexion (Fig. 14.40) shows a 2-mm anterior translation of the L4 vertebral body on L5, whereas extension reveals a 0.5-mm posterior translation of L4 on L5 compared with the neutrolateral view. We feel that 3 mm of movement is within stability at a disc level, so that this L4–L5 disc was not markedly unstable at the time.

Treatment of this case consisted of distraction manipulation with a small Dutchman pillow under the L4 vertebral body while contact was made on the L3 spinous process to allow flexion distraction to be applied. This patient was placed on knee–chest exercises, a strong course of hamstring stretching, abdominal strengthening exercises, and gluteus maximus strengthening exercises as well. Treatment resulted in a slow, yet progressive, relief of the patient's symptoms until, after approximately 6 weeks of care, he was approximately 75% relieved of his problem.

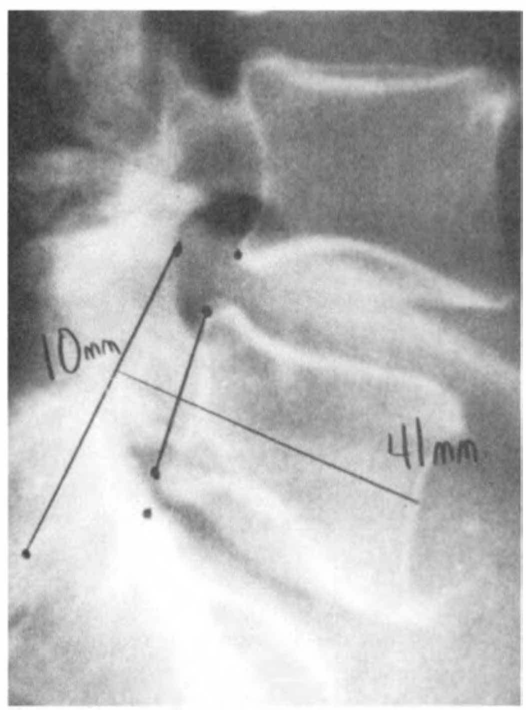

Figure 14.39. Eisenstein's measurement for stenosis reveals a 10-mm vertebral canal at the L5 level, with a 4:1 ratio of the vertebral body to the canal sagittal diameter.

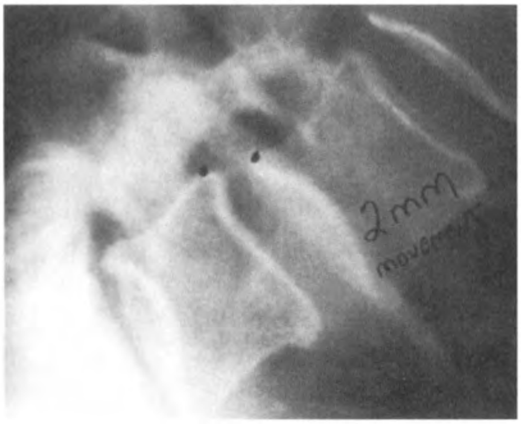

Figure 14.40. Flexion radiograph patient seen in Figure 14.38 shows only 2 mm of motion of L4 on L5, indicating stability of the functional spinal segments.

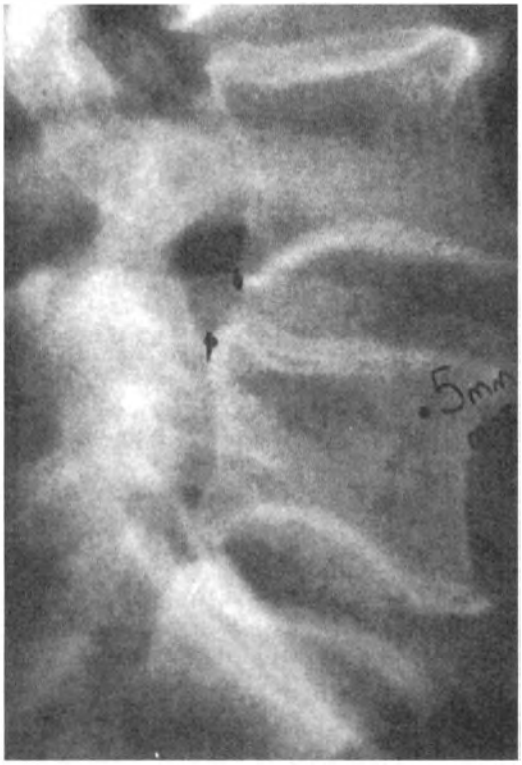

Figure 14.41. Extension reveals only 0.5 mm of motion of L4 on L5.

Tandem Lumbar and Cervical Spinal Stenosis

The triad of intermittent neurogenic claudication, progressive gait disturbance, and the findings of mixed myelopathy and polyradiculopathy in both the upper and lower extremities is the symptom complex of mixed cervical and lumbar spondylotic degeneration resulting in stenosis (113). Nineteen such patients were operated on for relief of symptoms, and none of them showed prognostically significant sphincter disturbance, radiculopathy, myelopathy, cerebrospinal fluid analysis, or electrophysiologic testing results.

Except for the intermittent claudication, the clinical presentation of tandem spinal stenosis is similar to that of classic cervical spondylotic myelopathy (107, 114–116). The insidious onset and the duration of symptoms are comparable. Although the prominence of radicular pain, spasticity, and sphincter disturbance is relatively diminished in tandem spinal stenosis, the extent of posterior column dysfunction is virtually identical. As with spondylotic myelopathy, tandem stenosis appears to be a diffuse rather than a segmental condition (117, 118).

Most patients with tandem stenosis complain of "numb, clumsy legs," analogous to the feelings reported with high cervical spine lesions. Also seen are complex gait disturbances caused by proprioceptive disturbance, lower extremity weakness, unbalanced stooped posture adopted to relieve the back and lower extremity pain, and compensatory hyperextension of the neck in order to see (113).

Tandem Spinal Stenosis

Case 5

The following is a study of tandem lumbar and cervical stenosis from the author's practice. Figure 14.42 shows a neutral lateral projection of a 64-year-old woman with gait disturbance, muscle weakness of the lower extremities, reduced ankle and patellar reflexes, pain into the lower extremities of a nonspecific dermatome nature, and equilibrium disturbance. L4 shows a degenerative spondylolisthesis on L5. Figure 14.43, a flexion study, reveals the instability of the lesion as evidenced by the marked anterior translational subluxation of L4. Figure 14.44, taken in extension, shows marked posterior translation. Figure 14.45 reveals a degenerative spondylolisthesis of C7 on T1. Also note the kyphotic curvature at the C4, C5, and C6 levels. Spondylolisthesis at both the L4 and C7 levels can inflict stenosis on the canal and its spinal contents.

Myelographic Finding in Degenerative Spondylolisthesis

Case 6

A 52-year-old woman complained of low back pain radiating into the right lower extremity for approximately the past 2 years. She sought consultation from a surgeon, and surgery was recommended to her.

Figure 14.46, a posteroanterior myelogram, reveals a filling defect posterior to the L4–L5 disc space, which represents the traction deformity at the pseudospondylolisthesis dye-filled subarachnoid space. Treatment of this patient consisted of distraction manipulation with a small flexion pillow placed under the L4 vertebral segment. The contact hand was placed on the spinous process of L3 while gentle flexion distraction was applied. This resulted in a slow but progressive relief of the patient's low back and right leg pain, and during this time she attended low back wellness school, where she was taught how to bend and lift in daily living to minimize stress to this low back. The treatment resulted in approximately 75% relief of her symptoms.

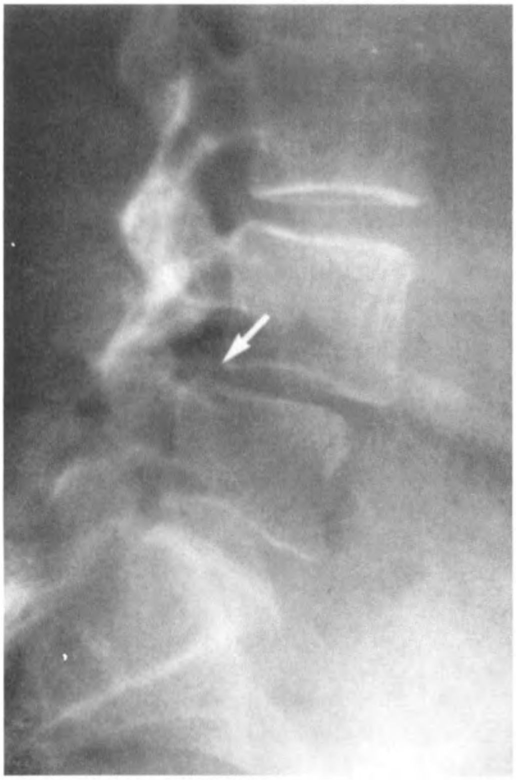

Figure 14.42. Neutral lateral projection of a 64-year-old woman with signs of stenosis of the lumbar canal shows degenerative spondylolisthesis of L4 on L5 (*arrow*).

Figure 14.43. Flexion study of patient in Figure 14.42 shows marked instability of the L4–L5 disc, as evidenced by the greatly increased translation of the L4 vertebral body on L5 (*arrow*).

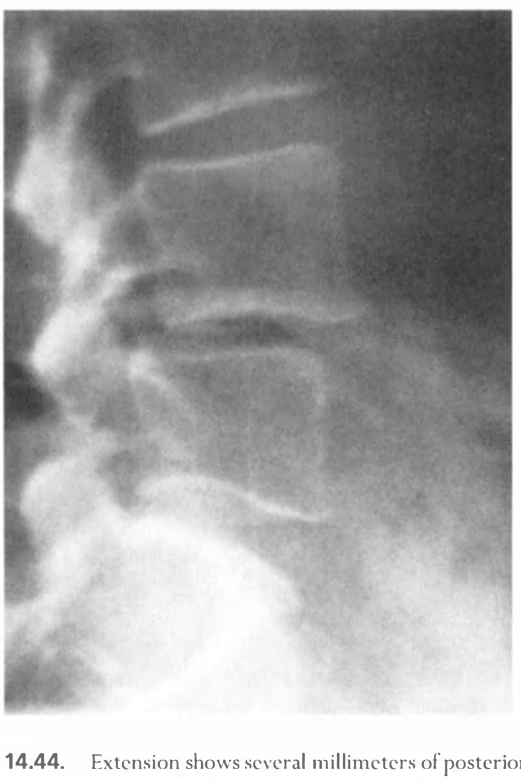

Figure 14.44. Extension shows several millimeters of posterior translation of L4, indicating marked instability.

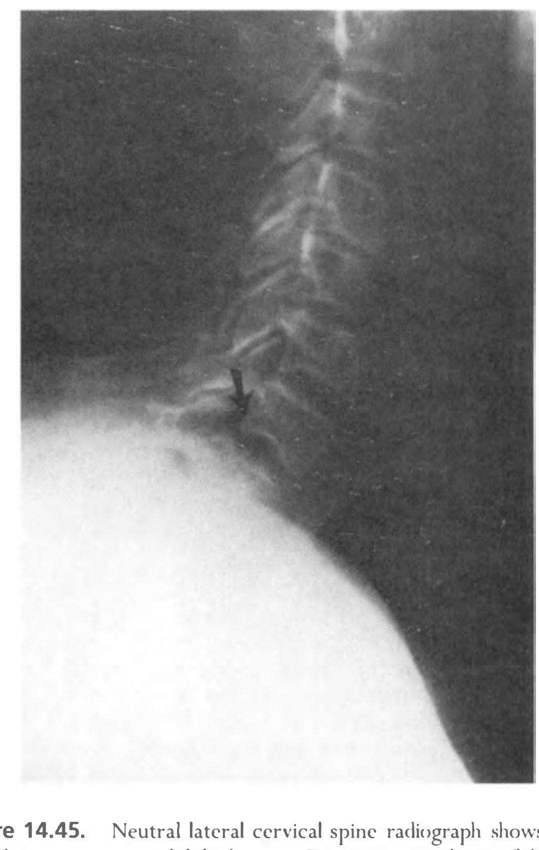

Figure 14.45. Neutral lateral cervical spine radiograph shows C7 to be in degenerative spondylolisthesis on T1 (*arrow*). Kyphosis of the sagittal cervical curve is also evident.

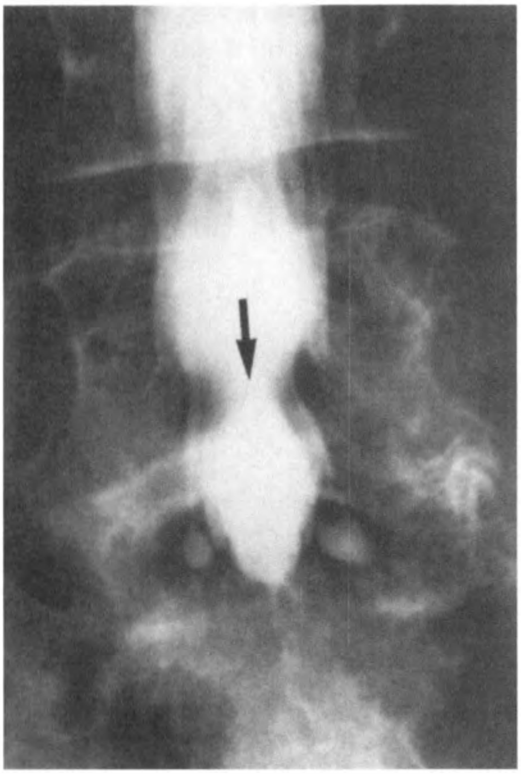

Figure 14.46. Posteroanterior myelographic study reveals narrowing of the dye-filled subarachnoid space at the level (*arrow*).

Typical Case of Degenerative Spondylolisthesis

Case 7

A 42-year-old man was seen for low back and buttock pain. Figure 14.47 reveals a degenerative spondylolisthesis of L4 on L5. Figure 14.48 is an anteroposterior view that reveals a suggestion of tropism at the L5–S1 level, with the right being sagittal and the left coronal. The facets at the remaining lumbar segments are sagittally oriented throughout. The oblique views show degenerative arthrosis at the L4–L5 and L5–S1 facet joints.

This is a good example of a patient who has vague low back and buttock pain, but no pain into the lower extremities. It is to be remembered that degenerative spondylolisthesis can often create muscle weakness and even diminished ankle jerks in the lower extremities. This patient responded well to flexion distraction with a small Dutchman roll placed under the L4 vertebral body while flexion distraction was applied with the contact hand on the L3 spinous process. Both the clinicians and the patient were satisfied with an approximately 75% relief of pain. As usual, this patient had short hamstring muscles, which is commonly seen in degenerative spondylolisthesis. The appropriate proprioceptive neuromuscular facilitation was used in stretching these hamstrings. The patient was given knee–chest exercise and abdominal strengthening exercises; he attended low back wellness school.

Figure 14.47. Neutral lateral view of a 42-year-old man with low back and buttock pain reveals an anterolisthesis of L4 on L5 (*arrow*).

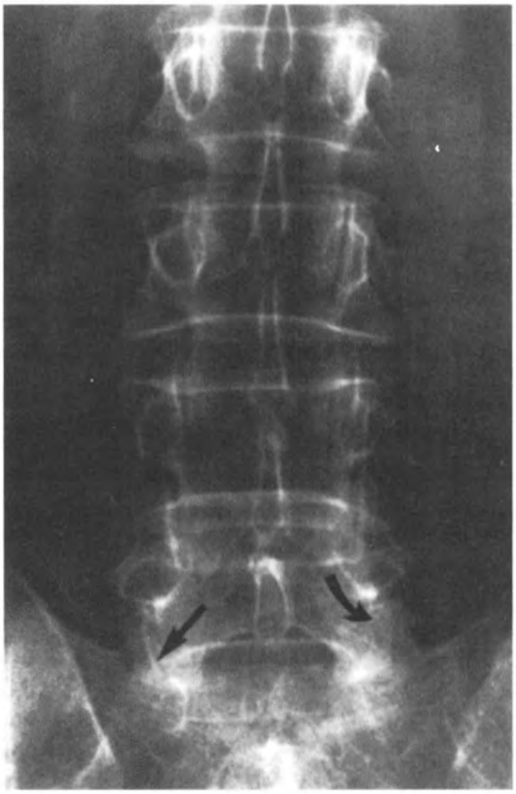

Figure 14.48. Anteroposterior view shows that tropism is suggested at L5–S1, with sagittal facet facings on the right (*straight arrow*) and coronal on the left (*curved arrow*).

Pseudosacralization with Pseudospondylolisthesis

Case 8

Degenerative spondylolisthesis at L4 is much more common in females than males. However, this case involves a 74-year-old married man who has low back and occasional bilateral leg pain, which was aggravated on ambulation. Figure 14.49 reveals the DS is approximately 10% of L4 on L5. Note the transitional segment at L5, realizing that pseudospondylolisthesis occurs four times more frequently with transitional segment (75) (Fig. 14.50). Treatment of this patient consisted of flexion distraction over a small flexion roll. The response of the patient was dramatic in that his low back pain eased, and he was able to walk without the discomfort previously encountered.

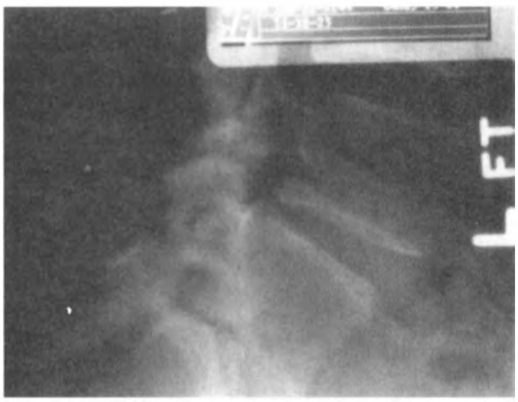

Figure 14.49. Lateral projection shows a 10% slippage of L4 on L5.

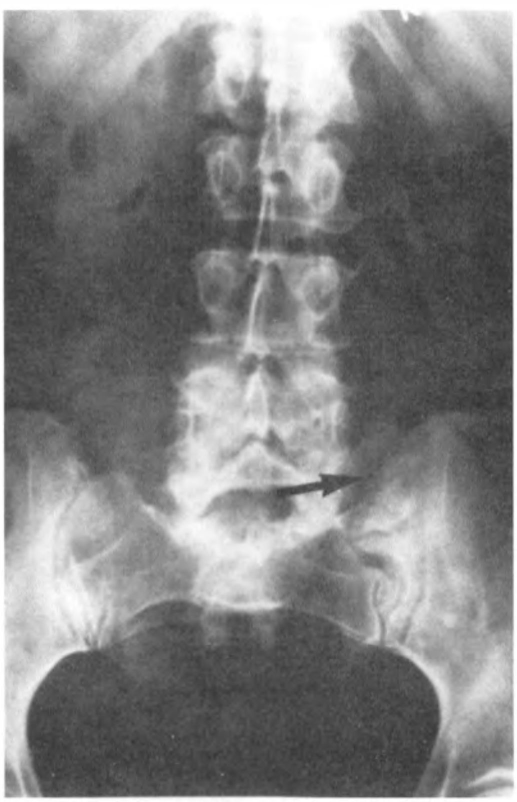

Figure 14.50. Anteroposterior study of patient in Figure 14.49 shows a right pseudosacralization of L5 (*arrow*). Here is a good example of a transitional fifth lumbar segment with degenerative spondylolisthesis of L4 on L5.

UNCOMMON VARIETIES OF SPONDYLOLISTHESIS

Traumatic

Traumatic spondylolisthesis is a fracture of any part of the vertebral arch other than the pars that allows forward displacement to occur. This type of spondylolisthesis is rare.

Pathologic

If the bony hook mechanism (articular facet, pedicle, pars) fails to hold the body of the articulation in place because of local or generalized bone disease, pathologic spondylolisthesis can occur. Because pathologic spondylolisthesis is rare, only one variant, spondylolisthesis adquista, is mentioned here. In this type, a fatigue fracture of the pars occurs at the upper end of a lumbar surgical fusion that allows forward slipping.

NONSURGICAL TREATMENT OF DEGENERATIVE SPONDYLOLISTHESIS

Distraction adjusting for degenerative spondylolisthesis is discussed later in this chapter. The following case presentations of DS are given.

Case 9

Figure 14.51 reveals 10% DS subluxation of L4 on L5 with degeneration of the L5–S1 intervertebral disc. The axial CT image (Fig. 14.52) reveals the broad-based pseudodisc of spondylolisthesis, which in this case is seen to contact the thecal sac (*arrow*). Note the anterolateral bone plate hypertrophy, indicative of the longstanding degenerative disc disease. Also note degeneration of the posterior facet joint and sacroiliac joint on the left side (*arrowheads*).

This 83-year-old man suffered from extreme low back and radiating nondermatomal pain of both lower extremities. Distraction manipulation was of minimal benefit to this patient.

Figure 14.53 reveals a more pressing problem for this man, namely an aortic aneurysm.

Instability of an L4 Spondylolisthesis Segment

Case 10

A 68-year-old man with low back and right leg pain and numbness extending through the first sacral dermatome was seen. Night pain disturbed his sleep. He sought care from his medical doctor who gave him exercises that made him worse. He was on medication for hypertension.

Lumbar spine ranges of motion are 60° flexion, 10° extension, 10° bilaterally for lateral flexion, and 20° of bilateral rotation. The sitting SLR sign was positive for both low back and leg pain. The

Figure 14.51. L4 degenerative spondylolisthesis of 10% on L5 is noted.

Figure 14.52. Axial computed tomography scan reveals the broad-based pseudodisc of spondylolisthesis (*arrows*). Note the anterolateral hypertrophic end plate changes of periosteal reaction to discal degeneration. The facet joint shows degenerative changes (*arrowhead*) as well as bone fusion caused by degenerative change of the sacroiliac joint (*arrowhead*).

patient could toe and heel walk normally; however, the right hamstring muscle was grade 4 of 5 strength compared with the left. The SLR on the right was restricted to 75°, creating both low back and leg pain. The deep tendon reflexes at the ankle and knee were bilaterally +2. Hypesthesia of the right L5 and S1 dermatomes was noted. Circulation of the lower extremities appeared normal.

Anteroposterior and lateral radiographic examination revealed L5 bilateral transverse process spatulization with bilateral true fu-

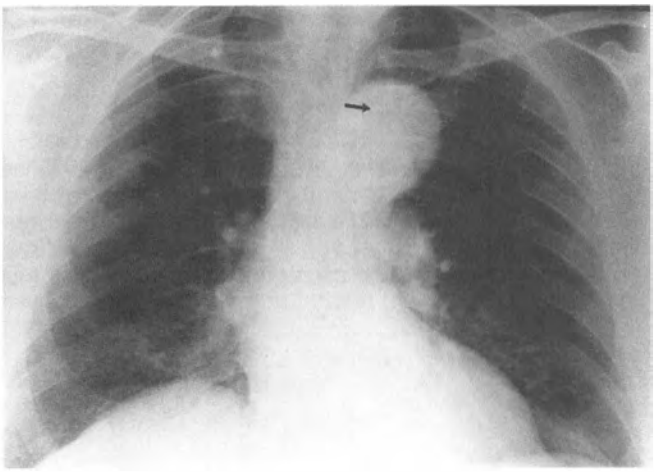

Figure 14.53. Note the aortic aneurysm (*arrow*) in the patient shown in Figures 14.51 and 14.52.

Figure 14.54. Bilateral sacralization of the L5 transverse processes is noted.

sion to the sacrum (Fig. 14.54) and L4 was approximately 11 mm anteriorly slipped on L5 (Fig. 14.55).

Vertical suspension study revealed the L4 anterior translation subluxation to reduce to 8 mm, representing a 3-mm translational motion from the 11-mm anterior slippage seen on standing neutral study.

Diagnosis was degenerative spondylolisthesis of 11 mm on neutral lateral and 8 mm on vertical suspension, representing instability. The combination of disc degeneration above a transitional segment is termed "Bertolotti's syndrome" (see Chapter 6, *Transitional Segment*) and in this case degenerative spondylolisthesis accompanies it.

Treatment

Distraction manipulation is applied to the lumbar spine with a small flexion pillow under the L4 vertebral body, and a spinous process contact on the L3 segment, while three 20-second distractive sessions are applied using the protocol of five 4-second pumps of the L3 spinous process during each 20 seconds. This is the protocol described in Chapter 9 for sciatica patients with herniated discs, and this patient with sciatica caused by DS stenosis required this approach. The adjustment was followed by positive galvanism into the L4–L5 disc space and right buttock region over the sacrotuberous ligament where the sciatic nerve passes through the pelvis. Tetanizing current was then applied to the paravertebral muscles and right hamstring muscle.

Home instructions of knee–chest exercise, hamstring stretching, and abdominal strengthening were given. The patient was instructed to take glycosaminoglycan (600 mg per day). A lumbar brace was placed on the patient's lumbar spine to stabilize the unstable L4 segment. The result was excellent relief of the patient's pain.

Surgical Fusion for Degenerative Spondylolisthesis

Pedicle fixation and fusion along with surgical decompression of degenerative spondylolisthesis is required because a significant percentage of patients have the potential to progress their listhesis slippage and clinical symptoms (119).

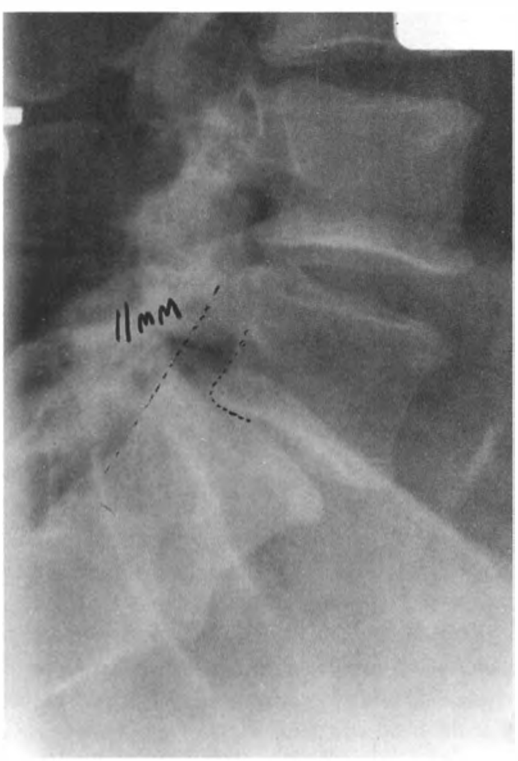

Figure 14.55. The rudimentary L5–S1 disc is noted with an 11-mm slip of L4 on L5 and marked disc degeneration of the L4–L5. This is Bertolotti's syndrome with degenerative spondylolisthesis of L4.

SAGITTAL FACET ORIENTATION EFFECT ON DEGENERATIVE SPONDYLOLISTHESIS

Sagittal facet joint orientation is seen at L4–L5 significantly often to support the hypothesis it predisposes patients to develop pseudospondylolisthesis (120). It also predisposes to postoperative spondylolisthesis, regardless of a preoperative diagnosis of DS or spinal stenosis (121).

ELONGATED PARS INTERARTICULARIS CAUSES PSEUDOSPONDYLOLISTHESIS

An intact but elongated pars interarticularis is seen in about a third of symptomatic children and adolescents with spondylolisthesis. This condition is known as "dysplastic spondylolisthesis," lumbosacral subluxation, or isthmic subtype B spondylolisthesis. The facets of the fifth lumbar vertebrae appear to subluxate on the facets of the first sacral vertebrae. As the slip progresses, the pars interarticularis becomes attenuated and elongated along with the pedicles. If the slip progresses beyond 25% and the neural arches remain intact, pressure on the cauda equina is likely. Children and adolescents may require surgical fusion as would adults with root compression symptoms (122).

TREATMENT OF ATHLETES WITH SPONDYLOLYSIS OR SPONDYLOLISTHESIS

Athletic Incidence of Spondylolisthesis

Competitive weight lifters seem to develop stress fractures of the pars interarticularis, unaccompanied by spondylolisthesis (123). It was felt that the hyperextension involved with lifting in such maneuvers as "clean and jerk" and "the snatch" in Olympic lifting and "the squat" and "the dead lift" in power lifting caused the pars interarticularis fracture. The suggestion was made that weight training by physically immature athletes should be done, in most instances, in the sitting position and avoid squats and overhead lifts.

A study found 19 of 145 freshman football players who were radiographed to have spondylolysis (13.1%). This study concluded that most affected players entered college with previously acquired spondylolysis, which seemed to indicate that their problem arose in the adolescent years during athletics or other stress situations. Linemen were felt to be more susceptible to development of this defect (124).

The intensity and repetitive nature of athletic training create situations involving a jerking motion, which is the most probable mechanism causing the fracture (27).

Semon and Spengler (20) found that spondylolysis was not a predisposing factor in low back pain in a study of college football players. Thus, it is questionable whether spondylolysis or spondylolisthesis is a cause of back pain.

Eighty-two athletes with spondylolysis or spondylolisthesis of the lumbar spine were treated with restriction of activity, bracing, and physical therapy. Of the 62 patients with spondylolysis, 84% had excellent results. Twenty patients had spon-

dylolisthesis with 12 patients (60%) requiring surgery. Indications for operative intervention include unremitting symptoms despite 6 months of nonoperative management, spondylolisthesis progression, or neurologic deficit (125).

CONSERVATIVE TREATMENT

From Chapter 9, follow the protocol for the treatment of spondylolisthesis, both true and false types, as outlined and shown in Figure 9.46 for the treatment of the patient with or without sciatic radiculopathy. Side lying range of motion adjustment is shown in Figure 9.36 for flexion, Figure 9.38 for lateral flexion, and Figure 9.39 for circumduction in patients in too much pain to lie on the abdomen.

Further Technique and Treatment Description

The paravertebral muscles can then be treated with physical modalities if needed. These modalities might well include positive galvanism to reduce inflammation and sedate irritated tissues or sinusoidal currents to return normal tone to the musculature (Figs. 14.56 and 14.57).

Figure 14.56. Application of positive galvanism or tetanizing current.

Figure 14.57. Cryotherapy is added to relieve inflammatory effects of low back pain.

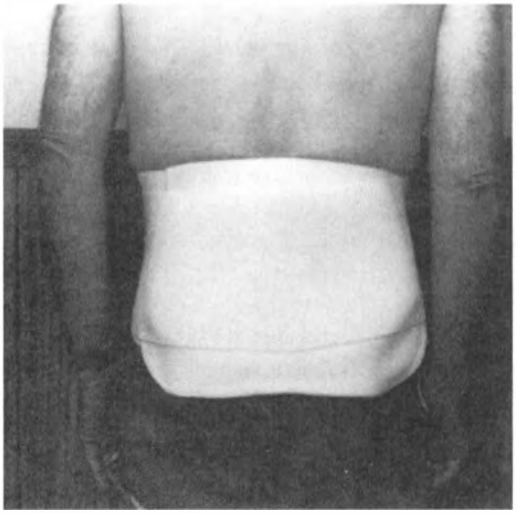

Figure 14.58. A lumbosacral support is worn in unstable spondylolisthesis cases.

Figure 14.59. Proprioceptive neuromuscular facilitation is used to stretch the hamstring muscles in spondylolisthesis cases.

A belt support can be worn if the patient is in acute pain, but this is only a temporary measure (Fig. 14.58). Exercises for the spondylolisthesis patient are extremely important. We most often use the first three Cox exercises in the treatment of spondylolisthesis. Hamstring stretching exercises (Fig. 14.59) are important to regaining normal lumbopelvic rhythm.

In a study of 47 patients with symptomatic back pain secondary to spondylolisthesis who were treated with flexion and extension exercises of the lumbar spine, it was found that patients treated with flexion type exercises were less likely to require back supports, require modification of their jobs, or limit their activities because of pain (126). Eighty-two percent of those who underwent flexion exercises stated that they had less pain, whereas 37% of those who did only extension exercises stated that they had less pain. The flexion group was found to have less pain, less need to modify their work, less need for continued use of bracing, and a greater chance of recovery. The type of spondylolisthesis had no effect on the response to flexion exercises.

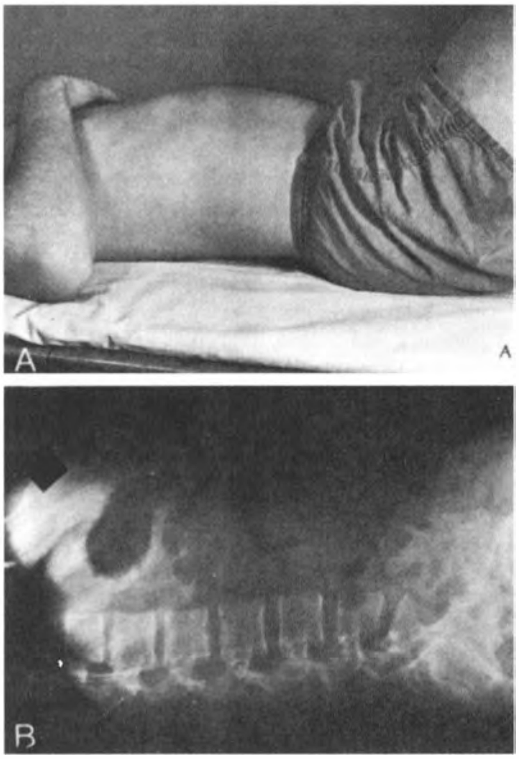

Figure 14.60. **A.** Photograph showing a decrease in lumbar lordosis while the subject is lying supine with the knees bent. **B.** Radiograph of spinal column while the subject is lying supine with the knees bent and is performing the pelvic-tilting exercise. (Reprinted with permission from Gramse RR, Sinaki M, Ilstrup DM. Lumbar spondylolisthesis—a rational approach to conservative treatment. Mayo Clinic Proc 1980; 55:681–686.)

Figures 14.60 and 14.61 reveal the effects of flexion exercises on the lumbar spine in a patient performing abdominal strengthening exercises. Note that extension exercises are to be avoided in spondylolysis and spondylolisthesis, as they have been shown to increase the pain not only in gymnasts but also in the general public.

Effectiveness of Spinal Adjustment for Spondylolisthesis

The effectiveness of spinal manipulation therapy for low back pain was compared in two groups of patients: 25 patients with lumbar spondylolisthesis and 260 patients without spondylolisthesis. The result of manipulative treatment was not significantly different in those patients with or without lumbar spondylolisthesis (127). The authors of this study chose to manipulate the level of the spine above or below the demonstrated level of spondylolisthesis. They found that 80% of the spondylolisthesis group had a good result with manipulation, versus 77% in the overall study. They concluded that spondylolisthesis does not contraindicate spinal manipulation, and in fact manipulation is an appropriate treatment for this condition.

RESULTS OF TREATMENT

Analysis of the Results of Chiropractic Treatment

Using the "time-honored" specific side posture techniques, 10 cases of "olisthesis" (3 males, 7 females) achieved the following results: 4 of the 10 (40%) showed excellent, good, or fair results and 6 of the 10 (60%) either showed no change or, unfortunately, were made worse.

Since learning the Cox lumbar flexion distraction technique, a more favorable outcome can be reported in 15 cases of olisthesis and 1 case of "oloptosis": 86.6% showed a favorable outcome (excellent, good, or fair results) and only 13.4% showed poor outcome.

Patient ages ranged from 20 years (8 years since onset of symptoms) to 62 years (30 years since onset of symptoms). Of these 15, 4 were men and 11 were women. The sex or age of the patient did not affect the results.

In only two cases could any measurable difference be detected in the olisthetic movement following treatment and that was only a 3% and a 5% improvement in position. In all of the other cases, no measurable difference was seen. In the one case of oloptosis, no detectable change of position occurred.

The procedure used was mild lumbar flexion distraction with the use of a Dutchman roll under the abdomen. After distraction, any severe lateral misalignment of adjacent bony

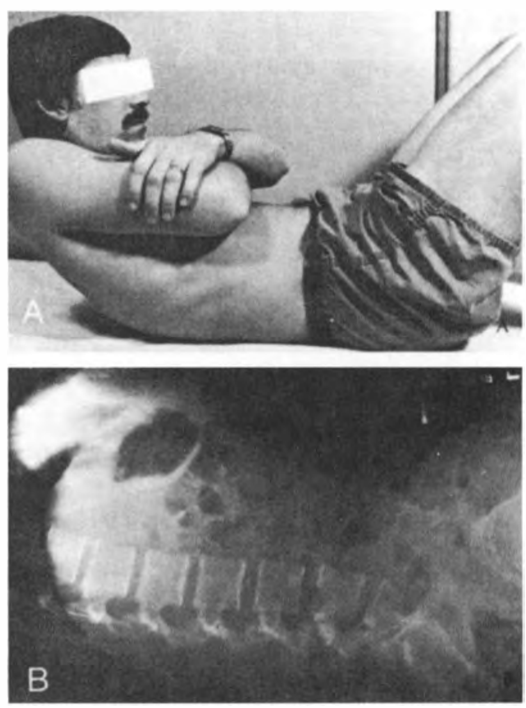

Figure 14.61. **A.** Photograph of the subject performing abdominal strengthening exercise. **B.** Radiograph of spinal column while the subject performs abdominal strengthening exercise. (Reprinted with permission from Gramse RR, Sinaki M, Ilstrup DM. Lumbar spondylolisthesis—a rational approach to conservative treatment. Mayo Clin Proc 1980;55:681–686.)

structures was specifically corrected (as much as possible) by a mild side posture technique. The patient was then instructed in a simple exercise to do at home to loosen the hamstring muscles.

The patient was then placed in a supine position with both the hips and the knees flexed at 90°, with the calf of the leg supported in that position. While in this position the patient was given a 20- to 30-minute period of very mild shortwave diathermy, with one pad under the cervical area and the other under the lumbar area. Only one patient required the use of an orthopedic lumbar restraint, which was used more for abdominal support than for lumbar support (128).

To conclude this chapter, two more cases are presented. The first case was referred to me by Jerry Wright, DC, who co-managed the case with me.

Case 11

A 41-year-old teacher was twisting sideways while restraining an unruly student, which caused low back pain, and a week later bent over and felt low back pain and right leg pain. She sought medical care, underwent diagnostic imaging with MRI, CT, myelography, and bone scanning, which were all read as negative.

At the time, the patient was depressed over not being given time off work because of her pain; she abused alcohol and attempted suicide because of depression. During hospitalization, epidural steroid injections were given; drug therapy and 2.5 months of diagnostic testing ensued. Finally, her neurosurgeon referred her to Dr. Wright. At that time she had right lower extremity pain radiating primarily to the right great toe and to a lesser extent to the remaining four toes, abdominal pain, and a feeling of difficulty urinating.

Figure 14.63. Vertical suspension is shown here revealing slight motion posterior of L3 on L4.

Figure 14.62. Neutral lateral view shows a 10% anterior slip of L3 on L4 with discogenic spondylosis changes at L3–L4. Observe the pars interarticularis for possible defect.

The patient was referred to me. Atrophy of the right gluteus maximus muscle was noted and dorsi and plantar flexion motor strength of the right foot was grade 4/5. The leg was swollen and varicose veins were prominent. Sensory examination showed hypesthesia of the right L5 and S1 dermatomes.

Radiographic examination (Fig. 14.62) is a neutral upright x-ray film showing L3–L4 degenerative disc disease with a 10% anterior slippage of L3 on L4. Note that a defect of the pars interarticularis is not evident without hindsight. Figure 14.63 is a vertical suspension study and Figure 14.64 is extension study, both showing little suggestion of the pars defect. Figure 14.65 shows flexion study, and the pars interarticularis is seen to separate (*arrow*), creating patient symptoms. Figure 14.66 is the sagittal MRI showing the anterior subluxation of L3 and the decreased signal intensity of the L3–L4 disc, indicating internal disc disruption and degeneration. Also note the pseudoherniation of the L3–L4 disc into the vertebral canal.

It was the plain x-ray study that produced the unstable spondylolisthesis diagnosis that led to distraction adjusting shown in Figure 14.67 and the stabilization with a Taylor brace shown in Figure 14.68. This combined care over a 2-month period was given as follows. The patient was treated daily with an immediate relief of the low back pain but with right lower extremity pain involving differing dermatome areas continuing. Treatment frequency was reduced to three distraction sessions a week at 50% relief and continual relief took place until at 2 months of care the patient was performing her home exercises of knee–chest and hamstring muscle stretching and was released at 2 months of care with relief of her right lower extremity pain and only occasional low back pain. A nice relief from attempted suicide to return to work without pain!

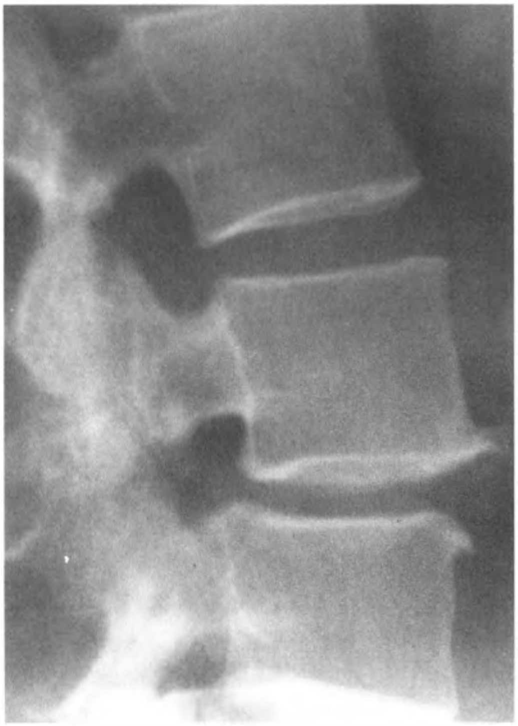

Figure 14.64. Extension motion study shows no change over neutral or extension views in Figures 14.62 and 14.63.

Figure 14.65. Flexion motion study shows the pars interarticularis to separate (*arrow*). This is the only motion study to reveal this diagnosis of instability.

Figure 14.66. Magnetic resonance imaging shows loss of signal intensity of the L3–L4 disc with pseudodisc bulge into the anterior vertebral canal and the anterior slip of L3 on L4.

Figure 14.67. Distraction adjusting with the lumbar flexion roll under the L3 spondylolisthesis subluxation and contact for distraction made above at the thoracolumbar spine when distraction is applied.

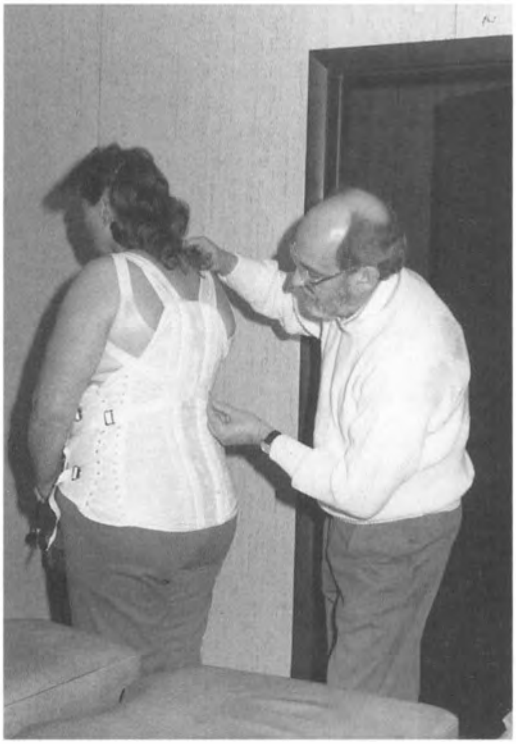

Figure 14.68. Brace worn for stabilization of the L3 spondylolisthesis condition shown in Figures 14.62 to 14.67.

Figure 14.69. Lateral radiograph shows the anterior slip of L4 on L5 and the pars interarticularis defect.

Figure 14.70. Oblique view confirms the pars interarticularis defect (*curved arrow*).

Figure 14.71. Axial single photon emission computed tomography (SPECT) scan shows the right pars interarticularis increased uptake indicative of fracture.

Figure 14.72. Coronal single photon emission computed tomography (SPECT) scan shows the increased uptake of the right L4 pars interarticularis.

Case 12

The final case is a professional hockey player injured in a game. He developed severe low back pain, and plain lateral lumbar x-ray study (Fig. 14.69) shows spondylolisthesis of L4 on L5, whereas Figure 14.70, an oblique view, shows a pars interarticularis defect (*arrow*). Because of extreme pain, a SPECT scan was performed. Figure 14.71 shows the increased uptake of radionuclide on axial view, and Figure 14.72 shows the increased uptake of the right pars on coronal SPECT scan. Treatment of this patient consisted of rest, bracing, gentle distraction adjusting, and rehabilitation exercises as pain and healing of the fracture progressed. This hockey player was back to full action within 6 weeks of the injury. An excellent case to conclude this chapter.

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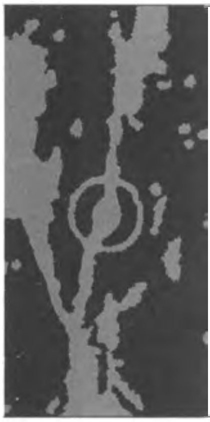

Rehabilitation of the Low Back Pain Patient

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chapter **15**

A hundred times a day I remind myself that my life labors of other men, living and dead, and that I must exert myself in order to give, in the measure as I have received, and am still receiving.

—Albert Einstein

An exciting model of functional restoration for low back pain and dysfunction has been rapidly evolving. This model draws from the disciplines of manipulation and physical medicine, and physical therapy and rehabilitation. It empowers the clinician with the ability to identify and successfully treat simple, acute cases as well as complicated, chronic cases of low back pain. Strong emphasis is placed on rapid recovery of function, early patient involvement, and prevention of disability. Catalyzing this shift in our clinical approach to low back pain (LBP) was Gordon Waddell, who exposed the shortcomings of traditional medical care in LBP management (1). He criticized the tendency to overprescribe bed rest and surgery, to overemphasize structural diagnosis, and the failure to recognize abnormal illness behaviors. Waddell challenged us to rethink our treatment approach and consider instead an integrated biopsychosocial model emphasizing functional recovery (1–3).

Recovery of function in the locomotor system includes rehabilitation of both the muscular and joint systems. Lewit and Janda have provided a working formula of dysfunctional muscular and joint chains as they relate to disturbed motor function (4–9). Recently, in the chiropractic profession, Liebenson has contributed a synopsis of these and other concepts and procedures that make the transition into the active care model systematic and practical (10–12). The use of manipulation, passive modalities, and exercise will be reviewed briefly. We will examine spinal function as it relates to production of stability through coordinated muscular activity and balance. Next, we will explore low technologic methods of assessing functional capacity of the low back patient that drive our treatment and rehabilitative decision-making. Ultimately, we will reveal exercise procedures designed to correct functional deficits using several techniques.

OVERVIEW: EXERCISE, FUNCTIONAL RECOVERY, AND LOW BACK PAIN

Exercise for the treatment of LBP is not a novel idea, and it is becoming increasingly clear that functionally oriented care for lumbar spine management is the growing trend (13). Liebenson et al. have stated: "Functional restoration of activity limitations is considered the standard of care for patients with subacute, recurrent and chronic low back pain" (14). Active care approaches for chronic LBP emphasizing functional recovery are demonstrating superior results when compared with passive approaches that emphasize pain relief (15–18). Interestingly, recovery of function seems to be correlated well with pain relief. In one study, physical agents (i.e., hot packs and ultrasound), when used alone, were found to be no better than no treatment at all. Two exercise groups were also compared in this study. One group was considered "high tech," and exercise was achieved through equipment-based, in-clinic protocols. The second exercise group received McKenzie extension and spinal stabilization exercise. Both exercise groups attained significant improvements in chronic low back pain with the low tech exercise group having the longest period of symptom relief (18). In addition to the pain-relieving potential, exercise speaks to the issues of enhanced functional capacity. Saal and Saal (15) sparked much interest in using aggressive spinal rehabilitation programs in patients with documented herniated lumbar disc with associated radiculopathy. They demonstrated a high return to work rate following their program, and further concluded that nonoperative treatment in this patient group is a viable option. Their rehabilitation approach included the use of spinal stabilization exercise integrated with cardiovascular conditioning, flexibility routines, and isotonic strengthening

(15). Other programs have documented the beneficial effects of specific exercise approaches in improving physical and psychological parameters in low back pain patients (19–22).

MANIPULATION AND EXERCISE: THE CONTINUUM

Spinal manipulation for LBP is an established method of care, especially in the acute stage of recovery (13, 23–26), and evidence exists supporting its effectiveness in chronic and complicated populations (24, 27). Yet providing passive care, including spinal manipulation only beyond the acute stage, is not well supported (13, 17, 18, 23). In the chiropractic setting, blending manipulative care with exercise is necessary. This has been the focus of recent work that demonstrated a superior clinical result in low back pain patients (17, 18). Sacroiliac manipulation combined with flexion and extension mobilization exercises produced superior functional recovery when compared with extension exercises alone (22). Although the rationale for implementing exercise in the management of chronic and recurrent LBP patients is strong, the practical transition can be challenging. Liebenson has described an active–passive care continuum that is both logical and useful in understanding the timing of active care implementation. In the early stage of injury recovery, passive modalities are indicated, and the main goals of care include pain reduction, relative rest, and prevention of further injury. As healing continues, progressive movement is strongly indicated and gradually implemented. In most cases, patients can begin a transition toward active care in the late acute, early subacute stage of recovery. Chiropractic management of lumbar spine patients, especially those at risk for developing chronicity, must include timely conversion of the care plan from a passive emphasis of treatment to an exercise-based active regimen (12, 23, 28–30).

UNDERSTANDING SPINAL STABILITY

The spinal column must accomplish two primary tasks that, from a mechanical standpoint, seem diametrically opposed. On the one hand, the spinal column must be flexible. On the other hand, it must be stiff and rigid, especially when under load, to maintain anatomic relationships and protect the neural elements. The ability to perform these two functions without compromise is the essence of stability. The prevailing model describing how spinal stability is produced has been developed by Panjabi. According to this model, spinal stability is initiated when the neural subsystem receives movement, load, and position information from receptor organs located in joints, muscle, and ligaments. The neural subsystem determines specific requirements for postural control and movement and activates the muscular system. Coordination and integration of the central (neural), passive (osteoligamentous), and active (muscular) subsystems produce balanced reactions to allow the spinal column to carry out its dual role (31–33). These components and their functions are outlined in Table 15.1.

According to Wilder et al. rapid activation of muscular patterns is chiefly responsible for producing spinal stability (34).

Table 15.1

Panjabi Model of Spinal Stability

Subsystem	Components	Function
Passive (Osteoligamentous)	Vertebrae Intervertebral discs Facet articulations Spinal ligaments Joint capsules	Stability toward end range Relay position and load information
Active (Muscular)	Spinal column muscles	Force generation Movement Stability
Control (Neural)	Force/motion transducers located in muscle, ligament, and tendons	Process information from active and passive systems Coordination of stabilizers and movers

Based on reference 31.

These muscular responses are governed by central nervous system control mechanisms, chiefly by spinal reflexes. The osteoligamentous structure provides the base upon which the previous systems will act, and it provides a source of feedback information regarding position and load. In short, the muscular system provides the first line of defense against buckling of the osteoligamentous column. This "defense" is coordinated by the nervous system.

MECHANISMS OF SPINAL STABILITY

Four mechanisms influence spinal stability and they are directly related to the stabilization of the osteoligamentous subsystem by the muscular subsystem. These mechanisms include fast activation speed of key spinal stabilizers, (12, 31, 35–37), coordinated muscular co-contraction, adequate endurance (12, 38–40), and sufficient prime mover strength (12, 40, 41). The relationship of these mechanisms to specific muscle groups is detailed in the next section.

Spinal Extensors

The spinal extensor muscles may be anatomically categorized as the deep multifidi (Fig. 15.1) and superficial erector spinae (Fig. 15.2) divisions. The erector spinae by way of their length and larger size are equipped to perform large sagittal plane movements, maintain lordosis, preserve lumbar posture, and counterbalance loads during lifting (33, 42). The multifidi are considered spinal intersegmental stabilizers, and they are active throughout the entire trunk flexion range, especially when rotational forces are introduced (43). The multifidus provides segmental stiffness and motion control. The proximity of the

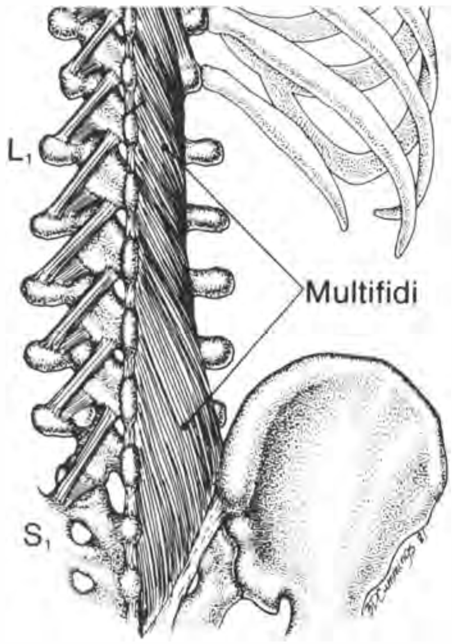

Figure 15.1. The deep multifidi muscle.

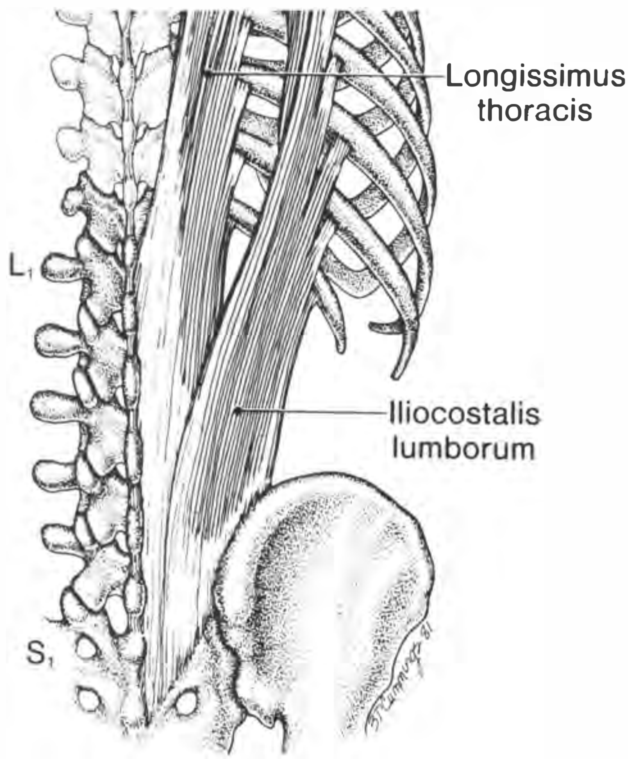

Figure 15.2. Superficial erector spinae (longissimus thoracis, iliocostalis lumborum).

multifidus to spinal segments provides a biomechanical advantage for fine tuning stability through rapid speed of contraction (33, 40, 42, 44). The histology of this muscle demonstrates a predominance of postural fibers correlating well with its proposed stabilizing function (45–47).

Abdominal Muscles

As a group, the abdominal muscles (Figs. 15.3) have received significant attention with respect to low back pain management and rehabilitation beginning with Williams (48). The rectus abdominus parallels the superficial erector spinae in that it is primarily responsible for producing large trunk movements, in this case forward flexion, and it has an important overall postural role in preserving lumbar lordosis.

The transversus abdominus (TrA) and oblique abdominals have gained considerable notoriety as important stabilizers of the lumbar spine (42, 49–52). They enhance stability of the lumbar spine by limiting translation and rotation. (51). The TrA is the first abdominal muscle to be recruited during small amplitude, rapid trunk movements (53), and when limb movements are initiated (54). It inserts posteriorly into the thoracolumbar fascia and anteriorly to the rectus sheath. The internal oblique abdominus and TrA are the only muscles to have both anterior trunk and spinal connections (42). Of special notation, the TrA was the only muscle to demonstrate marked activity with isometric trunk extension, and it was the muscle most consistently related to changes in abdominal pressure for increased spinal stability (42).

Quadratus Lumborum Muscle

The quadratus lumborum (QL) (Fig. 15.4) is another important stabilizer of the spine (39, 55). Its attachment to each lumbar transverse process (56–58) and multilayered arrangement increase lateral stability. It has been shown to be significantly active during a variety of daily activities that require dynamic spinal stability, including trunk bending and twisting. A rehabilitation exercise called the "isometric side support," (see Fig. 15.20) has been demonstrated to maximally recruit the QL for spinal stabilization (55).

LINKING MUSCULAR DYSFUNCTION WITH LBP

Altered co-contraction of agonist-antagonist muscle groups, decreased speed of contraction, and stabilizing muscle atrophy all seem to play a role in the link between LBP and decreased spinal stability. Grabiner et al. showed that LBP patients demonstrate asymmetric timing of paraspinal muscle contraction and asymmetric amplitude of contraction. They suggested that LBP is related to physiologic disturbance in paraspinal muscular control (59). Hodges and Richardson examined the speed of contraction of several trunk muscles including the TrA. They found that during rapid movement of the upper extremity (flexion, extension, abduction), the group with LBP showed significantly slower speed of activation of the trunk stabilizers, specifically the TrA, when compared with pain-free controls (60).

Clinically important information exists linking dysfunction of the multifidus muscle and low back pain. Pathologic changes have been identified in the multifidus muscle (particularly the type I fibers) of patients with recurrent LBP (44). Although these changes in the chronic population may not be surprising,

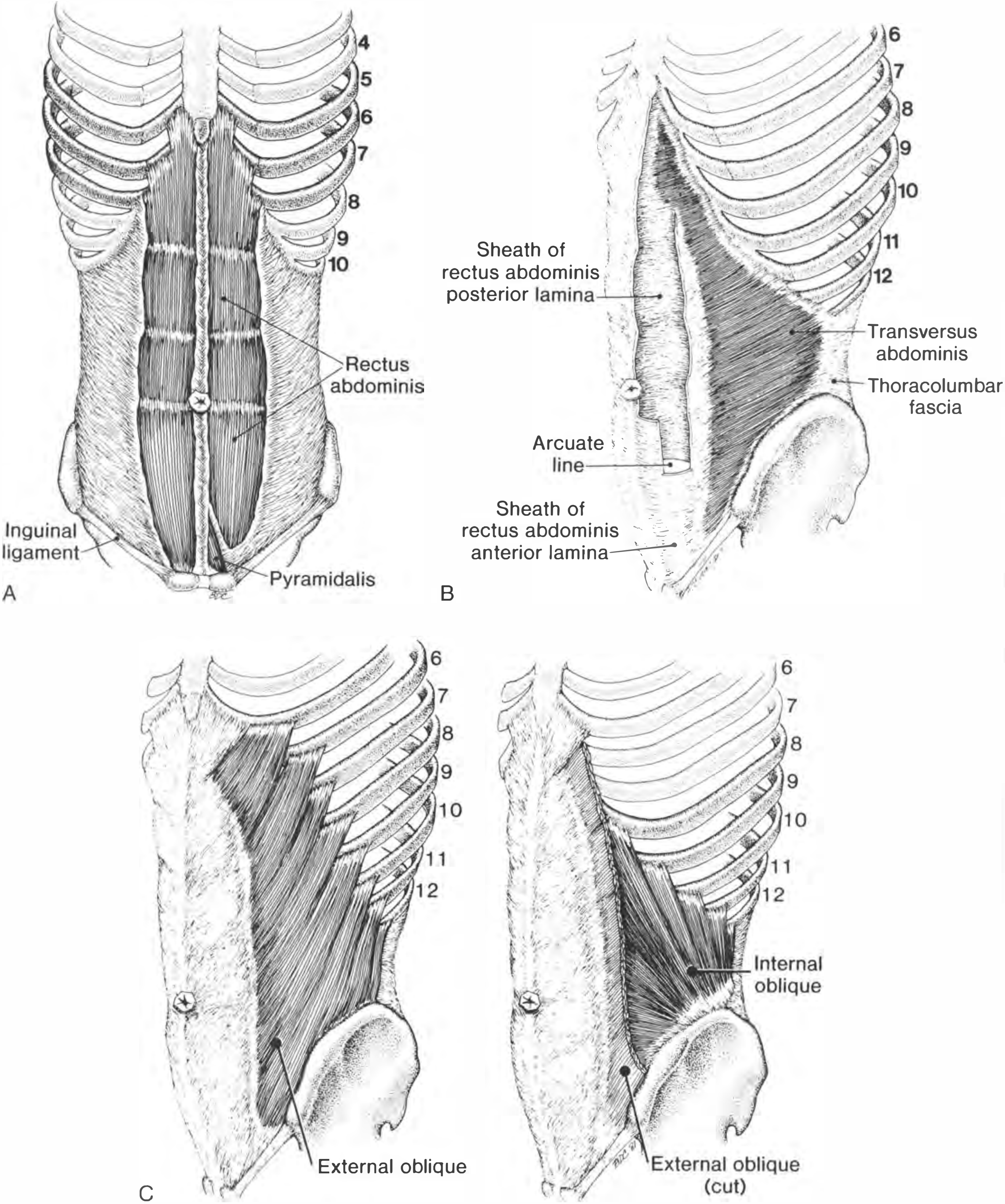

Figure 15.3. **A.** Rectus abdominis muscle. **B.** Transversus abdominis muscle. **C.** Internal/external oblique abdominal muscles.

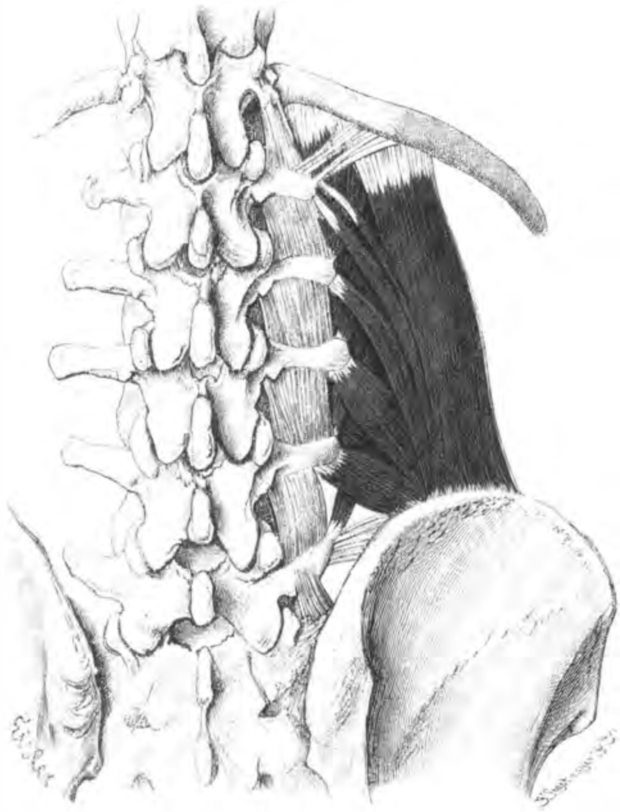

Figure 15.4. Quadratus lumborum muscle.

multifidus muscle wasting has been demonstrated on the symptomatic side shortly following the first episode of LBP (61). Hides et al. reported segmental multifidus muscle atrophy ipsilateral to low back pain in acute and subacute patients as well (44). This atrophy does not spontaneously recover but does respond to exercise (44). Others have shown increased fatigue tendency in the multifidus muscle of chronic low back patients (62). Poor endurance capacity of the spinal extensor muscles has been used to predict both first-time occurrences and recurrences of low back pain, which provides the rationale for an important functional test, namely Sorenson's static back extensor endurance test (see Fig. 15.8) (63–66).

In addition, other physiologic parameters show association to LBP. Chronic LBP sufferers have been shown to exhibit significantly lower peak torque and decreased electromyography (EMG) activity in the paraspinal muscles (67). Other indicators of paraspinal dysfunction include altered strength ratios between trunk flexors and extensors and abnormal relaxation responses (68). These deficits in the superficial paraspinals may contribute to muscular insufficiency during demanding tasks, such as lifting (55).

EVALUATING MUSCLE BALANCE: CONCEPTS AND COMMON SYNDROMES

Although the current literature regarding spinal stability, dysfunction, and instability is compelling, the clinician needs a conceptual and practical framework to understand muscular dysfunction. Janda has provided a model of assessment of locomotor function that interdigitates well with Panjabi's model. According to Janda, stereotypic muscular responses can be determined and related to predilections toward either tightness (overactivity) or weakness (inhibition) (41, 69). These tendencies are based on the tonic (postural) or phasic (mover) roles these muscles play in posture and movement. In essence, postural muscles have a tendency toward hyperactivity whereas the phasic muscles tend toward hypoactivity. Although both slow twitch (postural, type I) and fast twitch (phasic, type II) fibers exist in all muscle, a predominance of fiber type reflects the imposed demand of that particular muscle group. These activity tendencies are influenced heavily by how the muscle is used. For example, postural muscles (e.g., the iliopsoas and quadratus lumborum) have spinal stabilizing functions and higher populations of slow twitch fibers, enhancing endurance capability. Phasic muscles such as the gluteus maximus and tibialis anterior have a predominance of fast twitch fibers relating to movement generation (41).

Modern sedentary lifestyle has a major impact on the development of muscular dysfunction. A tendency is seen toward overuse of postural muscles because of prolonged constrained postures (i.e., flexed postures during sitting). Phasic muscles, on the other hand, tend to become inhibited and weak primarily because of disuse (12, 70, 71). A compilation of muscular tendencies particularly relevant to the low back pain patient is shown in Table 15.2. This table has been summarized to include those muscles of significant interest in the LBP patient.

In addition to understanding muscular tendencies, we must also consider how muscles are involved in the production of movement. For a particular movement, it is necessary to un-

Table 15.2

Muscle Tendencies

Tightness Prone	Inhibition Prone
Iliopsoas	Gluteus maximus
Rectus femoris	Gluteus medius
Erector spinae (iliocostalis lumborum and longissimus thoracis)	Lower trapezial fibers
	Serratus anterior
	Rectus abdominis
Quadratus lumborum	Oblique abdominals
Piriformis	Transverse abdominis
Hamstrings	Tibialis anterior
Tensor fascia latae	Peroneus longus
Thigh adductors	
Gastrocsoleus complex	

Modified with permission from Janda V. Muscle weakness and inhibition in back pain syndromes. In: Grieve GP, ed. Modern Manual Therapy of the Vertebral Column, New York: Churchill Livingstone, 1986:197–201; Janda V. Evaluation of muscle imbalance. In: Liebenson C, ed. Rehabilitation of the Spine: A Practitioner's Manual. Baltimore: Williams & Wilkins, 1995; and Jull G, Janda V. Muscles and motor control in low back pain. In: Twomey L, Taylor J, eds. Physical Therapy for the Low Back: Clinics in Physical Therapy. New York: Churchill Livingstone, 1987.

derstand which muscle is the primary mover (agonist), which muscle assists the primary mover (synergist), and which muscle performs the opposite motion (antagonist). Agonist and antagonist muscle groups are governed by Sherringtons' Law of Reciprocal Innervation (72).

When agonist–antagonist relationships are disrupted because of injury, constrained postures, or overuse, muscle imbalance results. These imbalances lead to disturbed movement during functional activities, and they interrupt coordinated muscular activity required for stabilization. It is common to observe weakness in a primary mover with corresponding overactivity of the movement synergist and antagonist. These patterns of imbalance can lead to common and clinically significant consequences. A common sequela of muscle imbalance is the development of muscular trigger points. The negative effects of trigger points on muscle function has been well documented (73). Additional consequences of muscle imbalance include altered joint mechanics causing uneven distribution of articular pressure and altered centers of rotation, which ultimately results in joint dysfunction and pain. Additionally, areas of joint hypomobility are often accompanied by hypermobility in adjacent segments. Poor proprioceptive processing with impaired reciprocal relationships between agonists and antagonists is the result. Finally, alteration of entire motor patterns and gait can occur, leading to commonly encountered muscle imbalance syndromes.

Muscle Imbalance Syndromes

Muscle imbalance in the pelvic region results in a clinical scenario known as the "lower crossed syndrome" (LCS) also called the "pelvic crossed syndrome." The LCS is characterized by overactivity of the hip flexor and spinal erector muscles and weakness of the abdominal and gluteal muscles. The pelvis commonly tilts anteriorly with resultant lumbar hyperlordosis. Decreased hip extension during gait is often observed. Clinical consequences include increased thoracolumbar facet and sacroiliac joint strain, altered hip mechanics, and overstress of the lumbosacral junction (9, 41, 69).

The "layer syndrome" involves generalized deconditioning and extensive muscle imbalance throughout the body. Alternating layers of tight and weak muscle groups with disturbance of several movement patterns is found. Overactivity is found in the hamstrings, thoracolumbar erector spinae, scapular elevators, and deep neck extensors with weakness in the gluteals and lower scapular fixators, abdominals, and deep neck flexors. Clinical consequences include poor trunk stabilization, joint hypomobility especially in transitional regions, symptom chronicity, and potential for poor clinical outcome. These syndromes and muscular imbalance in general, are identified by postural observation, gait evaluation, and movement assessment. These factors are discussed in the following section (9, 41, 69).

Muscle Imbalance, Instability, and Injury

Instability occurs in the spinal column when abnormally large or poorly controlled intervertebral motions excite nociceptors to the degree that pain is produced (31). The presence of injury, degeneration, or disease, in one or all of the three stabilizing subsystems, has the potential to lead to instability. Instability can arise in the presence of joint hypomobility or hypermobility, impaired sensory processing, muscular weakness or fatigue, incoordination, or muscular hypertonicity. Persistent dysfunction, beyond the compensatory ability inherent in the stabilizing system, can result in tissue deformation, activation of nociceptors, and eventually pain (31). Cholewicki and McGill suggested that injury risk in the spine can occur under two circumstances. Injury risk is highest when the stabilizing system is either partially active (i.e., during low demands) or when task demand is exceedingly high, causing tissue failure (39). Injury during obvious tissue overload is easily understandable, but acute pain episodes arising from trivial movement or activity is somewhat less obvious. Bending down to pick up a pencil or reaching across a desk are simple tasks that are commonly reported as precipitating events of LBP. Low activity of the muscular stabilizers is the probable cause for instability and injury during such simple incidents. This may explain why an individual can work at a demanding occupation, yet experience an acute pain episode following an unsophisticated activity (39).

Mechanoreceptors located in the skin, joints, and muscles provide afferent feedback to the brain and spinal cord. This sensory information is vital for producing coordinated motor output. Stimulation of mechanoreceptors activates muscular stabilization of the joint system via spinal reflexes. This mechanism can be interrupted by discrete or repetitive injury causing damage to mechanoreceptors altering proprioception and reflex stabilization if left uncorrected. This impaired relationship may be significant enough to change motor regulation of posture and movement. Recent studies have documented the effect of pain, injury, and muscle fatigue on spinal function (74, 75).

Subjects with a known history of low back problems have demonstrated great discrepancies in their ability to detect passive movement in the lumbar spine. Increasing age and increasing number of years on the job positively correlated with diminished spinal proprioceptive ability. Exposure to cumulative trauma was theorized to play a role in these findings (74). Onset of muscular fatigue during repetitive trunk motion resulted in progressively erratic motion. These findings were attributed to decreased neuromuscular control, which may provide a causative link between muscular fatigue, instability, and spine injury (75).

Peripheral joint injury at the ankle appears to have a detrimental effect on local sensory perception as well as hip and pelvic motor function. Significant differences in vibration sense between injured and noninjured subjects have been demonstrated following ankle injury. While testing prone active hip extension, the onset of gluteus maximus activity was significantly delayed in the injury group. It was suggested that decreased extensor activity on the side of ankle injury, not just at the site of injury, is common. Alteration in gluteus maximus activity beyond the painful period may be attributed to gait pattern change during injury recovery (76). Additionally, deficits

in postural reflexes and standing balance have been identified in low back pain patients. These deficits are thought to contribute to injury susceptibility and recurrent pain (34, 77).

PATIENT ASSESSMENT

Functional evaluation for rehabilitation begins in the initial interview by identifying interruption of daily activity and employment demands. Patient-generated outcome tools are helpful in assessing activity levels and limitations. The goal of functional testing is to identify deficits in flexibility, strength, coordination, and endurance of muscles related to LBP. Testing strategies include both quantifiable (measurable) and qualifiable (graded as pass or fail) tests that together provide a clear picture of the patient's functional deficits.

Patient-Generated Outcome Tools

Self-administered questionnaires such as the Oswestry Low Back Disability i

are extremely useful in patient evaluation. These tools are quickly and easily administered. They are valid and reliable indicators of perceived functional abilities and pain levels (30). The various categories within the Oswestry questionnaire focus on limitations of activities. For example, if the patient scores high in the sitting tolerance section, the clinician can focus on the details of the intolerance during the history. During the physical examination, the clinician can select specific movements and functional tests related to those intolerances. Ultimately this will influence rehabilitation decisions, and improvement in scores over the course of treatment indicates clinical progress. A more complete discussion of these tools is found in other sources (30, 79, 80).

Functional Testing

Quantifiable tests allow comparison of the patient's current functional status with that of similar individuals matched for age, sex, and occupation (66). These tests prompt the decision-making regarding continuation of care, allow change in the treatment strategy, guide the implementation of rehabilitation. The clinician can measure the response to the rehabilitation program over time. This is important in making clinical decisions and for documenting the necessity of rehabilitative exercise (79, 80). The qualifiable tests, on the other hand, possess utility in their ability to direct specific aspects of the rehabilitation treatment approach. These tests are based primarily on the work of Janda (Table 15.3) (9, 41, 69).

Quantifiable Tests

Quantifiable tests establish a baseline level of functional or physical capacity. Yeomans and Liebenson have compiled a number of helpful procedures termed the "Quantitative Functional Capacity Evaluation" (79, 80). These tests are a low-cost alternative to expensive, computerized testing procedures. These tests are safe, reliable, valid, and practical, and they have

Table 15.3

Summary of Functional Testing

Quantifiable (Measurable)	Qualifiable (Graded)
Modified Thomas'	Hip extension
Repetitive squat	Hip abduction
Repetitive trunk curl	Trunk curl
Static back endurance	Squatting
Single leg standing	

a high degree of utility. The quantifiable procedures directly applicable to the low back pain patient measure flexibility, strength, and endurance parameters.

Flexibility Test

The modified Thomas (81–83) flexibility test (Figure 15.5) is outlined below.

Test goal: This test provides an assessment of the psoas, rectus femoris, and thigh abductors and adductors.

Rationale: Janda (9, 41, 69) has identified the hip flexors, abductors and adductors as hypertonic prone muscles that hold the capacity to alter hip and pelvis mechanics. Hip joint function is integral in restoring lumbopelvic function and it is often overlooked in traditional lumbar spine evaluation.

Test mechanics: The patient begins in a seated position with the ischial tuberosities against the examination table. The patient flexes the contralateral hip and knee and holds that position while the examiner assists the patient into a supine position. The lumbar spinous processes should be flat against the examination table in a neutral lumbar spine posture during testing. Inclinometric measurement of the hip flexion angle can be done. The degree of knee extension present is an indicator of rectus femoris length. Soft tissue resistance in the abductors (iliotibial band and tensor fascia lata) and adductors (adductor magnus and longus) should be estimated in this position. Assessing the hamstrings and gastrocsoleus complex has been covered in detail elsewhere (79, 80). Inclinometric measurement of these parameters enhance quantification. These muscle groups are important in squatting and gait mechanics, and in controlling lumbopelvic rhythm during forward bending.

Strength and Endurance Tests

Following are outlined strength and endurance tests.

Repetitive Squat (65, 66) (Fig. 15.6).

Test goal: Quadricep and gluteal dynamic endurance.

Rationale: Squatting is an essential task that is frequently deficient in the LBP patient. Several strategies for reducing lumbar spine stress center on the ability to transfer forces to the lower extremity during bending and lifting. Studies have demonstrated that quadriceps fatigue during repetitive squatting leads to a stooping posture with loss of lordosis

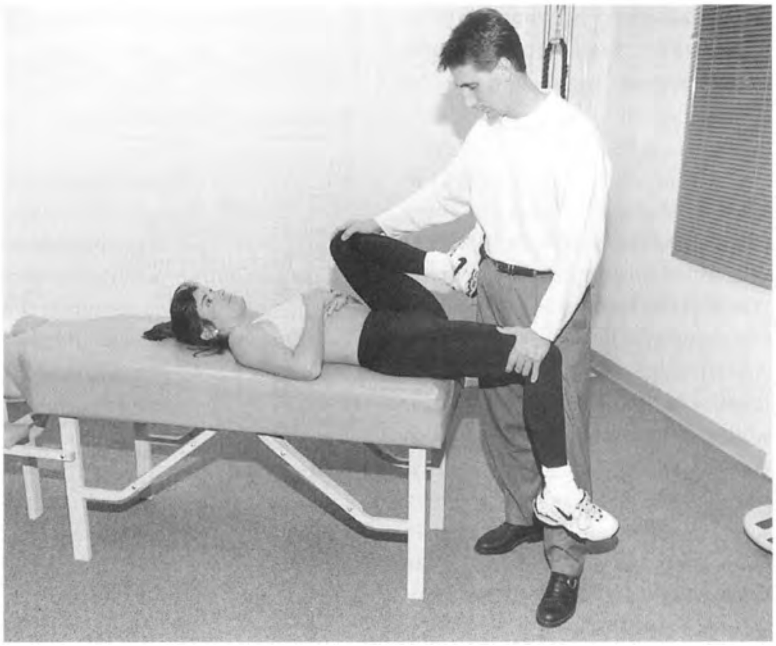

Figure 15.5. Modified Thomas' correct test mechanics.

Figure 15.6. Repetitive squat correct test mechanics.

during lifting (84–87). Inappropriate squat technique could be a source of recurrent stress to the lumbar spine.

Test mechanics: The patient stands with feet 15 cm apart (roughly shoulder width) and squats until the thighs parallel the floor. Repetitions should take 2 to 3 seconds to complete. The patient is instructed to perform as many repetitions as possible with a maximum of 50. Normative data are age, gender, and occupation specific as outlined by Yeomans and Liebenson (79, 80).

Repetitive Trunk Curl (65, 66, 88) (Fig. 15.7).

Test goal: Abdominal and hip flexor dynamic endurance.

Rationale: Rectus abdominus is an important trunk postural stabilizer in the sagittal plane and a producer of trunk flexion. It is often weak in LBP patients (41, 69).

Test mechanics: The patient is supine with knees 90° flexed and ankles fixed. The patient is instructed to perform a sit-up until the thenar eminence reaches the patella. The patient then uncurls back to the supine position. Repetitions are counted to a maximum of 50 or best effort. Normative data are age, gender, and occupation specific (79, 80).

Sorenson's Static Back Endurance Test (63, 65, 66, 83) (Fig. 15.8).

Test goal: Static back extensor endurance.

Rationale: The multifidi muscles are key lumbar spinal segmental stabilizers.

Test mechanics: Patient is prone with anterior superior iliac spine (ASIS) at end of the examination table. The trunk and arms are extended off the table with the ankles and thighs supported or strapped. The patient attempts to maintain a neutral position (not hyperextension) and holds for as long as possible or a maximum of 4 minutes. The test is positive when the patient can no longer hold the position or develops back pain. Compare with normative data (79, 80).

Single Leg Stance (12, 77) (Fig. 15.9).

Test goal: To assess balancing ability during single leg stance activities and to gauge rapid reflexive corrective spinal movement.

Test rationale: Single leg standing requires quick reflex activation of postural stabilizers in the trunk and lower extremity. Chronic LBP patients demonstrate poor control of an-

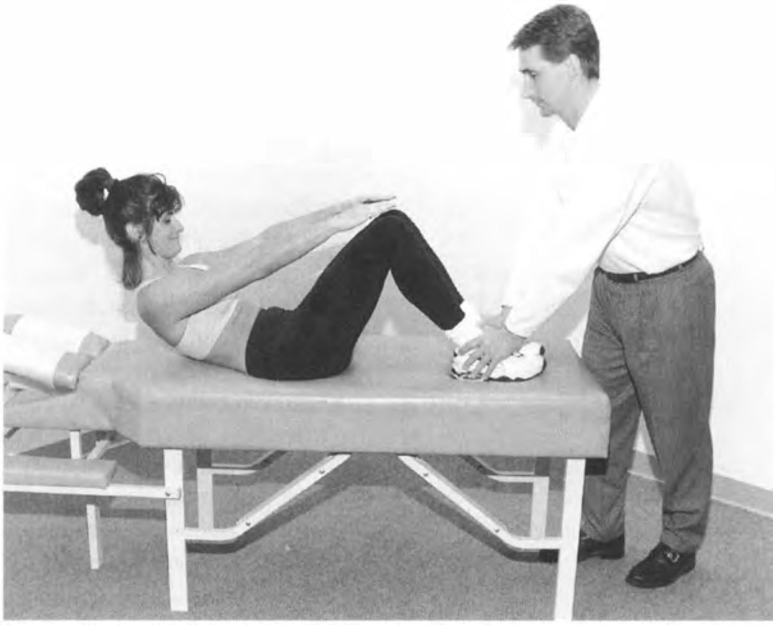

Figure 15.7. Repetitive trunk curl correct test mechanics.

Figure 15.8. Sorenson's test correct test mechanics.

Figure 15.9. Single leg stance correct test mechanics (also with eyes closed).

terior to posterior sway and tend to perform poorly in this test (77). Although practical direct measurement of lumbar spine proprioception is still relegated to the laboratory, measurements of single leg standing balance can be useful in identifying coordination and balance deficits (77, 89, 90).

Test mechanics: The patient is barefoot with arms at the sides. The patient is asked to flex the hip to 45° and knee to 90°. Observe for pelvic unleveling (weak gluteus medius), excessive arm movement, trunk sway, or foot touch down indicating poor proprioception and coordination. Patient should be able to maintain single leg position for at least 20 seconds with eyes closed. Closing eyes removes the visual system and imposes greater demand on the vestibular, cerebellar and peripheral systems.

Qualifiable Tests

The qualifiable tests are based on the work of Janda and Jull (9, 41, 69). The movement patterns in these tests allow for evaluation of the patient during commonly performed movements during daily activities such as lifting and ambulating. These tests relate well to the quantifiable indicators but add another dimension to the clinical and functional picture. These tests include postural evaluation, movement pattern assessment, and gait analysis.

Postural Evaluation

Postural analysis is a static representation of movement. Assessment of static posture can be predictive of muscular imbalance and impairment of the locomotor system. The evaluator should strive to identify obvious muscular imbalance or asymmetry. Table 15.4 outlines key relationships between postural faults and functional pathology.

Movement Pattern Assessment (9, 41, 69)

Analysis of movement patterns provides useful clinical information above and beyond traditional muscle strength testing procedures. The assessment of kinesiopathology is diagnostic for muscular imbalances, and it provides information that guides therapeutic and rehabilitative decisions. Three main objectives emerge when analyzing a particular movement: (*a*) determine where the movement is occurring, (*b*) assess the quality of the movement, and (*c*) assess the range or quantity of the movement. The following tests outline how to perform and interpret the tests.

Hip Extension (41) (Fig. 15.10)

Test goal: Evaluate gluteus maximus contraction to produce hip extension. This is related to the propulsion phase of gait.

Test rationale: The gluteus maximus is the primary mover in hip extension. The ipsilateral hamstring is a strong movement synergist with the hip joint being the focal point of the motion. Poor hip motion leads to compensatory lumbosacral hyperextension. This can be observed in gait during toe off. Inhibition of gluteus maximus may be caused by hip or sacroiliac joint pathomechanics or overactivity of the antagonistic musculature (iliopsoas, rectus femoris). Weakness of the gluteus maximus is common in patients with sedentary lifestyles.

Test mechanics: The patient is prone and is asked to extend the hip. Observe for hip joint and lumbosacral mechanics. The bulk of the movement should occur at the hip, although slight lumbosacral extension may occur at end range. Excessive thoracolumbar paraspinal activity, lumbar lordosis, trunk rotation, or shoulder girdle activity indicate a compensatory pattern of motion. Some paraspinal muscle activation may be observed when hip extension is initiated. It has recently been shown that paraspinal muscle activity is likely caused by a coactivation stabilizing effect (40).

Hip Abduction (41) (Fig. 15.11)

Test goal: Evaluate gluteus medius during hip abduction. This relates to stabilization of the pelvis during the midstance phase of gait.

Test rationale: Gluteus medius is active during midstance to stabilize the pelvis. Pelvic stability is accomplished by the stance leg gluteus medius, which allows smooth transition and absorption of ground reaction forces from the lower extremities. Inability of the gluteus medius to maximally contract may be caused by weakness, pathomechanics of the sacroiliac and hip joints, or overactivity of the hip adductors. The synergists of hip abduction are the quadratus lumborum (QL) and tensor fasciae latae (TFL). Dysfunction of the QL is frequently identified in low back pain patients (91). Faulty gluteus medius stabilization may be an underlying promulgator of QL overactivity and dysfunction.

Test mechanics: Patient is side posture. The top leg is positioned with slight internal rotation. The patient abducts the top leg to approximately 45°. Trunk rotation, hip hiking, and hip flexion are the most common compensatory patterns seen. Posterior trunk rotation indicates paraspinal hyperactivity. Hip hiking indicates QL substitution. Overactivity of

Table 15.4

Postural Analysis and Associated Functional Pathology

Observation	Predicted Functional Pathology
Posterior View	
Pelvis	
Lateral shift	Weak gluteus medius on contralateral side
Anterior pelvic tilt	Shortened iliopsoas, rectus femoris (deep, short lordosis); shortened erector spinae (shallow, long lordosis); weak gluteus maximus and abdominals
Posterior pelvic tilt	Shortened hamstrings
Unleveling	Shortened quadratus lumborum
Flattened buttocks	Weak gluteals
Lower extremities	
Proximal adductor notch	Shortened hip adductors
Hamstring prominence	Shortened hamstrings
Broad Achilles' tendon	Shortened gastrocsoleus
Cylindrically shaped calf	Shortened gastrocsoleus
Back	
Thoracolumbar erector spinae hypertrophy	Lack of hip extension and poor lumbar stability
Anterior View	
Abdominals	
"Love handles"	Weak transversus abdominus
Lateral abdominal groove	Shortened ipsilateral external obliques and poor lumbar stability in anterior to posterior direction
Lower extremities	
Lateral patellar notch	Shortened ipsilateral rectus femoris
Patella alta	Shortened ipsilateral rectus femoris
Lateral thigh groove	Shortened ipsilateral tensor fascia latae/iliotibial band
Laterally deviated patella	Shortened ipsilateral tensor fascia latae/iliotibial band
External rotation	Shortened ipsilateral piriformis

Weak = inhibited, shortened = tight.

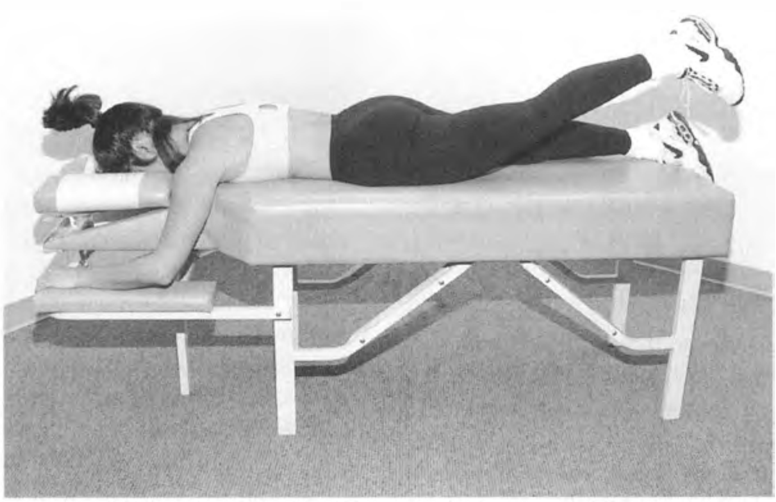

Figure 15.10. Hip extension correct test mechanics.

Figure 15.11. Hip abduction correct test mechanics.

Figure 15.12. Trunk curl (without stabilizing feet, and knees slightly flexed).

the iliopsoas produces excessive hip flexion. TFL substitution often elicits hip flexion with external rotation. Poor abduction range can often be traced to tightness of the thigh adductors or gluteus medius inhibition. Hip joint dysfunction should also be considered as a primary source of poor movement.

Trunk Curl (41) (Fig. 15.12)

Test goal: Assessment of rectus abdominus and iliopsoas in producing trunk flexion.

Test rationale: Rectus abdominus is the primary mover in trunk flexion with the iliopsoas acting as a powerful synergist (15, 34, 41, 51).

Test mechanics: The patient is positioned supine with knees flexed slightly and feet flat and is asked to curl up slowly until the inferior scapular angle clears the table. Lumbosacral hyperextension indicates poor stability, overactive erector spinae, and poor abdominal control. Lifting feet off the table demonstrates iliopsoas hyperactivity. Poor abdominal recruitment is indicated by shaking of the body during movement. Abdominal protrusion is correlated with lack of transversus abdominus and multifidi co-contraction (42, 51).

Squatting (41) (Fig. 15.6)

Test goal: To determine squat mechanics and quality of motion.

Test rationale: Poor quadricep strength and endurance correlates with inadequate lifting mechanics and poor balance control (84, 86). Maintenance of neutral lordosis during lifting limits shear forces across the intervertebral disc (85).

Test mechanics: The patient stands with feet shoulder width apart and squats until thighs are parallel with floor. Note loss of lordotic posture and inability to keep heels on the ground. Loss of lumbar lordosis with trunk flexion may be indicative of overactive hamstrings or poor intersegmental mechanics. Anterior translation of the knee over the foot, varus or valgus sway of the knee is attributed to poor quadricep control. Heel rise is from shortened gastrocsoleus complex or poor ankle mortice dorsiflexion.

EXERCISE PRESCRIPTION FOR LUMBAR STABILIZATION

The treatment continuum for functional reactivation and rehabilitation centers on the correction of dysfunctional joint mechanics, beginning with mobilization and manipulative techniques. Next, reducing hypertonicity in overactive muscular patterns is necessary. Finally, specific exercises are used to facilitate hypoactive and weakened muscle groups. These exercises include stabilization and sensory motor routines. See Table 15.5 for information on the treatment continuum used in rehabilitation of low back disorders (92).

Manipulation

The rationale, indication, and clinical effects of manipulation are beyond the scope of this chapter. Lewit has heralded the key role of manipulation in the rehabilitative process (5). He has compiled correlations between joint and muscular dysfunction that add to our understanding of spinal muscular reflexes. An understanding of these relationships is a useful adjunct to determining the application of manipulation. The articulations and muscles most germane to lower back pain syndromes are summarized in Table 15.6.

Table 15.6

Lewit's Functional Chains: Muscle and Joint Relationships

Spinal Level	Muscular Relationship
T10–L2	QL, psoas, abdominals, T/LE/S
L2–L3	Gluteus medius
L3–L4	Rectus femoris, L/SE/S, hip adductors
L4–L5	Piriformis, hamstrings, L/SE/S, hip adductors
L5–S1	Iliacus, hamstrings, L/SE/S, hip adductors
Sacroiliac joint	Gluteus maximus, piriformis, iliacus, hamstrings, hip adductors, contralateral gluteus medius
Coccyx	Levator ani, gluteus maximus, piriformis, iliacus
Hip	Hip adductors

Modified with permission from Lewit K. Manipulative Therapy in the Rehabilitation of the Motor System. Boston: Butterworths, 1985.

Myofascial Release and Flexibility Training

Manual resistance techniques are primarily used to reduce muscular hypertonicity. These techniques are based on the concepts of proprioceptive neuromuscular facilitation (PNF) and involve light muscular contractions in positions of mild stretch. Following the contraction, a period of muscular relaxation occurs and the muscle can be relaxed to a new resting length. Postisometric relaxation (PIR) is a gentle muscular release technique. All techniques require comfortable patient positioning. The muscle(s) is placed on slight tension at the point of its physiologic barrier to further movement. A mild contraction of the muscle is facilitated via verbal and tactile commands. Contractions are typically held for 10 seconds at which time the patient is instructed to relax the contraction. The muscle is allowed to relax and then gently elongate to its new resting length. Three to four repetitions are typically required. Self-release techniques can then be employed to maintain treatment gains. Liebenson's text contains a detailed description of myofascial release techniques (93). See Figure 15.13 for PIR and self-releases for the psoas, hamstring, and piriformis muscles.

Table 15.5

Treatment Continuum in Rehabilitation of Low Back Disorders

1. Manipulate/mobilize stiff joints
2. Relax / lengthen tight myofascial stuctures
3. Train motor control of spinae, pelvis, and lower extremity
4. Train strength of large prime mover muscles (quadriceps, gluteals, rectus abdominis)

Modified with permission from Liebenson C, Hyman J, Gluck N, et al. Spinal stabilization. Top Clin Chiro 1996;3(3):63.

Spinal Stabilization Training (12, 94)

The overall goal of the spinal stabilization program is to recondition key spinal stabilizers while improving control and coordination. A distinct advantage to this exercise approach is that it provides a training effect for target muscle groups without aggravating symptoms (15, 12). The focus is on producing quality movement. This type of exercising provides several training possibilities in a number of positions that correlate to functional deficits noted during the physical examination.

Goals of the initial phase are to explore lumbopelvic movement, identify the functional training range, and attain initial lumbopelvic control through basic skills such as muscular cocontraction and pelvic tilting. These skills introduce the patient to the notion that movement is good and that by performing

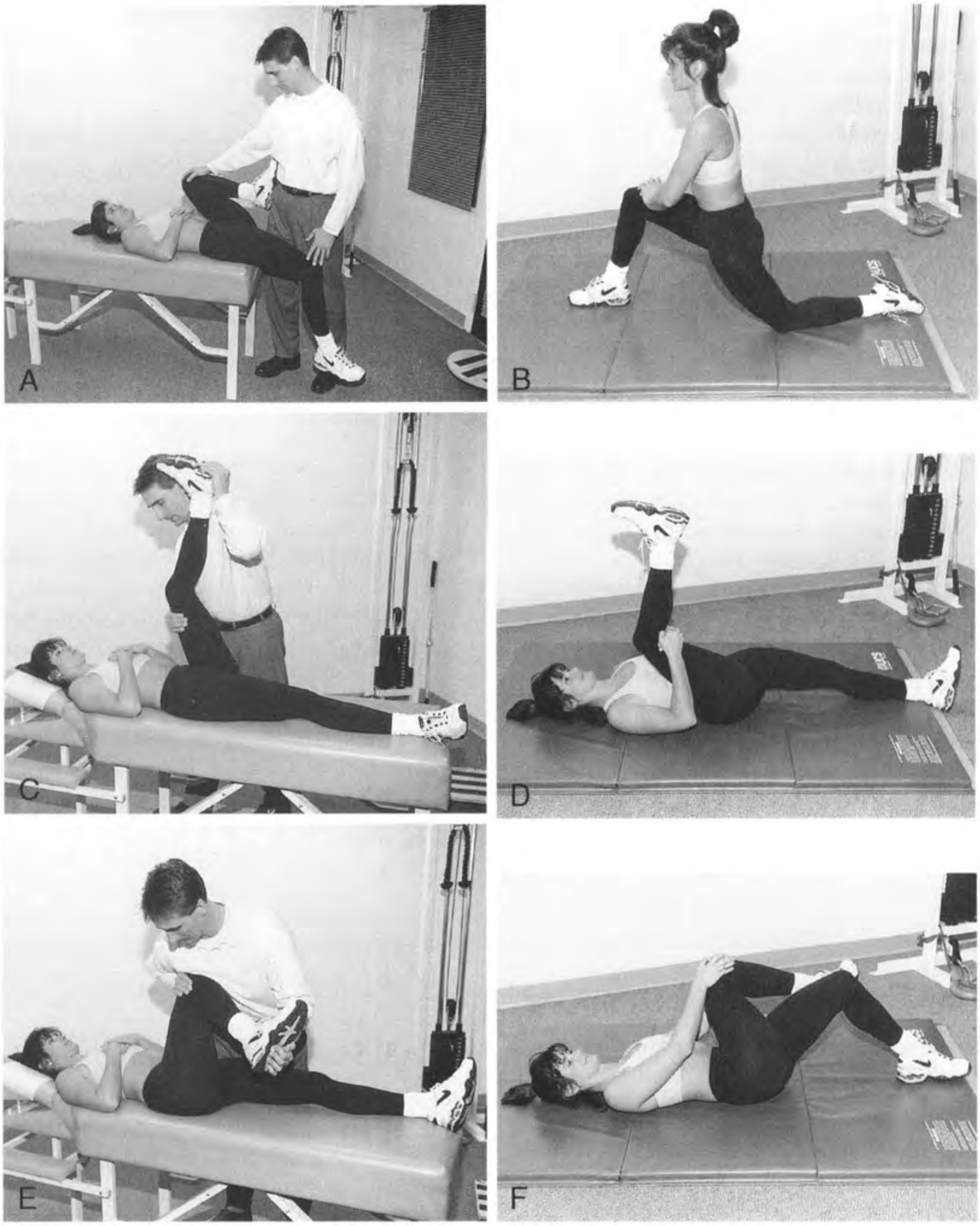

Figure 15.13. **A.** Psoas postisometric relaxation (PIR). **B.** Psoas self-stretch. **C.** Hamstring PIR. **D.** Hamstring self-stretch. **E.** Piriformis PIR. **F.** Piriformis self-stretch.

certain movements, painful episodes can be controlled and reduced. Some initial-phase exercises are safe to begin in the late acute or early subacute LBP patient, even those patients with radicular symptoms. In the acute care phase identification of movements that control pain, centralize extremity pain toward the spine, and allow self-mobilization are emphasized. Empowering patients with effective self-care tools in the early stages of management speeds the recovery process and limits chronicity. The three main features of our lumbar spine rehabilitation prescription are abdominal co-contraction, lumbopelvic control, and fast coordinated muscular activation.

Abdominal Co-Contraction: Hollowing and Bracing

Richardson stated: "It is inappropriate to load the spine when a basic level of active protective stability cannot be achieved" (95). Enhancing stability can be accomplished through facilitating co-contraction ability. Co-activating the abdominal wall and the deep back extensors is the initial goal in achieving basic stability and kinesthetic awareness of the lumbopelvic region. Co-contraction movements activate important stabilizing muscles such as the multifidus and transversus abdominus. Begin by asking the patient to draw the navel toward the spine (Fig. 15.14). This action activates the abdominals and multifidus muscles, resulting in co-contraction. The use of the oblique abdominals and transversus abdominus with minimal contribution from the rectus abdominus requires the action of pulling the abdomen inward toward the spine and slightly upward without deep inspiration. With efficient coactivation a small muscular prominence will be apparent when the transversus and oblique abdominals are active in the co-contraction. This can be palpated just medial to the ASIS (36). If the patient is experiencing difficulty with this maneuver, attempt coordinating the hollowing with forced exhalation. Exhalation facilitates the lower abdominal muscles to contract, causing the lower abdomen to flatten. The abdomen should expand with inhalation and flatten with exhalation. Abdominal bracing is another way to improve lumbar stabilization but this is chiefly accomplished by increasing intra-abdominal pressure in conjunction with a mild co-contraction. The next step is to explore lumbopelvic motion through pelvic tilting.

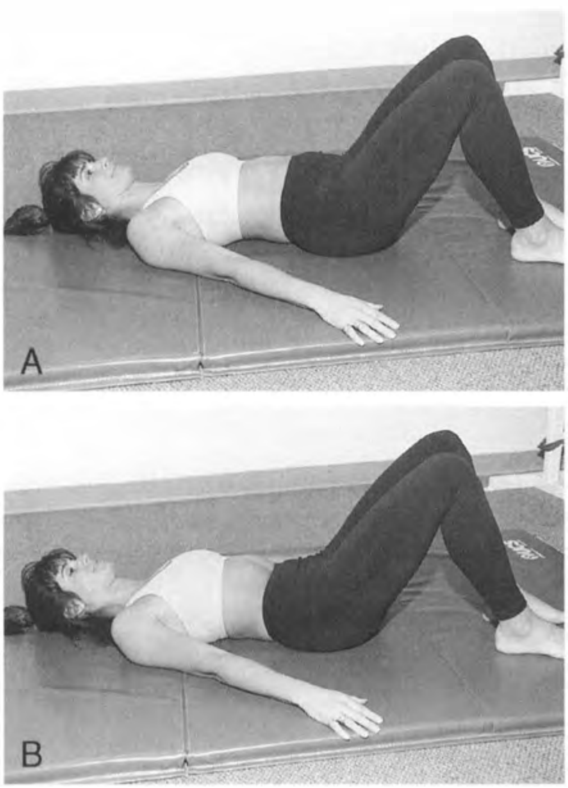

Figure 15.14. **A.** Abdominal breathing showing lower abdominal protrusion. **B.** Abdominal hollow.

Lumbopelvic Control: Pelvic Tilting

The basic purpose of pelvic tilting is to initiate movement, gain kinesthetic awareness of lumbopelvic control, and explore the patient's lumbopelvic range. Initially, one or two positions will be successful for the patient. Six basic positions are usually explored: supine hook-lying, quadruped, sitting, standing, lunge, and kneeling. Any or all of the positions can be used, but those positions most representative of the patient's functional intolerances should be emphasized. For example, patients with radiculopathy typically find sitting to be most provocative. Improving sitting tolerance provides the patient with a management tool to avoid provocation and minimize symptom exacerbation. A combination of verbal and tactile cues tends to offer the best result when facilitating the patient to perform the desired movement. Ask the patient to tilt the hips backward and forward. Place one hand on the lower abdomen and the other on the lower lumbar spine and gluteals to assist the movement (Fig. 15.15).

Fast Coordinated Muscular Activation: Sensory Motor Stimulation

Sensory motor stimulation (SMS) is a simple, yet effective training method to improve postural reflexes (9, 41, 69, 96). The goals of SMS training can be achieved by using tools such as rocker boards, balance sandals, and physioballs. These training goals are summarized in Table 15.7. Postural reflexes are improved by increasing the stimuli from peripheral structures (i.e., skin, muscles, and lower extremity and spinal joints) to elicit increased activity of the postural stabilizing muscles.

Sensory motor stimulation has been most commonly applied in the rehabilitation of lower extremity sports injury. Gains in local muscular strength as well as improved balance performance have been documented following training on rocker and wobble boards (100–103). A small number of asymptomatic patients were studied to determine the effect of using balance sandals on the gluteal musculature. Great increases in speed of contraction of the gluteal muscles were documented while using the sandals. The treatment effect carried over into barefoot walking as the patients continued to demonstrate and maintain dramatic contraction speed of the gluteal muscles (96). Several key stabilization and sensory motor tracks will be discussed below. In several stabilization exercise

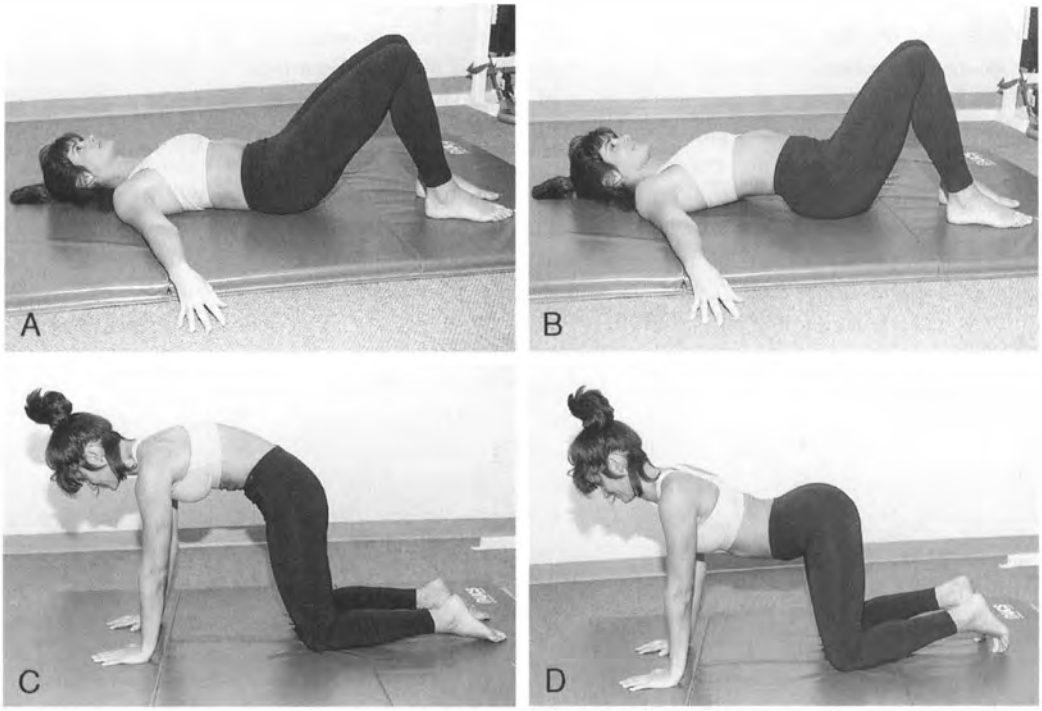

Figure 15.15. **A.** Posterior pelvic tilt—supine hook lying. **B.** Anterior pelvic tilt—supine hook lying. **C.** Posterior pelvic tilt—quadruped. **D.** Anterior pelvic tilt—quadruped.

Table 15.7

Sensory Motor Stimulation Goals

1. Retrain altered afferent pathways
2. Enhance sensation of joint movement
3. Enhance reflex stabilization
4. Achieve automatized control of postural muscles

Based on references 97–99.

Table 15.8

Common Errors and Corrections During Stabilization Training

Error	Corrections
Hyperlordosis	Neutral lumbar lordosis
Abdominal protrusion	Abdominal hollowing
Poor lower extremity alignment	Specific corrections for specific exercises
Chin poking	Neutral cervical spine
Pelvic unleveling	Level pelvis
Valsalva maneuver	Exhale on exertion

examples, we have incorporated the use of the physioball. Liebenson's text has an excellent description of other floor and physioball routines (94).

Exercise Prescription

Exercise protocols are developed to be used as guidelines. Attention must be paid to the patient's individual tolerances, activities of daily living, and demands of employment or sport. The doctor and patient must be familiar with common errors that must be corrected. These corrections have been outlined in Table 15.8. Neutral lumbar lordosis refers to the midpoint between the posterior and anterior pelvic tilt. This is considered a safe point to begin most stabilization exercises. The spinal joints are near the midrange where the likelihood for joint stress is negligible (40). Abdominal hollowing, which is performed in conjunction with the neutral spine posture, facilitates muscular stabilization. Lower extremity alignment differs with specific exercises. For example, when squatting, the knee should not pass the forefoot nor should there be valgus or varus sway. During supine bridge exercises the knees should be hip width apart except for single leg versions.

Exercise Tracks

Stabilization exercises are organized in tracks to progress the patient through increasingly difficult exercises. Isometric holds with co-contraction are performed first. By adding extremity

movement, resistance, and hold times, exercise difficulty increases. The number of sets and repetitions is determined by the patient's ability to correctly perform the exercise. Progressing a patient to the next level is based on ability to demonstrate good postural control and coordination. Always allow the patient to succeed with an exercise. If it cannot be performed appropriately, make the exercise easier by decreasing the complexity of the movement (i.e., decreasing repetitions and resistance). This is the concept of "peeling back." For example, a patient unable to perform a bridge exercise on a physioball may need to be "peeled back" to bridging on the floor. Table 15.9 outlines the rules for exercise progression. An isometric contraction on a stable surface is the safest and least demanding form of an exercise.

The exercises within these tracks are a condensed compilation of frequently used exercises. Progression within the track is in increasing order of difficulty. Any of these exercises can be made more difficult with the use of longer hold times, manual resistance, and unstable surfaces.

Table 15.9

Rules for Exercise Progression

Unloaded to loaded
Simple to complex
Stable to labile
Isometric > concentric > eccentric > resistance
Endurance > strength
Slow > fast movements

Modified with permission from Liebenson C, Hyman J, Gluck N, et al. Spinal stabilization. Topics Clinical Chiropractic 1996;3(3):67

Bridge Track: Floor and Physioball Routines (Fig. 15.16)

Indication: '+' hip extension and hip abduction tests. This track emphasizes gluteal and quadricep recruitment. The patient first explores lumbopelvic motion, finds neutral lumbar spine, and hollows the abdomen. Next, active gluteal contraction occurs as the patient raises the pelvis to the maximal height where neutral spine can be maintained. Maximal recruitment of the erector spinae, gluteals, and transversus abdominus muscles occurs when manual resistance is applied to the pelvis over the anterior superior iliac spine in conjunction with slight alternating pelvic rotation movements. (55) This can be incorporated into any of these bridge variations.

1. Floor bridge (Fig. 15.16*A*)
2. Ball bridge (Fig. 15.16*B*)
3. Floor bridge with marching (Fig.15.16*C*)
4. Ball bridge with single leg (Fig.15.16*D*)
5. Bridge with feet on ball (Fig.15.16*E*)
6. Hamstring bridge with feet on the ball (Fig.15.16*F*)

Dead Bug Track: Floor and Physioball Routine (Fig. 15.17)

Indication: '+' trunk curl or repetitive curl up tests. This track trains the abdominals with specific emphasis on maintaining spinal stability while incorporating extremity movement. The beginning position is supine with knees flexed and arms at the

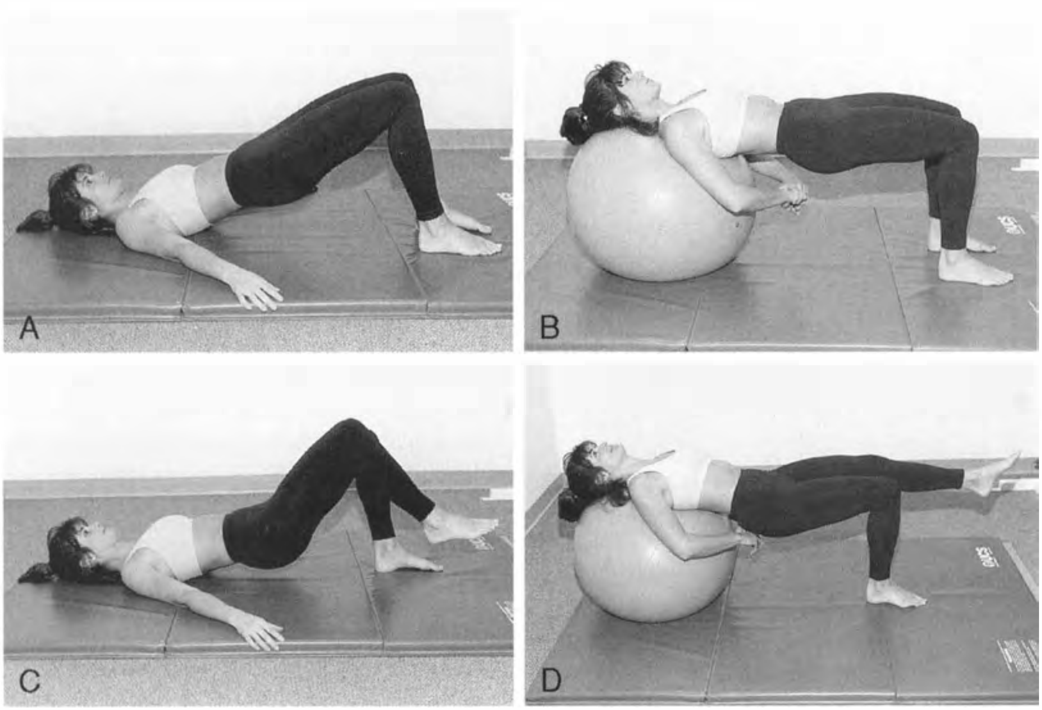

Figure 15.16. A. Floor bridge. **B.** Ball bridge. **C.** Floor bridge with marching. **D.** Ball bridge with single leg.

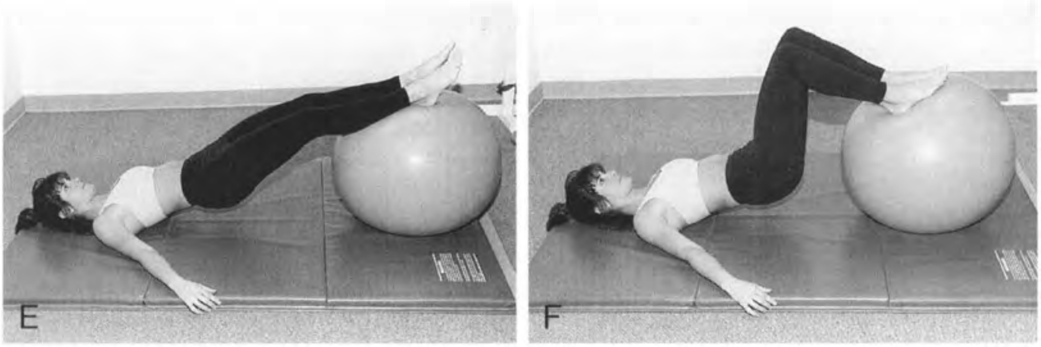

Figure 15.16—*continued.* **E.** Bridge with feet on ball. **F.** Hamstring bridge with feet on ball.

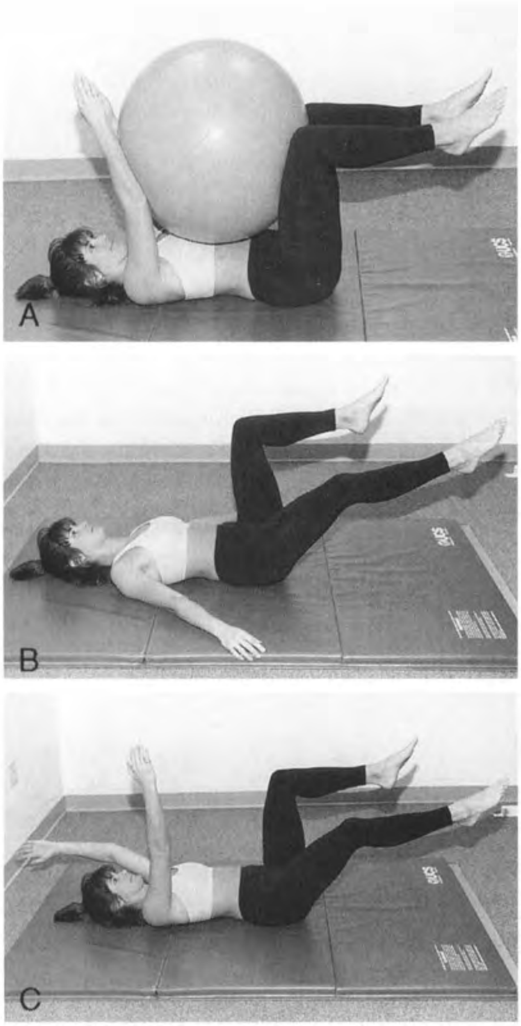

Figure 15.17. **A.** Isometric ball squeeze. **B.** Alternate leg movement with stable trunk. **C.** Combined alternate arm and leg movements with stable trunk.

side. Extending arms overhead has a tendency to increase lumbar lordosis. Be aware of this and make the appropriate corrections. This exercise incorporates progression to both arm and leg movements in an opposite and alternating motion. The initial movement of using the upper extremity is not shown. Once the lower extremity motion is combined with the upper extremity movement, coordinated function and spinal stability is difficult to maintain. The diagonal isometric hold emphasizes oblique abdominal training.

1. Isometric ball squeeze (Fig. 15.17*A*)
2. Alternate leg movement with stable trunk (Fig.15.17*B*)
3. Combined alternate arm and leg movement with stable trunk (Fig. 15.17*C*)

Quadruped Track: Floor Routine (Fig. 15.18)

Indication: '+' hip extension and Sorenson's tests. Similar to the 'Dead Bug' position, the quadruped position also incorporates arm and leg movements. However, the training effect emphasizes multifidus, gluteal, and transversus abdominus muscles. In the quadruped position the hands are directly under the shoulders and the knees under the hips. A stable and neutral cervical and lumbar spine is maintained while the extremity movement is performed.

1. Alternate leg movement with stable trunk (Fig. 15.18*A*)
2. Opposite arm and leg (cross crawl) with stable trunk (Fig. 15.18*B*)

Supine Track: Physioball Routines (Fig. 15.19)

Indication: '+' trunk curl or repetitive curl up tests. These exercises are modified abdominal crunches that focus on rectus abdominus, oblique abdominals, and transversus abdominus muscles. The patient begins in the supine hook-lying position with feet on the ground and hips and knees moderately flexed. The arms can be in several positions in increasing order of difficulty: (*a*) reaching forward, (*b*) crossed over chest, (*c*) crossed behind the head, supporting the neck, and (*d*) elbows extended over head. The concentric phase of the crunch is complete when the inferior angle of the scapulae just lift off the floor or the ball. Remember, the lower abdominals should flatten when the crunch is performed with a neutral lumbar spine emphasized. The combination of the center and oblique abdominal crunch is most effective for training all the abdominals.

Figure 15.18. **A.** Alternate leg movement with stable trunk. **B.** Opposite arm and leg (cross crawl) with stable trunk.

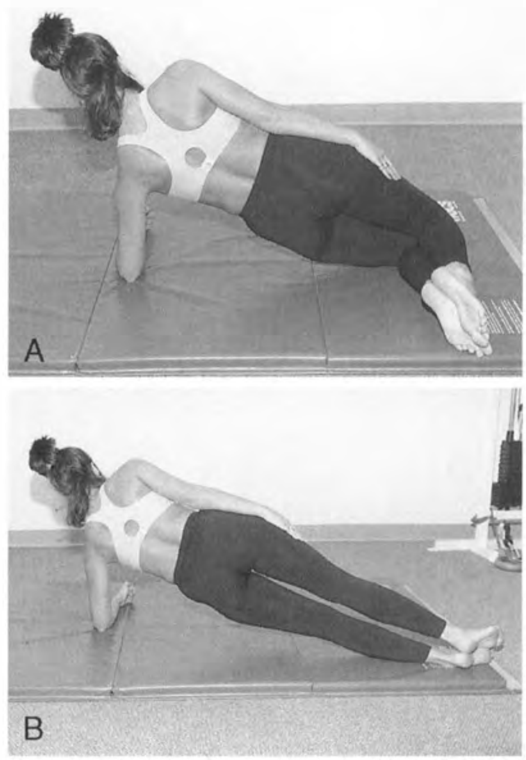

Figure 15.20. **A.** Side-support with knees flexed to 90°. **B.** Side-support with knees fully extended.

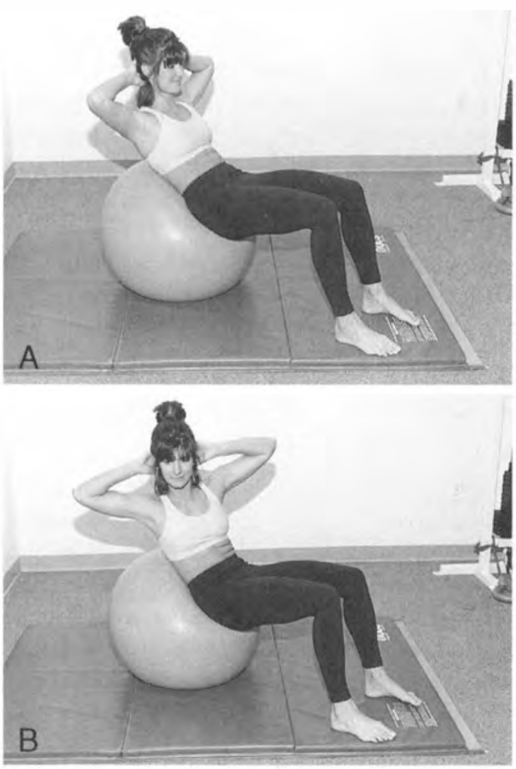

Figure 15.19. **A.** Center crunch—ball. **B.** Oblique crunch—ball.

1. Center crunch— ball (Fig. 15.19*A*)
2. Oblique crunch—ball (Fig. 15.19*B*)

Side-Support Track: Floor Routine (Fig. 15.20)

Indication: '+' hip abduction test. This routine effectively trains the quadratus lumborum muscle (55). The difference between the two forms is knee position. When the knees are extended, exercise difficulty increases. The key technique points are (*a*) maintain the hips in slight extension and (*b*) shoulders, knees, and ankles (in the second exercise) form a straight line. The elbow is flexed at 90° and directly positioned under the shoulder. The pelvis is then raised until the spine is straight. The downside quadratus lumborum muscle is being trained.

1. Side-support with knees flexed to 90° (Fig. 15.20*A*)
2. Side-support with knees fully extended (Fig. 15.20*B*)

Prone Track: Physioball Routine (Fig. 15.21)

Indication: '+' Sorenson's test. This track emphasizes the deep and superficial spinal extensor muscles. The patient begins with feet against a wall for support, knees flexed, and touching the floor, and then leans over the ball and extends the legs. As the ball rolls forward, the patient will begin to straighten the spine to neutral, not hyperextension. In the first exercise the focus lies in training static endurance of the multifidus muscle, using increased hold times. This exercise can be performed with various arms positions. The further the extremities are away from

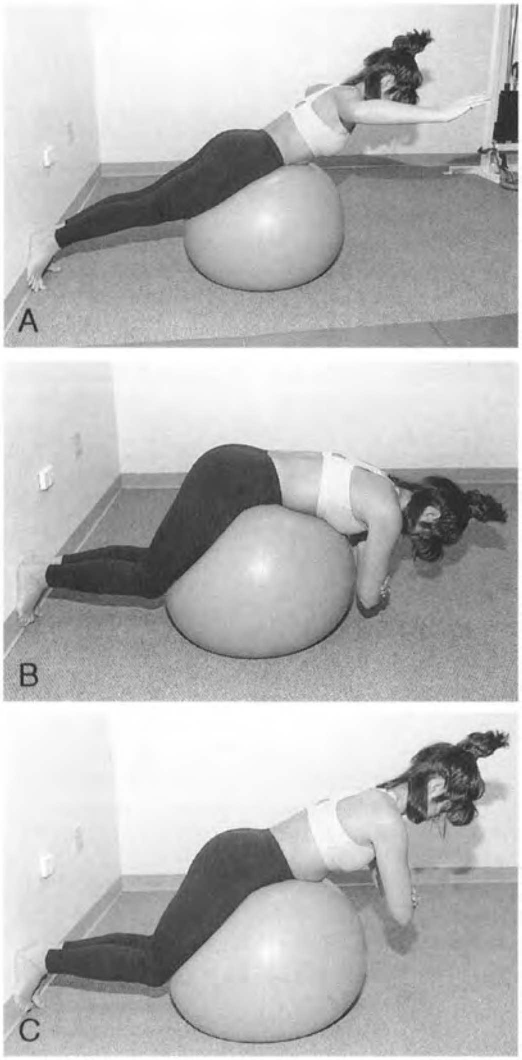

Figure 15.21. **A.** Superman over ball with arms at 90°. **B.** Repetitive spine extensions over ball—starting position. **C.** Repetitive spine extensions over ball—final position.

the body the more demanding the exercise. First begin with the arms by the side then progress the arms to 120° abduction. The second exercise concentrates more on phasic control with the incorporation of repetitions to train the superficial erector spinae. In the second exercise, the arm position is crossed hanging in front of the chest.

1. Superman over ball (shown with arms 90° abducted) (Fig. 15.21*A*)
2. Repetitive spine extensions over ball—starting position (Fig. 15.21*B*)
3. Repetitive spine extensions over ball—ending position (Fig. 15.21*C*)

Standing Track: Physioball Routine (Fig. 15.22)

Indication: '+' single leg stance and repetitive squat test. This routine will train the quadriceps and gluteals while teaching the patient to maintain lumbar lordosis during squatting and lifting tasks. The physioball is placed in the small of the back. Feet are hip width apart with the body leaning into the ball. This leaning posture helps facilitate proper lower extremity alignment. When performing the squat, patients are cued to press the buttocks into the ball as they lower their body. This will emphasize maintaining lumbar lordosis during lifting and squatting tasks. Initially, a shallow squat may be most appropriate. As the patient progresses to a full squat, the thighs should be parallel with the floor. The side squat variation emphasizes the gluteus medius. First, the pelvis is slightly dropped on the inside leg. The patient begins by pressing the pelvis into the ball. This is followed by leveling the pelvis to complete the movement, which is necessary to maximally recruit the gluteus medius on the stance side. The final standing exercise is the lunge. The lunge is performed without the ball in either a repetitive static position or with dynamic alternating repetitions. Lower extremity alignment is crucial. The forward leg knee and ankle are aligned as are the back leg, hip, and knee. The forward step should be hip width apart.

1. Full ball squat (Fig. 15.22*A*)
2. Single leg ball squat (Fig. 15.22*B*)
3. Side ball squat (Fig. 15.22*C*)
4. Static lunge (Fig. 15.22*D*)

Sensory Motor Stimulation Track (98, 99)

Indication: Malcoordination observed during functional testing. The use of rocker boards, wobble boards, balance sandals, foam rollers, and physioballs enhances sensory motor control and coordination (Fig. 15.23). General rules to be observed include maintenance of posture and alignment. One strategy for integrating SMS exercises into a lumbar stabilization program is to use sensory motor devices between each exercise activity. For example, stationary bike warm up, rocker board, abdominal exercises, wobble board, extension exercises, and balance sandals. Exercises on the balance boards typically are maintained for 20 to 30 seconds per repetition and repeated frequently.

Foot prepositioning may be necessary at first to facilitate the patient's lower extremity kinesthetic awareness. This can be accomplished first by stroking the sole of the foot, then by approximating the rear and forefoot to slightly raise the longitudinal arch. This is termed the "short" or "small foot." The patient is instructed to gently grip the toes, while contracting the intrinsic muscles of the longitudinal arch, without lifting the first metatarsal head. (Fig. 15.24) The patient should feel most of the weightbearing on the lateral aspect of the foot.

Monitor the patient for several common faults: insufficient stabilization of the metatarsals (excessive plantar flexion), insufficient knee posture (valgus or varus), hyperlordosis of the lumbar spine, or trunk flexion. Maintenance of posture, increased body awareness, and balance control will be enhanced with the use of sensory motor training. Progression to the semisquat and single leg stance follows (Fig 15.9). Single leg stance with slow then fast pushes is thought to enhance activation of spinovestibulocerebellar pathways essential for good

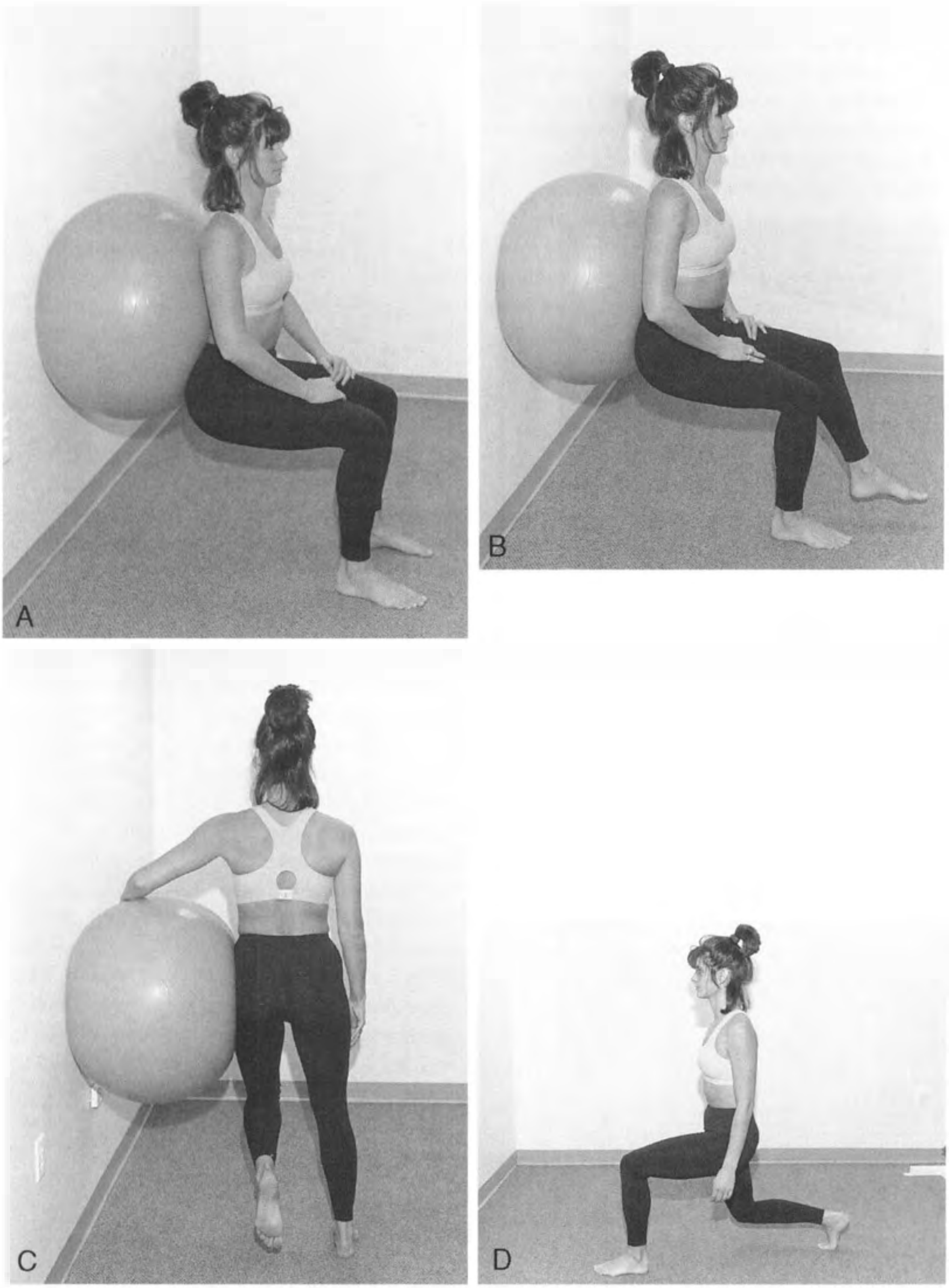

Figure 15.22. **A.** Full ball squat. **B.** Single leg ball squat. **C.** Side ball squat. **D.** Static lunge.

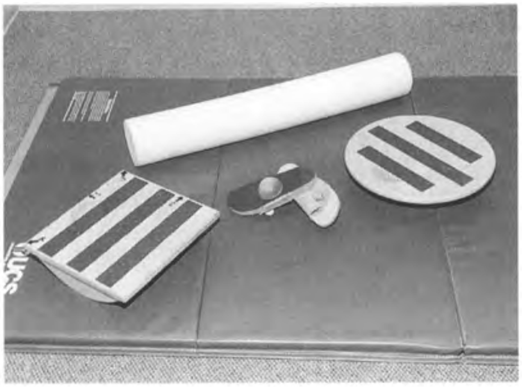

Figure 15.23. Rocker board, wobble board, balance sandals, and foam roller.

Figure 15.24. Short/small foot (forward leg).

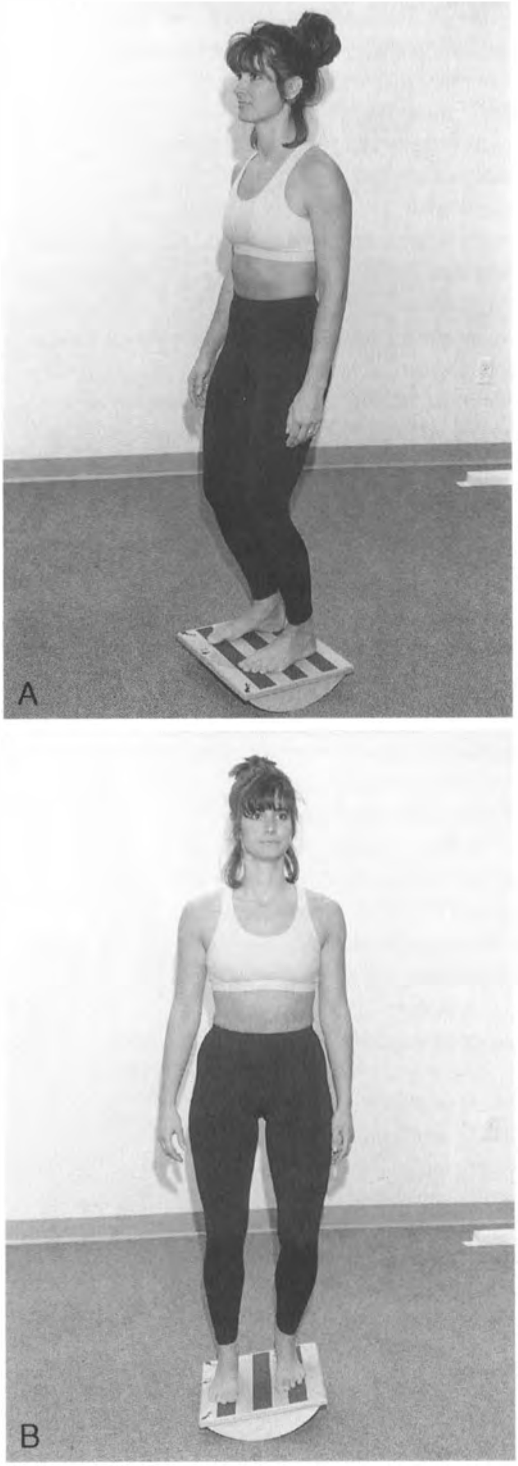

Figure 15.25. A. Rocker board—sagittal position. B. Rocker board—coronal position.

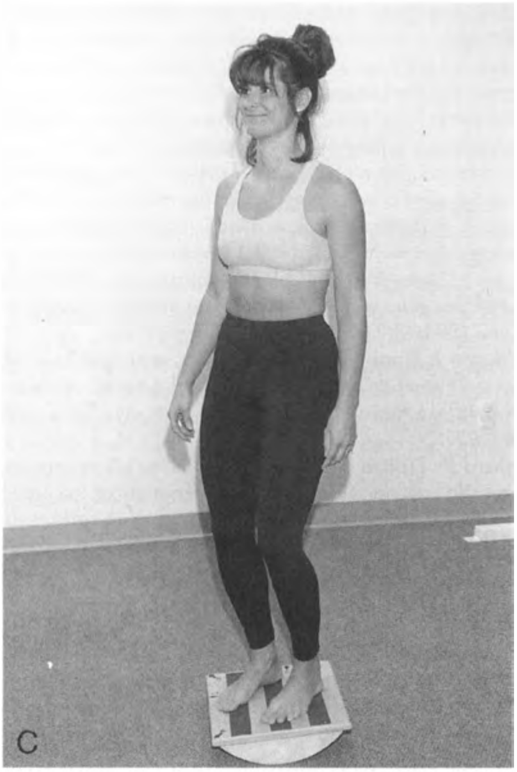

Figure 15.25—*continued.* **C.** Rocker board—diagonal position.

posture and spinal control. The patient's balance is continually challenged by having the patient close the eyes. Closing the eyes eliminates a major source of sensory input required for balance. This intensifies the activity and speed of muscular contraction.

Balance board activities increase the demand for balance by elevating the center of gravity and destabilizing the standing surface. Raising the center of gravity and performing activities on an unstable surface require much greater coordination and muscular effort. The rocker and wobble boards are typically used. The rocker board is unstable in a single plane at a given instance whereas the wobble board is unstable in multiple directions. Changing the foot position on the rocker board trains balance in various angles and emphasizes specific muscles. For example, when the board is rocking from side to side (coronal plane) the gluteus medius is emphasized (Figs. 15.25). The same progression as in standing can be used with the balance boards. Progressing the patient to perform a single leg stance with closed eyes on either balance board is challenging. More advanced exercises on the boards may include stepping, jumping, lunging, and incorporation of upper extremity movements. An example of an advanced activity with a high degree of complexity would be a single leg stance on a wobble board with eyes closed receiving push challenges or using arm movements.

Progression of the patient to functional activities as quickly as possible while maintaining good posture and quality of movement is the ultimate goal. Table 15.10 outlines the general rules for progression. The essence of progression is to challenge the patient by decreasing the stability of the task, but still allowing for patient success.

Also used to improve sensory motor control are balance sandals (Fig. 15.26). These sandals are an open toe and heel sandal with a hemisphere in the center on the plantar surface of the shoe. They are an effective tool because they enhance sensory motor coordination while simulating gait. The usual progression is standing in the shoes next to a chair for support. Marching in place is performed initially. Small half steps with accentuated hip flexion is then incorporated. Maintaining balance and coordination on the sphere is the primary goal. When forward walking is easily performed, the patient progresses to backward walking and side stepping. The common postural fault observed is overactivity of the quadratus lumborum muscle, eliciting a hip hike. Other low technology equipment we

Table 15.10

Progression of Difficulty for Sensory Motor Stimulation Exercises

Initial Level	Progression
Double leg stance	Single leg stance
Standing on floor	Standing on balance apparatus
Eyes open	Eyes closed
No activity	Challenge activity

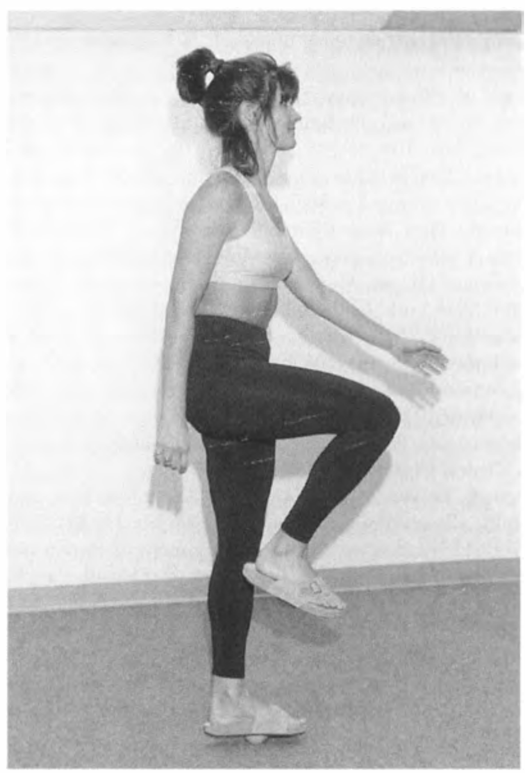

Figure 15.26. Balance sandals.

have used to facilitate proprioception are mini trampolines, balance beams, and the 'Fitter.'

CHALLENGE: SUMMARY

This chapter has reviewed current concepts of exercise as it relates to LBP. A transition from passive to active forms of care is clinically necessary to enhance functional recovery in the LBP patient. By using assessment techniques that both quantify and qualify altered function, simple and effective rehabilitation protocols can be designed for the LBP patient that reduce pain and restore function.

We would like to extend our appreciation to Dr. James Cox for giving us this publishing opportunity. Additionally, our thanks and gratitude are extended to Dr. Craig Liebenson, Dr. George De Franca, Dr. Matthew Kowalski, and Dr. Glenn Dodes for their continued patience, constructive criticism, support, and friendship.

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Psychological Perspectives in Treating Low Back Pain

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chapter 16

The jailor cannot go home, until the prisoner is set free.

—Unknown

Who can deny that the mind and emotional state play an integral part in one's health and well-being? As we learn more and more, that point becomes sharper when dealing with people suffering with low back pain. This chapter is written to briefly bring to light some of the most recent and relevant research data and theories relating low back pain and psychological health.

This area of research has received much more press in the last few years, and for good reason. We know that increased stress and tension can lead to an increased risk of heart attack. No one used to think of these aspects of health until too late. The more we look into how our psyche affects our health and wellness, the less and less we are able to dismiss it as a cause of disease. Yes, the word **cause** is being used here, not correlation, but cause. Psychological health plays a greater role than we understand. For reasons that follow, the number of back injuries has not risen in the last few years but the numbers of those disabled has (1). Why?

The number one somatic complaint of those with depression is low back pain. Why is that? Could it be that support for the body resides in the spine, especially in the low back? When a person's mental health support is lost, support being the spine of the psyche, can the psyche show its pain through the back? The body appears to be a mirror of the person who resides inside as well as the pain, loneliness, suffering, loss, and grief the person carries and needs to heal. Doctors are placed at a particularly advantageous spot to read these signs because of the fact that they have been trained to notice the body, how it is carried, and where its weak points are. Noticing and interpreting these signs and symptoms will be our biggest challenge.

Persistent pain has been found to be predicted by a combination of somatic, psychological, and social parameters (2). The more depressed the mood is before therapy, the more likely the patient avoids social or physical activity (2). As we know, the more control and responsibility the patient takes, the faster the heal and fuller the recovery. Those who avoid physical activity are less likely to heal quickly and more likely to develop chronic pain (2).

How persons communicate their pain is an important clue as to psychological health. Those more depressed are more likely to communicate pain indirectly: moan, grimace in pain, and so forth. Those who communicate indirectly are more likely to have pain at discharge from therapy and may be looking for secondary gain (i.e., more attention from a family member or spouse) (1, 2). By not sharing how one feels directly, a person is expecting others to be psychic and read his or her mind about how depressed, lonely, or despairing that person is. This is a direct path to frustration and failed therapeutic approach. Direct communication is a link to psychological health. One will find only frustration by expecting people to know how one feels.

Fordyce has a theory to explain why depression and other psychological factors can lead to chronic pain: those depressed do not exercise, which leads to deconditioned trunk muscles, which, under a state of physical stress, fail and incur injury (1).

One often-heard statement heard by doctors when suggesting psychological help is "You think this is all in my head?" or "You think I'm making this up?" When a patient communicates pain, then a pain problem is to be dealt with (3). Chronic low back pain patient (CLBPP) issues often become centered on fear of pain, which leads to avoidance and, finally, to disability (3). Others have stated similar ideas. I feel the point to be taken is that when someone expresses pain, it is real. Even if a psychological factor triggers the pain or the pain behavior, if a patient says the pain exists, then it does for that individual, barring that he or she is not malingering. We get nowhere and actually decrease our effectiveness as physicians when we attempt to make patients believe that they do not have pain.

PROFILE OF THE CHRONIC LOW BACK PAIN PATIENT

Let us look at some of the characteristics of those with chronic back pain. The following study represents 78 CLBPPs (4): 10 had psychiatric diagnoses, 34 showed maladaptive personality disorders, 34 had normal prepain personalities, 67 (86%) experienced reactive depression, 54 took higher than normal pain medications, 9 had a drug addiction, 58 misused narcotics, and 54 experienced withdrawal symptoms. In 52 subjects, preoperative imaging (radiograph, magnetic resonance imaging [MRI], computed tomography [CT]) was **normal.** Only 26 had a diagnosis based on radiologic findings that warranted surgery. Clinical criteria were met for intervention in 26 of the 78 cases; 52 were not. Clinical and imaging criteria were met in 40% of second surgeries. Of those who had a second surgery 73% met criteria for a subsequent surgery to treat the effects of an earlier surgery (4)!

As we have seen, many people choose surgery as a quick end to the pain with little to no other clinical criteria for the surgery. Many patients in one study had surgery based on persistent pain that was frequently coupled with underlying psychiatric abnormalities, without meeting the criteria generally accepted before undergoing surgery (4). Surgery, however appropriate in some cases, is not the cure-all that it is thought to be by the general population. It is a last resort, and those undergoing it need to be screened, as will be discussed later.

WHICH COMES FIRST?

Let us begin our study of how psychological disorders begin and how they affect the chronic low back pain patient. Musculoskeletal disorders were found to predate psychological problems in one study. That is to say, the pain came on before depression and psychological distress developed (5). In another study, psychological disturbance appeared equally to be the consequence and the cause of low back pain (6). However, pain diminution for the patient also relieved the psychological distress (6). Another author has found that depression can cause low back pain (7). One author feels that the approach to low back pain needs to be changed from an organic versus psychological argument with a refocus on treating the whole person with a holistic approach (6). This is a typical chiropractic approach, treating the person, not the disease and I do support it with the caveat that identifying the causes and preventing the result is always the most sound approach to wellness care.

A somatizer is someone who manifests emotional and mental disturbances in physical complaints. Essentially, emotional or mental distress is expressed in physical symptoms in these individuals (7). Physical symptom expression of emotions tells the clinician that this person has not the skills to cope with emotional life. In this overcrowded, overstimulated, and under relaxed world we live in, emotional and mental skills to deal with life's stresses are essential. On top of dealing with children, bosses, insurance, bills, and everything else in our daily lives that cause stress, some of us have histories of physical abuse, sexual abuse, and abandonment issues that have never been dealt with. If you have one of these persons as a patient, often the daily stresses of life on top of a past history of psychological trauma is too much to handle and may well be what pushes a person into a chronic pain situation (2). It is suggested that domestic stressors be identified, especially financial difficulties (7). Identification of these patients is important, and clues may be seen as lengthy visits, frequent appointments, multiple phone calls, history of addiction or dependency, or increasing medication requirements (7). The greatest fear low back pain patients carry is that of abandonment (7).

WHAT RISKS LEAD TO CHRONIC LOW BACK PAIN?

A correlation between pain drawings and nonorganic signs has been discovered (8). Nonorganic signs are Burn's bench test, axial compression tests, and other signs that appear from the outside as though they may be used to elicit pain from the patient, but do not. High scores on the nonorganic findings correlate to a non
are ones that are typically nonspecific and cover multiple body areas indicated with general markings.

Socioeconomic factors are clearly implicated in CLBP (2, 9). Three factors have been found that double the risk of chronicity in men and women: a monthly wage of less than $1000, family status of being divorced or widowed with no children, and older than 40 years of age doubles the risk of 25-year-olds (9).

Obesity, smoking, psychological distress, and poor general health also carry a higher risk of low back pain. These relationships may or may not be causal (10). Prevention potential is great if adequate tools for intervention are used (10).

Another risk factor that is showing itself in the research is the concept of congruency. Some LBPPs show clear breaks in the understanding of their conditions and the treatment. Differences, however, exist between congruent and noncongruent patients with respect to certain characteristics and behaviors (11). Congruent patients are ones who understand what goals are realistic and work with the doctor to achieve those goals. Noncongruent patients are those who do not understand the doctor's plan of care and goals and instead have their own goals, which often are unrealistic and typically are never achieved. Low socioeconomic class, compensation claims, use of opiates, increased disability, catastrophizing cognitions (thoughts), stronger emotionality, and passive coping were found to be more characteristic of acute and chronic noncongruent patients (11). In other words, some patients have their own agendas and fail to respond to therapy because they are not following the plan of care. For example, a patient may not notice an increase in range of motion of 60% if still experiencing pain. The patient may fail to see the very real improvement that has been achieved if pain relief is the only goal. This is where patient education is of paramount importance. As a physician, you may relieve sciatic pain for patients and they will still com-

plain of low back pain and say they are no better when asked how they feel. This is true frustration for the clinician.

One of the identified factors associated with psychological problems is not working. In one study, among a blue collar working group, a white collar working group, and a patient group (nonworking), those still working did not show psychological disturbance (12). Disability in working groups was positively linearly related to severity of pain (12). In those with psychological disturbance, the relationship between disability and severity of pain was not linear (12). That may explain why some patients who have minor injuries turn into the disabled patients, whereas those with much more severe injuries are back to work in 3 weeks.

Van Doorn studied veterinarians older than 34 years and dentists older than 44 years with low back pain lasting longer than 14 days. He found that psychosocial issues at the start of the disability were significantly associated with the duration of the disability (13). Those issues tended to predict a longer disability. As a side note of financial interest, nearly 25% of all claims greater than 6 months accounted for 90% of the cost (13).

A risk factor that may go unnoticed is that CLBPPs under laboratory-produced pain situations tended to underpredict pain (14). Not realizing to what extent pain exists, a patient will have a difficult time realizing the damage that he or she may be doing to the back.

A relationship with caffeine has been found as well. CLBPPs patients consume twice as much caffeine as patients without CLBP (15). Tobacco and caffeine use can be associated with CLBP (15, 16). CLBPPs consumed an average 392 mg of caffeine a day, whereas the average consumption of controls was only 149 mg/day (15). However, caffeine consumption was not found to be related to the global experience of pain and disability (17). Interestingly, caffeine users were found more likely to smoke (17). High caffeine use may be embedded in a context of unhealthy behaviors (17) that may be more likely to predispose persons to develop chronic pain. One bad habit may make it easier to develop others. The more research done, the more we may find associations like these. As usual, these types of relationships are only correlated and nothing causal has been determined. Some of these may simply be symptoms showing us the warning characteristics; nonetheless, they are valuable.

Most industrial claims have been found to be caused by minor traumas such as lifting less that 50 pounds (18). The insinuation here is that, when motivated by secondary gain, it may not take much for a person to claim an insignificant injury as a reason for disability. It has also been found in industry that a high incidence of LBP is related to high levels of education and job seniority (19). Interestingly, a low incidence of LBP is found in very religious persons (19). My practice runs contrary to the last statement, showing once again the inaccuracies in science.

Illness behavior is the way an individual perceives, evaluates, and reacts to symptoms. These behaviors operate on a continuum from hypochondriasis to complete denial (20). Abnormal illness behaviors develop out of that continuum, and they have been introduced with the idea in mind that attitudes and behaviors of patients are a more sound way of formulating a diagnosis than physical symptoms alone (20). The most common forms of abnormal illness behaviors (AIB) are hypochondriasis and pain disorder. Dealing with attitudes about the pain is crucial along with reintegrating these people into their lives and removing pain from the spotlight of attention.

A person assuming a sick role typically learns and understands what privileges this role allows. Furthermore, such persons can learn to take advantage of this, whether they feel they are doing it intentionally or not. The sick role grants exemption from certain duties and obligations. These people have feelings of deserving care and are regarded as not responsible for the condition (i.e., they are not malingering) (20). Who grants the sick role? Doctors are principally responsible for granting the sick role (20), which leads to possible preventive measures from the doctor's end. Research further supports a learned association between parents' being treated for LBP and children's reporting of nonspecific LBP (21). The prevalence of LBP in schoolchildren is high at 20% (21). These findings support a learned type response passed from parent to child.

A somatoform disorder is a psychological distress expressed in bodily form or physical complaint. This means of expression spares the person the awareness of unacceptable emotions or mental anguish, temporarily (20). These individuals are doing all they can to avoid having to see a deeper pain. Any attempt by the physician to point out this problem will likely result in anger (20). A physician will expect this, knowing that he or she may be the only one with the insight to point this out to the patient. Anything less is doing the patient a disservice. A physician may be surprised with the results of being the first person to suggest to the patient that more help may be available.

The final goal for these patients is not complete resolution of pain. They typically do not expect that. Rehabilitation with a strong program teaching how to cope successfully and healthily with the pain as it comes and goes is the final goal (20). CLBP is not cured. It is managed and controlled.

Low back pain is also related to becoming overworked and not receiving social support from colleagues (22). Being overworked appears to precede the LBP. Sedentary work and long-distance driving relates to low back pain occurrences (22). We have seen similar results when describing creep in truck drivers.

Two to three percent of LBP patients become chronic, amounting to about 5.2 million Americans, half temporarily and half permanently (23). Estimates of cost are greater than $20 million per year (23). LBP disability from 1960 to 1980 has increased 14 times that of the population (23). In 1957 through the mid-1970s disability for the Social Security disability program increased 347% for all conditions while for back pain it increased 2680% (23). A final thought from that paper was that keeping people at work is good therapy, especially when considering that those off for more than 6 months have about a 20% chance of returning to work, and that people are unlikely to become disabled if they only take short times off work (23). Other studies have shown that return to work of those in subacute situations, even if they still have some pain, is good ther-

apy to reduce disability. Often, simply reassuring these people of a return to work date and staying with it gives them hope, which is no small part of the therapy. Short bouts of pain after the initial incident is normal, and the patient needs to know that it is normal, even expected. When it happens, as is usually inevitable, they will not despair that they will never get back to work or gain relief from the pain.

Life distress, job dissatisfaction, and conflict with an employer can also lead to disability (24). From another author we find that lack of social support and opportunity to speak with the supervisor when problems arise at work does not relate to the experience of LBP (25). Unfortunately, no consensus is found on the issue of control over one's work environment and how it relates to development and progression of LBP.

WHAT PSYCHIATRIC RELATIONSHIPS HAVE BEEN IDENTIFIED?

A study of early psychological trauma and how it correlates with the success of back surgery has been presented (26). The risk factors were history of childhood physical abuse, sexual abuse, emotional abuse, neglect, abandonment, and chemical dependency of a care giver (26). These factors were measured as either present or absent. Of those in the study with three or more risk factors, the success rate was a staggeringly small **15%.** Of those with zero to two risk factors, the success rate jumped to 73%. On the flip side of the risk factors, 19 patients with no risk factors had a success rate of 95%, or failure rate of 5%. Those are good statistics! Of the 31 patients with zero to one risk factors, the success rate was still good at 87%. A final thought is that multiple childhood traumas may predispose one to developing CLBP (26). As is being found with many diseases, more factors are at play than we have instruments to measure. At this point, we can only speculate on the mechanisms of action. I believe that the more sensitive we get in our measurement of certain psychological factors, the more we will see how the psyche affects physical well-being and health and how interventions may be used successfully.

These findings highlight a population of patients that needs to be identified first, followed by exhausting all conservative approaches before surgery is even mentioned. If surgery is finally deemed necessary, one ought be prepared for a lengthy rehabilitation process and necessary psychological counseling to reach maximal improvement.

Goldberg lends further support to the above study by showing that those with a history of childhood physical and sexual abuse had increased depression rates (27). A positive and significant relationship between depression and the abuses was found (27). What is suggested is that a childhood history of physical and sexual abuse may predispose a person to developing chronic pain (27). However, the study does point out that thoughts today about depression point to its being a natural consequence of chronic pain (27) We are not yet able to establish cause and effect; however, childhood abuse and depression are somehow strongly associated with back pain and need addressing.

In one study, 25.8% of CLBP patients reported a lifetime incidence of 12 or more somatic complaints versus 4.1% of controls (28). Fifty one percent of CLBPPs reported seven to 11 symptoms versus 8.2% of controls (28). As can be seen, a significant disparity is found in the patients versus controls, which indicates a significant relationship. Further findings showed that with an increase in severity of somatization (reporting symptoms) major depression and alcohol abuse showed an association (28). Interestingly, pain intensity was not related to an increased number a somatic complaints, but decreased mood and increased impairment were (28). Intensity of pain does not appear to relate to neurosis, whereas the number of complaints does. Once again, pain and disability are not synonymous.

Our discussion would not be complete without exploring some insurance statistics. A Seattle HMO has shown that its most significant reported problem was LBP: 41% of reported claims (29). Pain conditions were found to be chronic and recurrent, mild to moderate in severity, and typically did not limit patients' activities (29). Comparing those with pain versus nonpain conditions, the pain group had higher levels of anxiety and depression, poorer self-rating of health status, more family distress, and more nonpain complaints (29). Nonpain complaints can take the form of depression, stiffness, fatigue, apathy, and so forth. One common theme in patients with psychological components to their condition is the reporting of nonpain complaints. Look for your patients with multiple complaints to be the more difficult ones to treat; they may need some degree of psychological intervention.

One important question to be answered is what happens psychologically for CLBPPs once they leave your office? How do they interact with their families and how does that interaction affect the sick role some have assumed? Some definitions need to be explained. First, a solicitous behavior is one that draws a specific reaction from a person. For example, "Oh, honey, are you in pain again?" The question is sympathy, and someone who needs the attention will respond to it affirmatively. Second, nonverbal pain behaviors can be manifested as facial grimaces, back holding, rubbing an injured limb, or just about anything else that can be done to indicate to others that pain is present without speaking. Spouse solicitious behaviors may precede and follow nonverbal pain behaviors (30). In other words, something like, "Honey are you still in pain," may be followed by rubbing of the back, or vice-versa. On the other hand, nonverbal pain behavior is less likely to follow a spouse's aggressive behavior (30) (i.e., the well spouse gets angry at the ill spouse, and one may then break up the sick role pattern). This study found that in couples, spouse solicitous behaviors preceded and followed nonverbal pain behaviors more than chance would have it (30). To put it briefly, the behaviors of those around the patient, spouses and family members, may unconsciously be maintaining or reinforcing the pain behavior or sick role that person may be assuming. For that reason, it is important to not place blame, simply identify and rectify these patterns within the family system, where applicable.

To identify what type of psychological characteristics are present in chronically ill persons, Sivik studied 41 chronically

ill men and women. The evaluators judged that almost all of the patients were depressive, alexithymic, passive, and antiaggressive, and showed aggressive reaction patterns (31). Certain patients showed much guilt and others, suicidal tendencies (31). Inter-rater reliability was found to be high (31). One main characteristic in these patients was denial of aggression and, as found in other studies, a lack of internal locus of control (31–33). Internal locus of control, as will be discussed later, is the sense that one has some control of his or her life. Depression is a common symptom found in chronically ill people. Those depressed feel differently about social support and the quality of the family environment (34). Social support involves friends, family, or any groups or organizations one may turn to for support, such as AA, ACOA, GamAnon, Codependents anonymous, or any of the popular and helpful support groups in place today. Quality of family environment is also important to one's happiness, or perceived happiness, as is one's sense of well-being and health. As is growing obvious, the factors that contribute to chronicity are many and they interact differently in each patient. No one approach exists that encompasses and explains every situation with which patients may present. Each case requires an individual approach.

After patients have had LBP for a long time and depression has been established to be present, these people tend to use more passive avoidant coping strategies (35). Such strategies of those depressed may include thoughts like, "It will go away" or "I can't do that because it will hurt my back," which either removes responsibility from the patient or justifies a nonaction stance.

One common highly visible problem in our society is marital dissatisfaction. Marital dissatisfaction in female CLBPPs is significantly associated with psychological distress; their husbands also feel the same (36). Interestingly, if the chronic pain patient is the husband, neither he nor his wife will be as dissatisfied as if the chronic pain patient was the wife (36). In yet another example, as current popular psychology has stated recently, the differences between men and women are real and far reaching, and they need to be understood to give the most appropriate care possible. Spousal relationships can affect pain behavior and elicit feelings related to the pain.

Somatization, expressing psychological needs in physical symptoms, is greater in patients with CLBP than their spouses (37). A common feeling in our society is guilt. CLBP appears to be the same. CLBPPs experience more guilt about having pain than do their spouses (37). As we have and will see, research has not been cohesive with its findings on this point. Some studies report that CLBP patients experience more internal locus of control (I have control over what happens to me), whereas their spouses feel more external locus of control (I have no control over what happens to me) (37). Other studies have found the opposite, stating that CLBP patients feel more external locus of control: they feel that their problems are not their responsibility but are caused by external happenings in their lives. Those with more internal locus of control take more responsibility for what happens to them and respond to therapy better (32, 33). They tend to look at what they do as the cause of pain, whereas external locus persons may feel their continued pain is because a physician did not fix them. Whatever the case, if patients want to get better they must be the ones to take control of their pain situation.

As discussed, solicitous spouses tend to draw pain behavior from their spouses in pain. More objectively, Lousberg found that when on a treadmill, measuring heart beat and pain intensity, patients with solicitous spouses report more pain and walk a shorter duration in the spouse's presence than those patients with relatively nonsolicitous spouses (38). This study lends much support to the theory that family interaction and psychological influence is much more influential on behavior than might have been previously thought.

To turn to more specific psychological problems we find that LBPPs exhibit features consistent with alexithymia and personality disorders (39). Another author has found that in 60 patients with LBP, 30 with clearcut organic findings and 30 without clearcut organic findings, the organic group showed symptoms consistent with a neurotic triad: hypochondriasis, depression, and hysteria (40). The more we study the more we see how the mind and body are linked in this and perhaps other pain conditions.

We have behaviors to react to anything, including illness. How one reacts to pain and the behaviors that accompany those reactions may be dysfunctional in nature if they do not help the person cope with the problem. An illness behavior is the way a person perceives, evaluates, and reacts to symptoms (20). An illness behavior or reaction to illness can take many forms, ranging from hypochondriasis to complete denial (20). The term "abnormal illness behavior" (AIB) has been introduced to describe behaviors that are inappropriate for the illness. AIB was introduced with the thought that studying a patient's ideas, attitudes, and behaviors is a more sound way to diagnose than basing a diagnosis on physical symptoms (20). The most common forms of AIB with LBP are hypochondriasis and pain disorder (20).

Behaviors can develop into role patterns patients may assume. The sick role appears to develop out of certain behaviors. For example, "I can't do yard work," "I'll hurt my back," or more subtly, before being asked to help with the yard work, the person moans loudly while getting out of a chair or grabs the back in pain when moving about the house, indicating that any work will cause further discomfort. Assuming the sick role, justified or unjustified, grants certain privileges. Some of these privileges are being exempted from certain duties, regarded as deserving of care, and thought of as not responsible for the condition (20). How often has a patient said, "Oh thank you doctor for the diagnosis, now my husband or wife won't think I'm making this up." I realize some of these thoughts are not "rocket science," but grasping them consciously may help our understanding.

The upside to the sick role is the permitted time to heal and rest. The downside to the sick role is taking advantage of the role at home and in the workplace, leading to disability. Insurance companies and employers hate disability, and for good reason. Where does the sick role emanate from? Who decides

who gets to assume the sick role? Doctors do. Doctors are not the bystanders watching all this take place. We as physicians are principally responsible for granting sick roles (20). Are we part of the problem of increasing disability in this country? Could this have been headed off if we could have recognized the signs of someone playing the sick role inappropriately? A very definite maybe.

Somatiform disorder, which may be looked at as an AIB, is a condition in which a psychological distress gets expressed or communicated as a bodily symptom (20). By expressing distress this way, the patient is temporarily spared the awareness of unacceptable emotions or conflicts and is granted a way out (20). The psychological issue appears to be too painful to deal with and therefore gets expressed as a physical complaint. In my opinion, this is what happens to the new population of fibromyalgia patients. Any attempt to suggest that a psychological or psychosocial issue may be involved in the condition is met with anger, as do referrals to therapists. At this point, reassurance that the pain is real is needed; however, the cause can be multifactorial. In my experience, these are the patients at risk for developing chronic pain: those who are harboring some type of emotional or psychological pain that is expressing itself through the body. Even worse is the patient who refuses to see that an issue must be dealt with and who attempts to make it go away with medication.

HOW DO WE CARE FOR THESE PATIENTS?

Accept the patient's experience as real. Treat the physical complaints, and accept a need for a transitional phase to deal with the psychosocial issues (20). Very few chronic patients expect to get 100% relief. This is not the goal. Rather, rehabilitation and development of healthy coping strategies are the goals (20).

Another study reports that nonphysical factors can be influencing less serious episodes of LBP (41). The course or seriousness of a back injury may be determined by how much press the injury gets (i.e., does it get reported to the employer?). Factors found to be influential to whether or not an injury gets reported on the job are feelings that the job is perceived to be a burden, unenjoyable, unfulfilling, and providing few assets (41). I have seen this point over and over again in younger men who have back trouble. They typically only work the job they do for the money or benefits, not because they like the job. Most of the jobs can also be linked to the cause of pain (i.e., heavy lifting or driving).

Further support is lent by Burton who found that persistent back trouble with a history of back problems may be caused more by psychosocial influences than to physical or medical conditions (42). His feelings with regard to stopping the progression to chronicity is that coping strategies need to be implemented so that AIBs are reduced or prevented (42). The physician's goal should be to help these people cope with the pain and maintain as normal a life as possible.

In the same light, but different condition, in families where the mother suffers chronic headaches significantly reduced intrafamily openness is found (43). That is to say that the family does not share experiences with one another as families usually do. The pain groups in this study were less active in their leisure time than pain-free groups (43).

Correlational studies comparing CLBPPs with controls has found lifetime depression rates to be 32 and 16%, respectively; alcohol abuse (which may increase the risk of developing CLBP) rates in CLBPPs versus controls were 64 and 38.8% respectively; and major anxiety disorders, 30.9 and 14.3% respectively (44). It was further found that late-onset mood disorder was common and that the initial major depressive episode began within the first 2 years of pain (44). It is further stated that men have a high risk of developing new-onset or recurrent major depression throughout their pain careers (44). Prevalence of depression in CLBPP is three to four times that of the general population (45). Depression is highest in the first 2 to 3 years after onset of CLBP (45). Substance abuse and anxiety disorder tend to precede onset of CLBP, whereas major depression appears to develop before or after CLBP (46).

Depression alone would not be such an issue if that were the only concern. Depression can lead sufferers to commit suicide, divorce, or withdraw from society. At Johns Hopkins Medical Center, 70% of those with CLBP divorced, and 20% had contemplated or attempted suicide (47). These are issues that need to be considered when deciding whether to recommend psychological intervention. A clinician must recognize those life-affecting decisions that need to be made for the patient's best good. A physician may lose a patient by suggesting therapy, but the risk also includes saving or improving a life.

Patients with CLBP syndrome (high levels of pain, disability, and depression) reported greater life adversity, more reliance on passive avoidant coping strategies, and less satisfaction with social support (48). In the same study, patients with good pain control (low levels of pain, disability, depression) reported less life adversity, less reliance on passive avoidant coping strategies, and more satisfaction with social support networks (48). A third group in this study is the positive adaptation to pain group, which showed less life adversity, more reliance on passive avoidance coping strategies, and more satisfaction with their social support (48). This is the group physicians want in the office. These people have accepted that they will have pain in their lives, but at the same time they take responsibility for the activities that make it better or worse. This group does not allow pain to dictate their activities. These are the congruent patients. Educational efforts such as low back wellness school, including activities of daily living training, will dictate how congruent these patients are and how successful interventions will be.

Many facets are involved in the diagnosis and care of pain in these individuals. The book is unfinished when it comes to which came first: psychological problems or LBP. What I hope to have conveyed is a sense that persons with CLBP have many areas of their lives affected by their pain and many reasons they can be trying to hide as causes of their disability. At this point the next logical question is how to identify these people and how to proceed after they are identified.

DIAGNOSIS AND IDENTIFICATION

A correlation between pain drawings and Waddell's nonorganic physical signs has been found (8). A large proportion of patients with high Waddell scores have nonorganic pain drawings (8). A nonorganic pain drawing is a pain diagram that is nonspecific, typically covers many body areas, and describes pain vaguely. If a diagram indicates use of specific symbols for sharp or dull or tingling, oftentimes the patient will mark these diagrams with haphazard lines and marks that poorly describe their symptoms. The pain diagram may be the first clue to look further for causes of pain.

It is suggested by Sandler and Becker that domestic stressors must be identified, such as family, work, and especially financial difficulties (5). More clues may be lengthy visits, frequent appointments, multiple phone calls, a history of addiction or dependency, or increasing medication requirements (5). These persons may also present with a history of childhood abandonment and abuse (5).

A patient's recall of pain is subject to overestimation and inaccuracy (49). In the same study, those who relied on pain medication reported more emotional distress, conflict at home, a less active lifestyle, and inaccurate memories of pain (5). Poor memory of pain makes treatment of manageable conditions a problem because as soon as the pain is gone, so is the patient if not well educated to prevention. This point is explicit in demonstrating how important education is in a patient's therapy.

How Do People Cope with Pain?

One obvious way people cope with pain is called "fear-avoidance" (50). Fear-avoidance is a means of coping in which a person, afraid of the possibility of returning pain, avoids behaviors that may produce the pain. The good thing here for the patient is that the pain may not return, hopefully. The problem is that it points on a dead-end course with disability, social withdrawal, and lack of the activities that may help the patient get well (e.g., exercise). The fear of the pain returning needs to be overcome. Abnormal illness behaviors and sick roles are coping mechanisms.

More than likely the pain will return, to some degree, at some point. With this likelihood pointed out to the patients, some will not fear the pain as much. Accepting that some pain is inevitable and that many activities can be done in the presence of the pain begins to relay to the patient that life does go on. Some LBPPs use attention diversion and praying or hoping as coping skills (51). Another passive technique is waiting or hoping for the pain to go away. This may be one of the factors that prolongs the occurrences of low back pain.

Good adaptation or coping was shown in one patient study group who experienced LBP. They were found to report less life stress, relied less on passive-avoidant coping strategies (waiting for the pain to go away and avoiding activity), and more satisfaction with social support networks (48). Good coping skills are needed to reduce AIBs (42). What is needed is a change from passive to active coping, avoidance to involvement. Instead of sitting and waiting for the pain to go away, the person must actively seek help. Instead of avoiding behaviors that can cause pain, the patient must replace them with behaviors that may help reduce the pain and strengthen the back.

As a coping side note, Japanese LBP patients were found to be significantly less impaired in psychological, social, vocational, and avocational functioning (52). Specifics are not forthcoming; however, LBP and disability are not universally experienced phenomena throughout the world.

How Are These Patients Being Treated?

First of all, from whom do these patients seek care? One study reports that for acute LBP, 24% of the people sought an allopath, whereas 13% sought out a chiropractor (53). Interestingly only 39% of people with pain in this study sought care (53). Those who did seek care had more prolonged and severe pain and sciatica (53). Pain down the thigh and leg gets people's attention! Seeking care from a chiropractor correlated with younger men and non–job-related pain (53). In this study, the decision to seek care was not related to gender, age, rural residence, or health insurance status (53). Other studies show similar results.

Many different ways are available to health care providers to treat CLBPPs. Some of the more popularly tried and researched methods follow. Waddell recommends that the fear avoidance beliefs should be considered in medical management of these patients (50). This may mean referral for psychological intervention. General improvement was found for the CLBPPs who received behavioral therapy to address issues of depression and decreased activity (54).

Treating CLBPPs with intensive physical therapy and psychosocial training was more efficient with regard to physical measures of pain and disability indexes than physical therapy alone (55). A bolder statement has been made saying that to improve occupational handicap, the activities of the society as a whole, including social legislation and labor market policies, need to be examined (55).

To take a different approach, Ruta defines what needs to change if quality of life is to be improved (56). Quality of life is defined as "The extent to which our hopes and ambitions are matched by experience" (56). His approach to improving the quality of life is to narrow the gap between patients' hopes and expectations and what actually happens (56). That can be done one of two ways: either change reality, or help the person align expectations with reality. In my opinion, the latter is much easier!

Significant improvement was found in 26 CLBPPs after a therapy regimen of biofeedback, physical therapy, behavioral management, pain measurement, psychotherapy with pain counseling, and medication (57). Strong emotional overlay was found in all 26 (57). In this study and others the psychiatric component was viewed as the anchor of all the therapies and as the key to success (57, 58). Stenger states that the emotional component in CLBP must not be overlooked because it plays a vital role in successful treatment (46, 57). Eisendrath concurs

that CLBPPs require psychological intervention (59). As in most research settings, conflicting information is forthcoming. CLBP research is no exception. In other studies, psychological interventions were found not to alter the standard rehabilitation outcomes (34, 60).

In treating CLBPPs the placebo effect can be considered effective. In a study of phentolamine versus placebo in reducing CLBP, significant reductions in pain were achieved prior to and in the absence of the active drug (61). As in other placebo situations, we see how powerful belief can be. I believe that those people who know they will get better, do in fact get better.

When conservative measures are overlooked and drug therapies fail as they eventually do, people are quick to choose surgery. When looking at surgical outcomes the picture is not what we would hope it to be. In two series of patients operated on for disc protrusion, 29% had not returned to work; worse yet, of the women operated on, 84% were subsequently unemployed (62). A 4-year follow-up study after low back operation showed that only 39% of people had returned to work comparable to previous employ (63); that is, 61% of them did not return to work comparable to previous employ. On a conservative note, Lindstrom found that during treatment of subacute LBP, returning people to work is very important in their rehabilitation (64).

Considering the above surgical outcomes, it behooves clinicians to know some ways to predict success? Wilfling et al. found four areas they feel are predictive of success of a spinal fusion: vulnerability in terms of occupational and interpersonal insufficiencies, presence of ego strength resources, presence of a neurotic triad, and presence of minor musculoskeletal complaints (65). Essentially, all areas of a person's life need to be assessed, even childhood (26), to get an idea of whether spinal surgery will be successful. Anything less appears to be a guess. However, not all research venues agree with that either. Gatchel et al. found that psychopathology did not alter the predictive value of successful return to work (66). As is usual, research does not have the definitive answer. More stringent criteria are needed to study the prevalence of LBP (67). Research is not conclusive in defining the predictive values of when patients will be better.

Long-term studies are few, but they are helpful in seeing whether or not people are benefitting from the therapies used. Gallon studied CLBPPs for 4 to 6 years. Statistics from medical treatment are as follows (68):

- 56% reported they were working or ready for work
- 58% were no longer receiving compensation
- 62% were taking medication
- 65% were receiving no or brief medical treatment
- 29% perceived themselves as impaired

Of the 48 who had surgery, 17% felt they were improving; however, **58%** considered themselves **worse**!

In a 4-year follow-up after surgery for low back pain only 39.5% of persons had returned to comparable work (63). As you can see these results are less than optimal, especially for surgical approaches.

Burton et al. take another approach. Their idea is to identify the at risk patients up front and arm them with the coping skills to prevent a back condition from becoming chronic (42). This approach may decrease the number of patients who take on the sick role and develop AIBs. One opinion is that rehabilitation improvements may be more the result of changes in pain behavior than to the psychological effects of therapy (69). When stress, depression, and secondary gain issues were addressed, patients improved significantly (57). Further support for addressing secondary gain is added from Sivik and Delimar's study of CLBPPs in Sweden. They found that accident groups with minor injuries held onto the injuries longer, possibly because of the liberal insurance policies in Sweden (70). Varying opinions abound. The approach to treating and addressing emotional issues is more consistently showing results than is not addressing those issues.

When we look at other pain producing conditions versus LBP we find that a LBP group rated higher in a neurotic triad than a fracture group (71). The LBP group had more emotional and psychological issues than the fracture group. This may be in part because of the fact that those with fractures were relatively certain that in a specified time their symptoms would pass and life would go on as usual; most LBPPs do not have that same assurance. Patients with fibromyalgia syndrome (FMS) were found to be less likely to report pain relief than those with a herniated nucleus pulposus (2).

FINAL THOUGHTS

No one approach will satisfy all patients. Holistic approaches are now becoming the norm and should continue to gain popularity as we learn more and more about the mind-body connection. The more we learn of our patients, the more insights we will gain about how best to serve them. The future of health care will not be emergency medicine and heroic efforts to save lives. Health will become the way of life. The situation will no longer be what we do to avoid illness, but rather, what we do to stay healthy, replacing avoidance activities with proactive healthy activities. We are currently looking outside ourselves for the answer, the cure. This approach has and will continue to fail if our goals are to remain healthy. The answer to health is not removing the disease, but rather, restoring the individual.

Physicians do not heal or fix or cure. Physicians educate. Their job is to help set up the circumstances that best allow the body to heal. Once doctors learn to get out of the way and let the body heal, the practice of medicine will have gained wisdom.

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ADDENDUM A: LITERATURE UPDATE

The following research was added just before book production to give the reader the most up-to-date research information available on low back pain. This addendum is divided into the following sections:

- Biomechanics and causes of low back pain
- Disc
- Incidence
- Treatment

BIOMECHANICS AND CAUSES OF LOW BACK PAIN

Subluxation Causes Disc Herniation Pain

The meningovertebral ligaments (ligaments of Hofmann) attach the dural sac to the posterior aspect of the vertebral bodies and the posterior longitudinal ligament (PLL) and could act as a tractive force on the dural sac in the event of nuclear bulge or herniation. A vertebral subluxation brought on by a disc bulge may place tension on the dural ligaments and, in so doing, place traction on the PLL and vertebral endosteum. Thus, reducing the subluxation will alleviate this tension and may reduce or abolish accompanying noxious effects (1).

Micturition Difficulties Found in 55% of Men at Surgery for Stenosis or Disc Herniation

Fifty-five percent ($n = 180$) of male patients who had lumbar disc herniation or spinal stenosis surgery had significant symptoms of lower urinary tract micturition problems. Eighty percent of the patients with spinal stenosis had symptoms: 33 patients had irritative symptoms, 36 had obstructive symptoms, and 23 had retention symptoms; 24 had severe symptoms. Median compression resulted in more symptoms than paramedian compression. No correlation was found between age, compression level, drug intake, or pain score and lower urinary tract symptoms.

Lower urinary tract symptoms of mixed type occur with a high prevalence in male patients with lumbar root compression syndromes referred for neurosurgical evaluation and treatment (2).

Lumbosacral Plexus Is Fixed to Sacral Ala with Fibrous Tissue

The lumbosacral plexus was dissected bilaterally in 20 adult cadavers and showed the width of the nerve roots of the lumbosacral plexus was greatest at S1. The L5 nerve root was the thickest in males, and the S1 nerve root was thickest in females. The fifth lumbar nerve root and lumbosacral trunk coursed across the sacroiliac at a level 2.0 ± 10.2 cm below the pelvic brim and were relatively fixed to the sacral ala with fibrous connective tissue (3).

Anular Tears Damage Disc Elasticity

Four 2-year-old sheep received anterolateral incisions (4×10 mm) in the outer one third of the anulus fibrosus of their L2–L3 and L4–L5 discs (lesion group). The anulus was not incised in another four sham-operated animals. After 6 months the sheep were killed, and it was found that the introduction of an anular lesion significantly affected the proteoglycan metabolism of endogenous disc cell populations. The focal depletion of aggrecan by anular lesions, therefore, may represent an important predisposing factor to the subsequent degeneration of these intervertebral discs (4).

Inner and Middle Anulus Behaves As Nuclear Material and Has the Highest Intradiscal Pressure

Intervertebral discs are probably the most common source of chronic low back pain. The outer anulus fibrosus has a nerve supply and pain may arise from abnormal mechanical stimulation after either posterior herniation of nuclear material or internal disruption of the disc structure.

Anulus fibrosus pressures have not been studied in proportion to the nuclear pressures, although the anulus contains the nerve endings of the disc and has the most active cells. The highest pressures are not in the nucleus pulposus, as is commonly believed, but in the inner and middle anulus fibrosus, especially posterior to the nucleus. This is because the inner anterior anulus shows a hydrostatic resistance to pressure and thus behaves as part of the nucleus, despite its distinctly lamellar structure.

Degenerative changes that most affect intradiscal stress distributions cause structural defects that damage the vertebral body end plate because of reduced pressure in and doubling the size of "stress peaks" in the posterior anulus (5).

Postinjury Healing of Anatomic Structures of the Lumbar Spine

Forty-four pigs were used in six chronic lesion models: sham, disc anulus, disc nucleus, facet capsule, facet joint slit, and facet joint wedge. Three months after injury, an instrumented linkage was used to measure continuously the sagittal kinematics of the L3–L4 motion segment during flexion–exten-

sion, with and without stimulation of the lumbar paraspinal musculature. Flexion–extension end point, maximal ranges of motion, and hysteresis were analyzed.

Discs that received a stab incision to the anulus healed in the outermost anulus but not in the inner anulus, thus leaving a cavity. The nucleus pulposus maintained its normal form and gel-like appearance. Discs that received an incision into the nucleus pulposus showed signs of severe degeneration in the anulus, the nucleus pulposus, and the end plate. Healing occurred in the outermost anulus, but there was considerable disruption of the inner anulus in the form of irregular fissures, and the nucleus pulposus was fibrous and discolored. Disc height was reduced, and osteophytes had formed in most cases.

With facet capsule injury, the incision into the collagen capsule of the facet joint induced an inflammatory process in the synovial cavity, resulting in discoloration of the cartilage.

Stimulation of the nerve endings in the outer anulus of the intervertebral disc and facet joint capsule elicits a response in the lumbar paraspinal musculature, which suggests that such activation may have a stabilizing effect on injured or diseased structures by constraining the motion in the lumbar spine. It is plausible that the more degenerative facet and disc lesions induced in this study, through the complex innervation network, affected the surrounding musculature to stimulation.

The lumbar paraspinal musculature is less efficient overall in providing stability during flexion–extension when chronic lesions occur in the intervertebral disc or facet joints, possibly because of altered mechanisms in the neuromuscular feedback system in the degenerated motion segment and, consequently, in the lumbar spine as a whole (6).

Spine Motion and Stabilization Determined by Disc and Facet Proprioception for Spinal Muscles

The outer anulus of the intervertebral disc, the capsule of the zygapophysial joint, and the ligaments are innervated by a network of fine nerves. The sources of the nerve endings in the lumbar discs are the lumbar sinuvertebral nerves, the branches of the lumbar ventral rami, and the gray rami communicantes. Innervation seems to be scarce and is not uniformly distributed in the disc. Innervation of the zygapophysial joints, which is confined to the capsule, is derived from the posterior ramus of the spinal nerves. Each joint receives innervation from the nerves on the same vertebral level and from the level above and below. Nerve endings in the disc anulus and in the joint capsule have different mechanoreceptors and free nerve endings. This innervation network is probably part of a proprioceptive system that recruits paraspinal muscles for motion and stabilization of the motion segments.

Using an experimental model, it was demonstrated that a neuromuscular interaction exists among the intervertebral discs, zygapophysial joints, and the paraspinal muscles. Injection of physiologic saline into the facet joints, most likely causing a stretching effect of the facet capsule, reduced the muscular response, suggesting the existence of a complex reflex system that is responsible for the motion and stabilization of the lumbar spine (7).

Stenotic Canals Are Developmental, Not Congenital

An archaeological study examining two different populations with different nutritional habits revealed that the trefoil canals were more frequent in individuals with a low protein intake. This finding suggests that malnutrition can be a factor responsible for such canal transformation.

It is well recognized that the fifth vertebra is almost complete in its development at approximately 5 years of age in terms of shape and diameter, and the fourth lumbar vertebra is largely mature at the age of 1. The factors that might be responsible for a trefoil canal should be in effect before the maturation of vertebrae. Currently, little is known about the development of the trefoil canal, and satisfactory explanations for its existence are lacking. It has been suggested that trefoilness may be caused by the lordosis of the vertebral canal that develops after the child is able to stand. As lumbar lordosis develops, this triangular tube gets bent so that a trefoil canal develops.

The results of this study suggests that (*a*) the trefoil configuration of the lumbar vertebral canal is not seen in newborns; (*b*) the canals in newborns are dome-shaped, and (*c*) the trefoil configuration is developmental in nature (8).

Abnormal Hydrostatic Disc Pressure May Accelerate Disc Degeneration

Hydrostatic pressure influences intervertebral disc cell metabolism. A physiologic level of hydrostatic pressure (3 atm) may act as an anabolic factor for stimulation of proteoglycan synthesis and tissue inhibitor of metalloproteinase-1 production, which may be essential to maintain the disc matrix. If the pressure were 30 atm or more or 1 atm or less, a catabolic effect would be predominant, with reduction of proteoglycan synthesis rate and increase of matrix metalloproteinase-3 production. Abnormal hydrostatic pressure, therefore, may accelerate disc degeneration (9).

Nerve Ingrowth Occurs Into the Deeper Anulus Fibrosus Layers in Disc Disease

In the healthy back only the outer third of the anulus fibrosis of the intervertebral disc is innervated. Nerve ingrowth deeper in diseased intervertebral disc has been reported, but how common this feature is and whether it is associated with chronic pain is unknown.

The finding of isolated nerve fibers that express substance P deep within diseased intervertebral discs and their association with pain suggests an important role for nerve growth into the intervertebral disc in the pathogenesis of chronic low back pain (10).

Spina Bifida Occulta Linked to Abnormalities

Data exist to support the premise that spina bifida occulta (SBO) can be linked to urologic dysfunction and manifestations of tethered cord syndrome, foot deformity, and an increased incidence of spondylolisthesis and intervertebral disc herniation. Currently, data supporting an association between epilepsy and SBO are equivocal. No data currently support an association of SBO with constipation (11).

Axial Loading of Lumbar Spine Enhances Stenosis Diagnosis

The diagnostic specificity of spinal stenosis increases considerably when the patient is subjected to an axial load. Axial loading of patients during computed tomography (CT) or magnetic resonance imaging (MRI) examinations showed pathologic features not detected in the regular, unloaded psoas relaxed position. In 29 of 84 patients with sciatica and neurogenic claudication, the load provocation disclosed relative and absolute central spinal stenosis in 40 sites. The specificity of the spinal stenosis diagnosis increases considerably when the patient is subjected to axial loading (12).

Functional MRI of the lumbar spine obtained in an upright position was compared with functional myelograms with regard to the sagittal diameter of the spinal canal. In addition, the influence of motion on the foramen was examined in various positions. Functional MRI in a sitting position can replace myelography with regard to spinal stenosis. Foraminal stenosis was not position dependent using this study set-up (13).

NERVE ROOT IRRITATION RESEARCH

Nerve Root Pressure Determines Symptoms and Signs

Nerve root pressure levels involved in 27 consecutive disc herniations showed the pressures varied from 7 to 256 mm Hg, with a mean pressure of 53 mm Hg. Pressure on the nerve root dropped to zero in all cases after the removal of the offending disc material. Pressures determined the clinical findings listed below.

Patients with no neurologic deficits: Of the 27 patients, only 4 had no neurologic deficits. In these cases, the mean nerve root pressure reading was 20 mm Hg.

Patients with neurologic deficits: Twenty-three patients had neurologic deficits such as muscle weakness and sensory disturbance. The mean nerve root pressure was 60 mm Hg in these cases.

Patients with severe deficits: Four subjects had exceptionally high pressure readings (> 100 mm Hg). These patients had severe neurologic deficits and trunk list.

Interestingly, no correlation was found between pressure readings and straight leg raising. In general, no clear patterns of differences in pressure readings were found among disc protrusions, extrusions, and sequestrations (14).

Fibrosis and Hypervascularity Occur After Four Weeks of Nerve Root Compression

Chronic nerve root compression is related to back pain and sciatic syndromes. Nerve root constriction with an initial inner diameter of 2.5 mm or 3.5 mm was found to induce a significant reduction in nerve conduction velocity in nerve roots compressed for 1 week compared with the noncompressed contralateral control nerve root. Nerve conduction velocity, using the 3.5 mm constrictor, also was reduced after 4 weeks, but not significantly more than after 1 week. All samples from compressed nerve roots showed some degree of nerve fiber damage as assessed by light microscopy, and severe changes were found in most animals. In contrast, samples from the noncompressed roots displayed no or slight nerve fiber damage. Endoneural bleeding and signs of inflammation were more common after 1 week than after 4 weeks in the compressed nerve roots. Epidural proliferation of fibroblasts and capillaries was observed more often after 4 weeks than after 1 week of compression (15).

Proprioception Is Disturbed in Low Back Pain Patients

Twenty patients with back pain and 20 without back pain were required to reproduce predetermined target positions, in standing and four-point kneeling, 10 times in 30 seconds. A computer screen provided visual feedback on position. Differences in proprioception do exist between individuals with back pain and those free from back pain. Further research needs to be undertaken on proprioception exercise programs and their effect on back pain (16).

Reflex Sympathetic Dystrophy Is Now Termed "Complex Regional Pain Syndrome"

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or is described in terms of such damage. The primary afferent nociceptor is generally the initial structure involved in nociceptive processes. Nociceptors respond to chemical, mechanical, and thermal stimuli. Two main fiber types, the faster-conducting myelinated A fibers and the slower-conducting unmyelinated C fibers, are involved in the transmission of nociception. Damage to a peripheral nerve results in a number of physiologic, morphologic, and biochemical changes that act as a focus of pain in themselves. Reduction in food supply to myelinated fibers results in demyelination. This demyelination results in the production of ectopic impulses that can be perceived as a sharp, shooting, or burning pain in conditions such as diabetic neuropathy.

Nociceptive stimulation also results in a neurogenic inflammatory response. This produces vasodilation and extravasation of plasma proteins as well as action on inflammatory cells to release chemical mediators. These interactions result in the release of a "soup" of inflammatory mediators such as potassium, serotonin, bradykinin, substance P, histamine, and products from the cyclooxygenase and lipoxygenase pathways of arachidonic acid metabolism. These chemicals then act to sensitize high-threshold nociceptors. After sensitization, low-intensity stimuli that normally would not cause pain are perceived as painful. This series of events that occurs after tissue injury is termed "peripheral sensitization."

The sympathetic nervous system also has an important role in the generation and maintenance of chronic pain states. Nerve damage and even minor trauma can lead to a disturbance in sympathetic activity that in turn leads to a sustained condition termed a "complex regional pain syndrome" (CRPS); this now replaces the previously used term "reflex sympathetic dystrophy." Complex regional pain syndromes are associated with features of sympathetic dysfunction, including vasomotor and sudomotor changes, hair and nail growth abnormalities, osteoporosis, and sensory symptoms of spontaneous burning pain, hyperalgesia, and allodynia. The dorsal root ganglion becomes innervated by sympathetic efferent terminals.

Inhibitory mechanisms: Descending projections arise from several structures including the hypothalamus, periaqueductal gray matter of the midbrain, locus ceruleus, ventromedial (nucleus raphe magnus), and the ventrolateral medulla to form descending inhibitory pathways. Projections from these regions directly or indirectly terminate at a spinal level to modulate incoming nociceptive signals. A variety of neurotransmittors have been implicated in descending inhibition. These include the endogenous opioid peptides (β-endorphin, enkephalin, and dynorphin) as well as other neurotransmitters such as serotonin and noradrenaline.

Clinical pain syndromes: Pain can arise from a number of structures within or adjacent to the spinal column as a result of fractures, tumors, infection, inflammation, and instability. These structures include the intervertebral disc, zygapophysial joints, vertebral bodies, and surrounding ligaments and muscles. Pain can also rise from compression and damage to nerve roots exiting from the spinal canal and damage to the spinal cord itself (17).

The term "complex regional pain syndrome" encompasses causalgia and reflex sympathetic dystrophy. Symptoms of burning pain with autonomic and tissue changes begin shortly after an injury, usually to a distal extremity. Diagnosis is based on the history and the clinical findings. No confirmatory tests are available, although plain radiographs or a three-phase bone scan may be helpful in diagnosing some cases. Despite treatment, many patients are left with varying degrees of chronic pain and disability.

Because complex regional pain syndrome is relatively uncommon and is often misdiagnosed, its actual incidence is unknown. The syndrome is estimated to occur in 1 to 5% of patients who have sustained peripheral nerve injury. It may affect as many as 30% of patients after Colles' fracture or a tibial fracture.

Clinical features include:

1. Pain: patients with CRPS describe their pain as constant, burning, aching, and throbbing. The pain usually begins days to weeks after the initiating incident, and it persists beyond the time normally expected for the injury to heal. Factors that aggravate the pain include changes in the temperature, active and passive movement of the extremity, and light pressure from air currents and clothing. Emotional distress or excitement can also exacerbate the pain.
2. Tissue changes: Early in the course of CRPS, the affected area is warm, erythematous, and dry. In time the skin becomes cool, cyanotic, and moist. Soft puffy edema progresses to tight shiny swelling with loss of skin creases. Accelerated hair and nail growth occurs early in the syndrome, but the hair soon becomes sparse and the nails become grooved and brittle. With a decrease in fat pads, the digits become thin and pointed. As these signs progress, they become irreversible. Muscle spasm and wasting occur and the joints thicken. Eventually, patients have marked bone and muscle atrophy, weakness, and flexor tendon contractures.
3. Psychologic sequelae: Depression, anxiety, and hypochondriasis.

Treatment is as follows:

1. Preventive measures: Prescribing antibiotics for secondary infection.
2. Sympathetic blockade and lytic procedures: Sympathetic ganglion blockade with lidocaine or intravenous regional sympathetic blockade with guanethidine should be performed by an anesthesiologist or a physician experienced in the technique. The blocks are repeated until the symptoms resolve or the blocks are no longer effective.

   Chemical or surgical sympathectomy is indicated only for profoundly disabled patients who have responded positively to sympathetic blockade and have no other treatment options. Pain commonly recurs within 6 to 12 months after sympathectomy.
3. Pharmacologic therapy: Propanolol (Inderal) phenoxybenzamine (Dibenzyline), and guanethidine (Ismelin). A clonidine patch (atapres-TTS) is applied to the sensitive area. Nifedipine (Adalat, Procardia) may help to relieve pain related to vascular instability.

   Nonsteroidal anti-inflammatory drugs (NSAIDs): Capsaicin cream (Zostrix) decreases pain by depleting the skin of the neurotransmitter substance P.
4. Other therapies: Transcutaneous electrical neuromuscular stimulation (TENS) unit, implantation of a morphine pump or a spinal stimulator, amputation of the affected limb, psychological counseling, and antidepressant drug therapy (18).

Chemical Irritant Effects on the Nerve Root

Phospholipase A_2 was injected into the rat L4–L5 epidural space, and the rats were observed for 3 or 21 days. Motor weakness

and altered sensation were observed. At 3 days of stimulation, squeezing the dorsal roots at the L4–L5 disc level evoked sustained ectopic discharge that lasted approximately 8 minutes.

Found in high concentration in herniated disc material, PLA_2 can cause nerve root injury and corresponding behavioral and electrophysiologic changes consistent with sciatica (19). Herniated cervical and lumbar disc specimens spontaneously produce increased amounts of nitric oxide, interleukin 6, prostaglandin E_2, and certain matrix metalloproteinases. These biochemical agents are in some manner involved with degenerative processes in the intervertebral disc (20).

Application of nucleus pulposus and anulus fibrosus material to the lumbar epidural space produces different forms of hyperalgesia (mechanical versus thermal), with different and distinct histologic changes. PLA_2 and nitric oxide produced in or around herniated disc materials may play important roles in pathomechanisms of radicular pain in patients with lumbar disc herniations (21). Nitric oxide in a lumbar disc herniation is mainly produced by cells in granulation tissue around the herniated intervertebral disc (22).

Sympathetic Sprouting to the Dorsal Root Ganglion Results in Neuropathic Pain

The time course of sympathetic nerve sprouting into the L4–L6 dorsal root ganglia (DRG) of adult rats following a chronic constriction injury (CCI) made on the sciatic nerve, or following sciatic nerve transection at the same site found sympathetic sprouting in the DRG by 4 days following CCI, paralleling the decreases in mechanosensory threshold and preceding changes in thermal thresholds. Thus, after CCI, sympathetic sprouting occurs with a sufficiently rapid time course for it to play a role in the genesis of neuropathic pain. The researchers suggest that the more rapid sprouting seen after CCI than after resection is caused by the availability of products of Wallerian degeneration, including nerve growth factor, to both spared and regenerating axons following CCI, but not following resection.

Sympathetic sprouting plays an important role in neuropathic pain following partial injury. No general consensus has been reached on the role sympathetic innervation plays in human neuropathic pain. Stimulation of sympathetic efferents innervating the axotomized DRG caused an increase in spontaneous ectopic discharge of sensory neurons originating within the DRG (23).

ATHEROSCLEROSIS RELATED TO LOW BACK PAIN

Aortic calcification level significantly correlates with the level of disc degeneration. Subjects in whom aortic calcifications developed between examinations had disc deterioration twice as frequently as those in whom aortic calcifications did not develop. Persons with severe posterior aortic calcification in front of any lumbar segment were more likely than others to report back pain during adult life.

Advanced aortic atherosclerosis, presenting as calcific deposits in the posterior wall of the aorta, increases the risk for development of disc degeneration and is associated with the occurrence of back pain (24).

TROPISM

Tropism as a cause of disc herniation is controversial—yet juveniles show a five times incidence over adults when disc herniation is present. This result indicates that facet joint asymmetry is a radiologic feature of lumbar intervertebral disc herniation in children and adolescents.

Farfan and Sullivan (25) emphasized that torsional stress of a magnitude encountered in daily activity plays a major role in the initiation of disc degeneration and herniation. They suggested that asymmetry of the facet joints is correlated with the development of disc herniation. Because the coronal facing facet joint offers little resistance to intervertebral shear force, the rotation occurs toward the side of the more coronal facing facet joint, and this possibly leads to additional torsional stress on the anulus fibrosus.

Cyron and Hutton (25) demonstrated that the coronal facing facet joint offers little resistance to intervertebral shear force, so the joint tends to rotate toward the side of the coronal facing facet joint, which could lead to an additional rotational stress on the anulus fibrosus. In contrast, Ahmed and Duncan (25) stated that no significant correlation exists between the facet joint asymmetry and the axial torque-rotation response. In clinical studies, Farfan and Sullivan (25) found a high association between the side of disc herniation and the coronal facing facet joint. They also found that the tear pattern of the posterior anulus of 200 fresh autopsy specimens appeared to be related to facet joint asymmetry. Noren et al. (25) concluded that facet joint asymmetry is a risk factor for the development of disc degeneration. Hagg and Wallner and Cassidy et al. (25) found no difference in the distribution of the more coronally or sagittally facing facet joints with respect to the side of herniation, and that the frequency of facet joint asymmetry does not differ significantly, regardless of the presence of disc herniation (25).

No correlation was found between degeneration of the cartilage and a small effect on sclerosis of the facet joint in another study (26).

In a study by Ko and Park, subjects ($n = 60$) were divided into two groups: 33 without disc herniation and 27 with disc herniation at one or more levels of lower lumbar motion segments. Facet joint tropism did not play a significant role in disc herniation in the lower lumbar spine (27).

DISC

Anular Tearing without Herniation Causes Radiculopathy

Pain drawings indicate that disc disruption passing into the outer layers of the anulus, but not resulting in deformation of

the outer anular wall, was as frequently associated with lower extremity pain as were discs with more severe disruption deforming the outer anular wall; however, outer anular tearing was associated with a greater degree of aching pain. These findings support the notion that lower extremity pain may be referred from the disc (28).

Groin Pain Incidence with Disc Herniation

Elderly patients with L4–L5 protruding herniation of the anulus fibrosus are likely to experience groin pain. The sinuvertebral nerve that innervated the posterior anulus fibrosus, the posterior longitudinal ligament, and the dura was indicated as the afferent nerve of groin pain. On MRI, more central herniation was noted in patients with groin pain. No significant difference was seen in degeneration of the discs or extent of herniation in the anterior and lateral direction between those with and without groin pain (29).

Serum Phospholipase A_2 As a Marker of Disc Inflammation in Sciatica Patients

Phospholipase A_2 activity was determined in the serum and discs of 31 patients (14 treated with acetaminophen and 17 treated with piroxicam) undergoing surgery for sciatica caused by lumbar disc herniation. Disc phospholipase A_2 activity was significantly higher in cases of sequestrated discs than in other herniations. Disc phospholipase A_2 significantly correlated with serum phospholipase A_2, and was significantly lower in patients treated with piroxicam than in those treated with acetaminophen.

Disc phospholipase A_2 is thought to participate in the physiopathology of sciatica and to be modulated by NSAID therapy. Serum phospholipase A_2 is suggested as a biologic marker of disc inflammation in patients with sciatica (30).

Normal Nucleus Pulposus Moves Differently Than Unpredictable Abnormal Degenerated Nucleus Pulposus

Ten men (aged, 21 to 38 years) with healthy backs were positioned in an MRI portal with their lumbar spine stabilized in flexion and extension by supporting pads. T2-weighted images were obtained, as was a computer- generated profile of pixel intensities along a horizontal middiscal transect. Mathematical curve-fitting regression analysis was used to characterize the shape of the intensity profile and to compute the point of maximal pixel intensity. A single equation fitted the profile for all normal discs. The intensity peak shifted posteriorly during flexion and anteriorly during extension.

It was reported that abnormal discs behave less predictably than normal ones. The distribution of pixel intensities observed in the nine abnormal discs in this study is highly variable and does not fit the single equation (curve) that the researchers found adequate for all normal discs (31).

Noncontained Discs Show Elevated Chemical Irritants

Thirty-seven patients undergoing surgery for lumbar disc herniation had the disc pathology of each patient classified into one of three groups: bulging disc, contained herniation, and noncontained disc herniation. Also during surgery, biopsy samples were taken from the nucleus. A significant difference was found in the levels of leukotriene B, and thromboxane B2 in contained *versus* noncontained disc herniation, and the highest concentration was found in the noncontained disc herniation group (32).

Degenerated Discs Show Increased Nerve Supply Deep in the Disc Anulus Fibrosus

Innervation of discographically confirmed degenerated and "painful" human intervertebral discs showed nerve fibers of different diameters in the anterior longitudinal ligament and in the outer region of the disc. In 8 of 10 degenerated discs, fibers were also found in the inner parts of the disc. Substance P immunoreactive nerve fibers were sporadically observed in the anterior longitudinal ligament and the outer zone of the anulus fibrosus. These findings indicate a more extensive disc innervation in the severely degenerated human lumbar disc compared with normal discs (33).

Extension Increases Posterior Disc Stress

Degenerated discs show extension increases posterior disc stress; extension does not cause nuclear material to migrate anteriorly. Severely degenerated lumbar discs vary in their response to extension and flexion movements with most degenerated discs showing extension movements increase stress concentrations in the posterior of the disc.

Backward bending usually increases stress concentrations in the posterior anulus, particularly in severely degenerated disc (personal communication from Michael A. Adams, PhD, and colleagues from the University of Bristol in England). However, in about 35% of discs, backward bending led to a reduction in stress, presumably because the neural arch shielded the posterior anulus from mechanical stress.

Two degrees of extension increased stress peaks by 33% in the intact discs, and by 43% in the "degenerated discs." However, in 7 of the 19 discs, extension resulted in a completely different mechanical response. In these discs, lumbar extension decreased posterior anular stress peaks by as much as 40% (34).

Interestingly, Adams et al. found no evidence that backward bending resulted in anterior migration of nuclear material (34).

Disc Degeneration Starts in Second Decade of Life

Disc degeneration starts as early as in the second decade of life. Therefore, early prevention of disc damage may inhibit disc degeneration and its sequelae (35).

Nutrient Deficiency May Lead to Degeneration of Disc

The variable metabolite concentration found in human discs suggests that these concentrations may change at different stages of the degeneration process. One could speculate that an initial fall in nutrient supply would lead to a fall in oxygen concentration and a rise in lactate concentration to a level that might result in cell death. Because demand would no longer be as high, oxygen levels would rise again to a level dependent on the remaining cells. A fall in end plate permeability, therefore, would eventually lead to cell death and high levels of intradiscal oxygen. To understand this process, it is not sufficient to measure metabolite levels alone; measurements of cell density and metabolic activity as well as end plate permeability and blood flow are required (36).

Right Anterior Quadrant of Disc Most Frequently Shows Tears

With the exception of radiating tears, which most commonly affect the posterior disc, the right anterior quadrant tends to show abnormalities more frequently than the other quadrants.

Two thirds of radiating tears involve the inner anulus but only one fifth extends as far as the outer anulus. Radiating tears overwhelmingly predominate in the posterior half of the disc, where they most commonly involve the inner anulus, whereas concentric tears are more frequent in the outer and middle zones of the anulus of the anterior half of the disc (37).

DURA AND LONGITUDINAL LIGAMENTS ARE POSSIBLE PAIN SOURCES

An extensive distribution of nerve fibers is seen in dura and longitudinal ligaments, which supports a possible role for these structures as a source of low back and radicular pain. Nerves destined for the dorsal dura may arise exclusively as branches of autonomic plexuses arranged around the origins of spinal nerve rami communicantes or they may be derived from ventral dural plexuses. The dorsal dura is considered to be less densely innervated than its ventral counterpart. Ventral dural innervation has been reported to be derived from the sinuvertebral (SV) nerve formed by joining of spinal and sympathetic fibers.

The PLL also derives its nerve supply from SV nerves of the sympathetic trunk and anterior branches of both the sympathetic trunk and the rami communicantes. Neuropeptide-reactive nerve fibers in the PLL have been reported (38).

Stretching of an irritated or inflamed dura could cause low back pain and sciatica during straight leg raising. Herniated disc material that is also inflammatory can leak into the epidural space and cause dural irritation. An inflammatory response initiated by extruded nucleus pulposus may sensitize nociceptive nerve endings in the ventral dura and dural sleeves (38).

INCIDENCE

Compensation Patients Do Not Get Well

Results of medical treatment are notoriously poor in patients with pending litigation after personal injury or disability claims, and for those covered by Worker's Compensation programs. Most exaggerated illness behavior in compensation situations takes place because of a combination of suggestion, somatization, and rationalization. A distorted sense of justice, victim status, and entitlement may further the exaggerated sick role. *Because any improvement in the claimant's health condition may result in denial of disability status in the future, the claimant is compelled to guard against getting well and is left with no honorable way to recover from illness.* Adversarial systems rewarding permanent illness or injury, particularly self-reported pain, are often permanently harmful (39).

Orthopaedic Impairments Second Only to Heart Disease Causing Work Limitations

According to data recently released by the National Institute on Disability and Rehabilitation Research, orthopaedic impairments of the back and neck and intervertebral disc disorders are among the top five chronic conditions causing work limitations. Heart conditions are first, causing 10.9% of all work limitations, with back and neck impairments, at 10.5%, a close second (40).

Back Surgeries and Fusions

The American Academy of Orthopaedic Surgeons indicates that the total number of disc procedures (partial or complete excisions) in 1994 was 317,000. The total number of spinal fusions in 1994 was estimated at 163,000 (41).

Low Back Pain in Teenagers

By the ages of 18 (girls) and 20 (boys) years more than 50% of teenagers had experienced at least one low back pain episode. A general tendency was seen for more women to report low back pain than men, but this difference generally was not statistically significant. The study of the causes and prevention of low back pain needs to be focused on childhood and adolescence (43).

Cost of Low Back Pain Care Increased in Three Years

The term "chronic pain syndrome" has come to mean pain persisting for at least 3 months. Liberty Mutual Insurance Company reported that the mean cost per claim attributed to the handling of industrial low back pain in 1986 and 1989 increased by more than $1500 to $8321 in that period. The me-

dian claim costs changed little, however, increasing from \$391 to \$396. The single major reason for a substantially increased mean cost and an unchanged median cost is that low back pain costs became more abnormally distributed, with a relatively small number of higher-cost cases accounting for a greater amount of the cost. These generally chronic cases have high rates of hospitalization, surgery, attorney involvement, and disability (44).

Medical School Sees More Low Back Pain Patients Than a Chiropractic College

Collaborative research by The National College of Chiropractic and Loyola University Stritch School of Medicine on "Biomechanics of Low Back Flexion-Distraction Therapy" sought to evaluate the similarities and differences between chiropractic and allopathic patients because of a conception that patients who elect chiropractic care may not be representative of the general population. Thirty-six percent of the 380 National College of Chiropractic Center patients who were screened had a primary complaint of low back pain, whereas 58% of the 309 Loyola patients reported this complaint. Another interesting finding was that 45% of Loyola patients screened reported previously receiving chiropractic care (45).

Medical Students' Physical Diagnostic Skill to be Enhanced

Nearly 1500 students from eight New York City medical schools are assessed annually at the Morchand Center for Clinical Competence at the Mount Sinai School of Medicine, and examination results commonly show weaknesses in the area of physical diagnosis. For the class of 1997, for example, in a standardized patient with shortness of breath, chest pain, and possible aortic dissection, only 4% of 1026 examinees evaluated blood pressures in both upper extremities, 50% auscultated the lungs, and 10% percussed the posterior lung fields.

Mount Sinai is planning to formally incorporate physical diagnosis teaching skills into the third-year medical school curriculum (46).

TREATMENT

Healing Time for Low Back Pain

Less Than 50% of Low Back Pain Sufferers Are Pain Free in 4 Years

Of 151 patients interviewed 4 years after reporting low back pain, only 21% of the responding patients said they had been pain free during the follow-up period. Those responding reported one additional episode (7%); two to five episodes (36%); more than six episodes (28%); 12% no recurrence. Less than half of the patients in this survey were without symptoms 4 years after the initial low back pain episode that propelled them into the treatment (47).

Most Back Pain Does Not Resolve Within Six Weeks

The natural history of back pain has been stated to show 90% of cases resolving within 6 weeks; however, studies do not support this. At 3 months, approximately 27% of patients are completely better, 28% improved, 30% had no change, and 14% are worse or much worse (48).

Disc degeneration and protrusion are associated with pressure on the epidural venous plexus with venous dilation. This venous obstruction causes edema of the nerve root. This bears a significant relation to restriction of straight leg raising. Fibrosis develops around and within the nerve root, and a statistical relationship is found between the degree of perineural fibrosis and the degree of venous dilation, which, in turn, shows a significant relationship to neuronal degeneration. An important relationship is found between degenerative disc disease with atherosclerosis and arterial stenosis (48).

A controlled study showed a decrease in PLA_2 activity in degenerated herniated nuclear material compared with healthy disc (48).

The complex regional pain syndrome (CRPS) includes both sympathetically maintained pain (CRPS-I) and causalgia (CRPS-II). Both can be associated with pain and paresthesia, hyperpathia and allodynia, and vasomotor changes and sensitivity to cold.

Increased dorsal horn activity occurs with prolonged periods of discharge after the nociceptive stimulation has been withdrawn. This means that the pain patient may continue to feel pain long after the physical cause of the pain has healed. The dorsal horn cells develop increased sensitivity to afferent impulses. This can lead to the patient experiencing disproportionate pain compared with the evidence of tissue damage and tenderness peripherally, giving rise to the phenomena of hyperpathia and allodynia (48).

First Time Back Pain in Men Continued at One Year

A cohort of 76 men experiencing their first episode of back pain was assessed prospectively at 2, 6, and 12 months following pain onset. At both 6 and 12 months after pain onset, most (78% and 72%, respectively) of the men in the sample continued to experience pain. Many also experienced marked disability at 6 months (26%) and 12 months (14%). At 12 months, no participants had worsened pain relative to the 2-month baseline. The clinical course of first onset back pain may be prolonged for many patients, and it involves a continuum of related disability and distress (49).

Low Back Pain Subtypes

Of 213 patients evaluated for low back pain 72% had acute pain (< 3 months) and only 15% had work-related injury. The patients fell into the following subtypes: acute low back strain (32%); radicular syndrome (28%); chronic back strain (14%); sacroiliac syndromes (10%); posterior facet syndrome (6%);

and 12 different syndromes (10%). Only about 10% had more than one clinical syndrome. Fifty-six percent of the patients were female; 93% were between the ages of 18 and 65 years; and 15% had a work-related injury. The average wait from initial visit to physical therapy was 2.5 weeks (50).

Fear of Work Effect

If back-injured workers have inappropriate beliefs about the nature of their problem and its relation to work, they will develop fear-avoidance behaviors in relation to work because of inadequate pain-coping strategies. They then begin to function in a disadvantageous manner and drift into chronic disability. Once a worker has developed back pain, it would seem that therapeutic programs combining physical challenges to the back, together with operant conditioning, organizational changes, (particularly involving management), and education are more successful than the traditional approaches involving rest and work restrictions (51).

Poorer Health Reported in Low Back Pain Patients

The annual prevalence of low back pain is 48% with 24% of patients consulting their primary care physician and 17% referred to a hospital specialist. Activities of daily living are restricted in less than half and few take sick leave. The general health status of those reporting recent low back pain was significantly poorer than those not reporting low back pain. Most felt that low back pain was self-limiting and would not consult health professionals for future episodes (52).

CHIROPRACTIC RESULTS AND OPINIONS

Chiropractic Physicians See 40% of Back Pain Patients

Patients are seeking alternative care in record numbers. Organized medicine has been slow to embrace these concepts, and this has additionally led to the public perception that medical physicians are not interested in their well-being but only in their fees.

Analysis of the spine care delivery system in the United States reveals two parallel systems. The first is the traditional medical model, servicing 60% of the market. This model has relied on bed rest, hospitalization, drugs, and surgery. The second is the chiropractic model, servicing approximately 40% of the spine market. The chiropractic model has received high patient satisfaction, but questionable health and economic outcomes (53). The density of chiropractors in the United States is 22 per 100,000 population (42).

Chiropractic Care of Chronic Pelvic Pain

Nineteen volunteer female subjects meeting inclusion–exclusion criteria for chiropractic treatment of chronic pelvic pain were treated for 6 weeks with flexion distraction and trigger point techniques. Eighteen subjects completed the study, with an attrition rate of 5%. This was a first step in designing a randomized clinical trial and it showed that the chiropractic treatment has positive short-term effects (54).

10% of Low Back Pain Claims Are 86% of Total Cost

A disproportionately small percentage of the costliest low back pain claims (10%) was responsible for a large percentage of total costs (86%), according to the Liberty Mutual Research Center. After 1 year of disability, the probability of being off disability at the end of the second year is 40%; 12.4% of claims that extended beyond 3 months accounted for 88% of total costs.

Workers off work for 1 month show approximately 50% will still be on Worker's Compensation at 6 months. For disability claims lasting a month or less, 79% of claim expenses represents medical costs and only 16% indemnity for lost time. For claims of a year or more, medical costs represented 29% of costs and indemnity for lost time, 67% of costs (55).

L3–L4 Manipulation Effect on Quadriceps Muscle

Manipulation to the L3–L4 motion segment resulted in a statistically significant short-term increase in quadriceps femoris muscle strength (56).

Malpractice Cause in Chiropractic

Disc problems are by far the most prevalent cause of malpractice suits against chiropractors (29% in 1990, 26.8% in 1995). Most disc problems occurring in 1995 involved the lumbar region (13.8%), followed closely by 12.2% in the cervical region. According to Harrison, "Research shows that numerous malpractice claims have arisen through a technique originally known as the 'million dollar roll' and in later years as the 'side-posture lumbopelvic adjustment." Most of the cases involving the side posture technique were reported to follow a pattern, in which patients suffered from long-standing back pain extending into one leg. Litigation records reveal a general tendency to increase the intensity and frequency of adjustments when patients have increasing pain. Cassidy et al. state that "the treatment of lumbar disk herniation by side posture manipulation is both safe and effective." Slosberg, however, points out:

> *While it's true, as Cassidy et al. mention, that Farfan et al. found that normal disks withstood an average of 22.6° of rotation before failure and degenerated discs an average of 14.3°, these large numbers refer to "ultimate failure" of the intervertebral joint. This means that the involved joint no longer offers any resistance to torsion. Farfan et al. commented that it may take many less degrees of rotation to initiate injury.*

Farfan et al. noted that:

> *. . . an intervertebral joint may be injured by rotation within the small range of normal . . . the derived torque-rotation curves showed a break at 2° to 3° de-*

grees of rotation which could be the result of anular injury; and the response to repeated loading indicated some change, possible injury at less than 3° of rotation. All of these findings suggest that the intervertebral joint may sustain injury at degrees of rotation of 2° to 5° (57).

Distraction Adjustment Relieves Herniated Lumbar Disc

Flexion-distraction manipulation is a treatment developed by James M. Cox. It is often used for lumbar disc injuries (herniation, bulges, and so forth), and for other low back and lower extremity radicular conditions. The technique involves the use of a specialized table that allows for passive distraction, flexion, lateral bending, and rotation. These different planes of motion, along with the use of appropriate adjunctive therapy and exercises, allow for reduction of symptoms attributable to lumbar disc syndromes. Contraindications and indications for flexion-distraction manipulation have been identified and enumerated.

Flexion-distraction manipulation is a treatment that should be investigated as a part of the algorithm for presurgical therapies of lumbar intervertebral disc injuries. This alternative in conservative care may be of benefit to many patients. The surgical option for treating intervertebral disc herniations might be reduced with propagation of flexion-distraction manipulation (58).

Patients Prefer Chiropractic Care

Patients who "cross over" between providers for multiple episodes of low back pain are more likely to return to chiropractic providers, which suggests that chronic, recurrent low back pain cases may gravitate to chiropractic care over time.

The cost-effectiveness of chiropractic care over single episodes as well as over long periods is particularly apparent when more comprehensive measures of costs and outcomes are analyzed. Chiropractic patients have lower overall rates of usage, especially of hospital care. When combined with strong evidence of reported high levels of patient satisfaction, it is difficult to ignore the potential of chiropractic in this nation's search for strategies to help contain costs while maintaining high levels of patient satisfaction (59).

Otitis Media Resolved with Chiropractic Care

The average number of adjustments administered for otitis media by types were acute otitis media (n = 127; 4.0 ± 1.03), chronic or serious otitis media (n = 104; 5.0 ± 1.53), mixed type of bilateral otitis media (n = 10; 5.3 ± 1.35), and in instances where no otitis was initially detected on otoscopic and tympanographic examination, but with a history of multiple bouts (n = 74; 5.88 ± 1.87). The number of days it took to normalize the otoscopic examination was as follows: for acute 6.67 ± 1.9, chronic or serious 8.57 ± 1.96, and mixed 8.3 ± 1.00. The number of days it took to normalize the tympanographic examination: acute 8.35 ± 2.88, chronic or serious 10.18 ± 3.39, and mixed 10.9 ± 2.02. The overall recurrence rate over a 6-month period from initial presentation in the office was for acute 11.02%, chronic or serious 16.34%, for mixed 30%, and for none present 17.56%. The results of this study indicate that a strong correlation exists between the chiropractic adjustment and the resolution of otitis media in children (60).

Improvement Found in Patients with Asthma

A self-reported asthma-related impairment study was conducted on 81 children under chiropractic care. Practitioners, representing a general range of six different approaches to vertebral subluxation correction, administered a specifically designed asthma impairment questionnaire at the appropriate intervals with parents or guardians or older subjects to self-report perceptions of impairment. Significantly lower impairment rating scores (improvement) were reported for 90.1% of subjects after 60 days of chiropractic care when compared with the prechiropractic scores. No significant differences were found across the age groups based on parent or guardian versus self-rated scores. Girls reported higher (less improvement) before and after care compared with boys, which suggests greater clinical effect for boys. Additionally, 25 of 81 subjects (30.9%) voluntarily decreased their dosage of medication by an average of 66.5% while under chiropractic care. Moreover, information collected from patients revealed that among 24 patients reporting asthma attacks in the 30-day period prior to the study, the number of attacks decreased significantly by an average of 44.9% ($P < 0.05$). Based on the data obtained in this study, it was concluded that chiropractic care, for correction of vertebral subluxation, is a safe, nonpharmaceutical health care approach that may also be associated with significant decreases in asthma-related impairment as well as a decreased incidence of asthmatic "attacks." The findings suggest that chiropractic care should be further investigated relative to providing the most efficacious care management regimen for pediatric asthmatics (61).

POOR OUTCOME WITH PEDICLE SCREW FUSION

Only one of seven randomized trials favors pedicle screws in posterolateral fusion surgery (62). Surgical results of 76 patients undergoing decompression and single level fusion showed that instrumentation did not improve patient outcomes (63). Results of 83 candidates for one- or two-level posterolateral fusion randomized to instrumented or noninstrumented groups (2-year follow-up) showed no differences in outcome or fusion rate between instrumented and noninstrumented groups (64). No significant difference in pain levels was found in 45 patients who were randomized to decompression, decompression with uninstrumented posterolateral fusion, or decompression with instrumented fusion (minimum 2-year follow-up) (65). No difference in fusion rate was seen between instrumented and noninstrumented groups when 27 patients

underwent L5 laminectomy and nerve root decompression (66). Instrumentation did not improve functional outcome, pain, or fusion rate; groups did better with fusion than did those with physical therapy when 67 patients were randomized to noninstrumented fusion, instrumented fusion, or physical therapy (2-year follow-up) (67). Functional outcomes and fusion rates were similar in both groups of 110 candidates (single-level posterolateral instrumented or noninstrumented fusion): a slight advantage in one subgroup of instrumented patients undergoing supplementary neural decompression was reported (68). At the 1-year follow-up, the clinical outcomes and fusion rates were superior in the rigid pedicle screw group of 124 patients undergoing posterolateral fusion (uninstrumented, with semirigid pedicle screw, or rigid pedicle screw). This study, positive for pedicle screw instrumentation, was quasirandomized as the randomization protocol was broken because of bone quality problems in patients (69).

EPIDURAL STEROID INJECTION RESULTS

Epidural steroid injection treatment offers no significant functional benefit, nor does it reduce the need for surgery. Only about a third of patients report marked improvement after a single epidural steroid injection (70). Some believe that epidural injections are a valuable weapon in the sciatica treatment arsenal. Others believe they have a positive effect on the natural course of sciatica. Another common premise for injection treatment is that epidural steroids help patients avoid surgery.

Epidural steroid injections provide only short-term improvement in leg pain and sensory deficits (70). Researchers randomly allocated patients ($n = 158$) with sciatica of 1 month's duration to receive an injection of either 2 mL of methylprednisolone (mixed with 8 mL isotonic saline) or saline alone (1 mL isotonic saline). The subjects received one, two, or three injections of the steroid or placebo over a 6-week period. They were allowed another injection at 3 and/or 6 weeks if they did not experience marked improvement from the first injection—and if they still had Oswestry Disability scores above 20.

At 3 weeks, the Oswestry Disability score had improved by a mean of -8 in the methylprednisolone group and -5.5 in the placebo group. By 6 weeks, the only difference between the two groups was greater improvement in leg pain in the steroid group. By 3 months, no longer was any significant difference found between the treatment groups.

The surgical rate in the two groups was virtually identical at 1-year follow-up. At 12 months, the cumulative probability of back surgery was 25.8% in the methylprednisolone group and 24.8% in the placebo group (70).

Epidural Steroid Injections May Retard Disc Fragment Absorption

An intriguing animal study from Japan suggests that the injection of epidural steroids to quiet inflammation around a painful disc herniation may have an unwanted side effect: the steroids may retard the resorption of the herniated disc tissue. Consequently, treating inflammation may be a two-edged sword, if this study can be applied to the human situation (71).

NSAID DANGER

Upper Gastrointestinal Bleeding Persists After Discontinuing Use

A common view of NSAID-related toxicity is that the risk of serious adverse gastrointestinal (GI) problems is highest in the early days of therapy and then wanes. Two new studies challenge this conventional wisdom. According to a new cohort study of 52,293 subjects in Tayside, Scotland, the risk of hospitalization for a serious upper GI event was absolutely constant over a period of 2 years. NSAID toxicity persists with continuous exposure. Risk for upper GI bleeding persisted after patients stopped taking NSAIDs.

Serious NSAID-related gastropathy did not decrease with time on the drug and serious events occurred without warning. The relative risk of serious NSAID-related gastropathy is similar for both NSAIDs and steroids (prednisone), but the combination of NSAID and prednisone therapy more than doubles the risk (72).

STENOSIS

Surgical Success

Success rates of surgical intervention for lumbar spinal stenosis vary, and few preoperative factors have been found to be significantly correlated to surgical outcome.

A total of 438 patients (183 women, 255 men) who underwent decompressive surgery for lumbar spinal stenosis were re-examined and evaluated for outcome 4.3 years after surgery. Outcome was based on subjective disability, which was assessed using the Oswestry Disability questionnaire.

The proportion of good to excellent outcomes of all 438 patients was 62% (women, 57%; men, 65%). Diabetes, hip joint arthrosis, and preoperative fracture of the lumbar spine seemed to be associated with poor outcome. The ability to work before or after surgery and a history of no prior back surgery were predictive of good outcome. Results suggest that clear myelographic stenosis and no prior surgical intervention, no comorbidity of diabetes, no hip joint arthrosis, and no preoperative fracture of the lumbar spine are associated with a good outcome in surgical management of lumbar spinal stenosis (73).

LESS DISC REDUCTION IN POOR CLINICAL OUTCOMES

Forty-eight patients with unilateral radiculopathy were studied: 94% with positive tension signs and 38% exhibiting muscle weakness corresponding to the symptomatic nerve root. In 17 of 22 patients, the enhanced area gradually thickened and intruded into the migrated disc material as the size of the herni-

ated nucleus pulposus decreased; the herniated nucleus pulposus disappeared in 9 and showed a marked decrease in 7 patients. These sciatica cases had a good clinical course. In the other 5 patients in whom no changes in the enhanced area resulted, less of a tendency was seen for the herniated nucleus pulposus to decrease in size and clinical results were poorer (74).

BACK BRACE EFFECTIVENESS QUESTIONED

A study of back pain treatment showed limited evidence that exercise has some effect in preventing back pain and that education is not effective. No conclusive evidence was found for or against the effectiveness of lumbar supports (75). One low-quality randomized trial reported a positive effect of wearing lumbar supports (76). Two low-quality randomized controlled trials found no effect (77). No evidence for or against the effect of lumbar supports currently exists because of the contradictory outcomes of the studies (78).

OSTEOPOROSIS

Osteoporosis is a skeletal condition characterized by decreased density (mass/volume) of normally mineralized bone. Reduced bone density leads to decreased mechanical strength, thus making the skeleton more likely to fracture. Postmenopausal osteoporosis (type I) and age-related osteoporosis (type II) are the most common primary forms of bone loss seen in clinical practice. Secondary causes of osteoporosis include hypercortisolism, hyperthyroidism, hyperparathyroidism, alcohol abuse, and immobilization. In the development of osteoporosis, often a long latency period precedes the main clinical manifestation, pathologic fractures. The earliest symptom of osteoporosis is often an episode of acute back pain caused by a pathologic vertebral compression fracture, or an episode of groin or thigh pain caused by a pathologic hip fracture. In the diagnostic process, the extent and severity of bone loss are evaluated and secondary forms of bone loss are excluded. A careful diagnostic workup that includes clinical history, physical examination, laboratory evaluation, bone densitometry, and radiographic imaging allows the clinician to determine the cause of osteoporosis and to institute medical interventions to stabilize and even reverse this frequently preventable condition.

Bone Mineral Density Values

The World Health Organization has established diagnostic criteria for osteoporosis that are based on bone mineral density (BMD) measurements determined by dual-energy x-ray absorptiometry (DXA). A patient is classified as having low bone mass if the BMD measures between 1 and 2.5 standard deviations (SD) below the mean value in a young reference population. The diagnosis of osteoporosis is made if a patient's bone density is 2.5 SD or more below the mean for young normal people.

Postmenopausal (type I) osteoporosis develops in women who have estrogen deficiency, whereas age-related (type II) osteoporosis occurs in men and women as their bone density decreases with aging.

Osteoporosis and osteomalacia are commonly confused osteogenic conditions in adults. Whereas osteoporosis is characterized by a decreased density of normally mineralized bone matrix, osteomalacia is a qualitative rather than a quantitative disorder of bone metabolism. In osteomalacia, bone density can be increased, normal, or (most commonly) decreased, and bone matrix is insufficiently mineralized (79).

Approximately 30% of postmenopausal white women in the United States have osteoporosis, and 16% have osteoporosis of the lumbar spine in particular. Spinal bone density is positively associated with greater height and weight, older age at menopause, a history of arthritis, more physical activity, moderate use of alcoholic beverages, diuretic treatment, and current estrogen replacement therapy, whereas later age at menarche and a maternal history of fracture are associated with lower levels of density. Low bone density leads to an increased risk of osteoporotic fractures. Fracture risk also increases with age. Vertebral fractures affect approximately 25% of postmenopausal women, although the exact figure depends on the definition used. Recent data show that vertebral fracture rates are as great in men as in women but, because women live longer, the lifetime risk of a vertebral fracture from age 50 onward is 16% in white women and only 5% in white men. Fracture rates are less in most nonwhite populations, but vertebral fractures are as common in Asian women as in those of European heritage. Other risk factors for vertebral fractures, which are less clear, include hypogonadism and secondary osteoporosis. Obesity is protective of fractures as it is of bone loss. Compared with hip fractures, vertebral fractures are less disabling and less expensive, costing approximately $746 million in the United States in 1995. However, they have a substantial negative impact on the patient's function and quality of life. The adverse effects of osteoporotic fractures are likely to increase in the future with the growing number of elderly people (80).

Up to 20% of patients die in the year after a hip fracture. Only approximately 33% of survivors regain the level of function that they had before the hip fracture. Although most vertebral fractures are not medically attended but are found incidentally on a radiograph taken for some other purpose, acute fractures can be painful. These fractures can lead to progressive loss of height, kyphosis, postural changes, and persistent pain that interferes with the activities of daily living; these difficulties, however, are mostly confined to those with severe or multiple vertebral deformities. The adverse impact of vertebral fractures on most of the activities of daily living is approximately as great as that seen with hip fractures. Only 4% of patients with a vertebral fracture become completely dependent because of the fracture, but the negative emotional impact of vertebral fractures may be an even more important determinant of reduced quality of life. Depression increases the risk of osteoporotic fractures by 40% among women over the age of 65 (83).

The total cost of fractures may be as much as $20 billion per year in the United States. These costs are likely to rise in the future as the number of elderly people increases (80).

Osteoporosis Pain in Young Men and Women

Bone mineral density studies are suggested for young adults in whom no clear underlying cause for low back pain is found. Thirteen female and seven male patients (mean age, 38.5 years) who had persistent low back pain but whose only obvious clinical or radiologic abnormality was evidence of bone demineralization on lumbar radiograph showed significantly diminished BMD of the lumbar spine on dual photon absorptiometry. Seven of the patients were ultimately classified as having osteoporosis, with BMD greater than 2.5 SD below the young adult reference point. The remaining patients were determined to be osteopenic, with BMD greater than 1 SD below the normal benchmark (82). Biochemically, bone-specific alkaline phosphatase and osteocalcin in serum are the best markers for bone formation (81).

Osteoporosis Is Treated in the Teenage Years

Calcium intake is critical in the teenage years. Low calcium intake during adolescence has been associated with the eventual onset of osteoporosis in elderly women. Young girls retain more than four times as much calcium as women only a few years older.

A total of 14 girls and 11 women signed up for "Camp Calcium," Purdue University's supervised diet program. Each participant received exactly 1332 mg of calcium per day. The results: Daily calcium retention for the adolescents was significantly higher than adult levels (326 mg compared with 73 mg).

Eighty percent of bone density is genetically determined, and an individual can only influence about 20%. But it is worth working on because a 5% increase in bone mass corresponds to about a 40% decrease in fracture risk. The National Institutes of Health report that young women ages 12 to 19 years consume well below the optimal recommended levels of calcium. Young girls can decide today their quality of life when they are older: "Picture what you want to look like in 70 years. Do you want to be jogging or in a wheelchair?" (84).

43% of Women Show 60% Low Calcium Intake

In a University of Illinois study, 43.2% of women surveyed reported calcium intake below 60% of their recommended daily allowance—and they could be considered calcium-deficient. It is a crisis of national important. Further, roughly 25% of calcium-deficient women are not even aware of the fact. Many contributing factors are given for low calcium intake, and gastrointestinal distress is a major one (85).

Morbidity of Osteoporosis

Osteoporosis is also an important cause of death among elderly people. Hip fractures related to osteoporosis result in death in up to 20% of cases. *Women's mortality rates from osteoporosis-related fractures are greater than the combined mortality rates from cancers of the breast and ovaries* (86). The number of osteoporotic fractures is growing faster than the number of elderly people in the population. Fracture treatment represents the most substantial direct cost of osteoporosis to the health care system. The annual cost of all fracture treatment in Canada is about \$1 billion (86).

Osteoporosis Predisposition and Treatment

Osteoporosis is a major health and economic problem. One of four women and one of eight men aged more than 50 years are believed to have osteoporosis. Osteoporosis increases in prevalence with age in both sexes. An estimated 1.4 million Canadians are affected by osteoporosis.

The US National Osteoporosis Foundation has estimated that 70% of hip fractures are the result of osteoporosis. More than 21,000 hip fractures were estimated to be related to osteoporosis in Canada in 1993.

Women's mortality rates from osteoporotic fractures are greater than the combined mortality rates from cancers of the breast and ovaries. Up to 20% of women and 34% of men who fracture a hip die in less than 1 year.

Certain conditions predispose to loss of bone and increased risk for osteoporotic fractures. Patients to target for investigation include the following (86, 87):

- Women who have had an early menopause (aged 40 to 45 years), premature menopause (before age 40), or bilateral oophorectomy before normal menopause (aged 45 to 55 years).
- Younger women who have amenorrhea or oligomenorrhea because of ovarian hormone deficiency states, eating disorders, stress, excessive or competitive exercise, hyperprolactinemia).
- Women not receiving ovarian hormone therapy (OHT) for at least 5 years after menopause. These women are thought to be at increased risk of osteoporotic fracture as a result of the accelerated rate of bone loss that occurs postmenopausally.
- Patients expected to undergo prolonged treatment (i.e., more than 3 months) with oral glucocorticoids.
- Patients with primary hyperparathyroidism.
- Patients with a strong family history of osteoporosis.
- Postchemotherapy patients (especially those with breast and hematologic cancers).
- Men who have hypogonadism for any reason.

Nutritional Supplementation

Calcium: The Osteoporosis Society of Canada (OSC) currently recommends that adults obtain 1000 to 1500 mg of elemental calcium per day for optimal bone health.

Vitamin D: Vitamin D increases calcium absorption in the GI tract. Current recommended nutritional intake for vitamin D is 200 IU in adults aged 50 years and older. The OSC recommends that people over 65 or those with osteoporosis have a dietary intake of 400 to 800 IU per day (87).

Chondroitin Sulfate Prevents Osteoarthritis of Finger Joints

A significant decrease was seen in the number of patients with new "erosive" osteoarthritic finger joints receiving chondroitin sulfate: 8.8% of the patients developed "erosive" joints, whereas in the untreated group, 29.4% of the patients developed erosive arthritis. Chondroitin sulfate prevents "erosive" osteoarthritis in the finger joints (88).

Cartilage Rehabilitation

Polysulfated polysaccharides significantly increase the synthesis rates and the immobilization of accumulation of aggrecan in the extracellular environment in vivo in osteoarthritic joints. This may lead to structural improvement of articular cartilage and to a retardation of disease progression (89).

Calcium Plus Vitamin D Daily for Osteoporosis

Participants (848 healthy, ambulatory men and women aged 65 years or older) were randomized in a double-blind fashion to take daily supplements of calcium (500 mg) and vitamin D (700 IU) or placebo. The primary outcomes measured were BMD and nonvertebral fractures. Secondary outcomes included several biochemical measures of bone metabolism.

A statistically significant improvement was noted in total body BMD in both male and female groups compared with placebo. Separate measurements of the femoral neck and spine, however, failed to show any significant improvement in BMD in women. Patients treated with calcium and vitamin D also had greater improvements in a number of biochemical markers of bone metabolism. There were 37 total fractures during the study period, including 11 in the treatment group compared with 26 in the placebo group.

Recommendations for clinical practice: A clinically relevant reduction in nonvertebral fracture rates can be achieved using calcium (500 mg daily) and vitamin D supplementation (700 IU daily) in the outpatient setting in elderly, white women not currently taking estrogen replacement (90).

Men Develop Vertebral Fracture As Often As Women

More than 20% of white women aged more than 50 years have osteoporosis of the hip. The prevalence of osteoporosis of the lumbar spine in women older than age 50 is approximately 16%. The lifetime risk of any fracture among white women from age 50 onward approaches 75%. The lifetime risk of a hip fracture is 17% in white women and approximately 6% in white men. The lifetime risk of a clinically evident vertebral fracture is approximately 16% among white women.

Vertebral fractures are as frequent in men as in women. Despite the widespread belief that osteoporosis is a disorder of women, recent studies from around the world indicate that the prevalence of vertebral fractures is as great in men as in women, affecting approximately one fourth of each group, depending on the definition of vertebral fracture used (91).

Augmentation in Osteoporosis Fractures

In addition to prophylactically stabilizing osteoporotic vertebral bodies at risk for fracture, augmentation of vertebral bodies that have already fractured may prove to be useful by reducing pain, improving function, and preventing further collapse and deformity.

A number of products are now available or are in clinical trials. The most promising products are injectable materials—polymethylmethacrylate or mineral bone cement. The early clinical results using polymethylmethacrylate in percutaneous vertebroplasty for fractured vertebral bodies and the results in vitro using an injectable mineral cement for vertebral body fortification are encouraging. Although the principle of vertebral body augmentation remains encouraging, data to support the widespread use of these techniques remain sparse, and the indications for their use should be more clearly defined (92).

Diagnostic Testing for Osteoporosis

Bone mineral density is an important component of bone strength. Dual-photon absorptiometry (DPA) has largely been replaced with dual-energy x-ray absorptiometry (DXA), which provides a much greater photon flux allowing for shorter examination times, greater precision, improved resolution, and longer source life.

Second-generation DXA machines use a fan-shaped beam of x-rays, which reduces the scan times to a few minutes or less, provide both bone density measurements and high-quality images of spinal morphology that allow morphometric analysis. These machines may have a C-arm configuration, allowing lateral projection with separation of the vertebral body and posterior elements. The radiation dose delivered by DXA is extremely low, making it the best method for measuring BMD, although it is still moderately expensive; currently, it is the best practical method for measuring BMD.

Up to 30 or 50% of trabecular bone must be lost before visible change occurs on radiographs. However, radiographs can reveal the presence of compression fractures and other disease that may or may not be related to osteoporosis.

Certain conditions predispose to bone loss and an increased risk for subsequent osteoporotic fractures. In these cases, bone densitometry tests are clearly indicated for the following reasons: menopause, amenorrhea in a younger woman for any reason, prolonged treatment (more than 3 months) with supraphysiologic doses of glucocorticoids, asymptomatic mild primary hyperparathyroidism, a strong family history of osteoporosis or the presence of other risk factors for osteoporosis, a diagnosis of osteopenia on the strength of a radiologist's interpretation of an x-ray study must be confirmed or denied, a patient has started osteoporosis therapy and the physician wishes to determine whether the treatment has been effective, low

body weight or significant decrease in body weight since the age of 25, poor vision, alcoholism, amenorrhea, anorexia or poor dietary habits, inability to rise from a chair to standing without using arms, and white or Asian descent (93).

Dual-energy x-ray absorptiometry is usually used to study the hip and the lumbar spine. Standard spine DXA analysis includes values obtained from L1 to L4 and a total value for the four sites combined. For each site and for the total, the area analyzed (expressed in square centimeters), bone mineral content (expressed in grams), and BMD (expressed in grams per square centimeters) are reported. In the normal person, the area, bone mineral content, and BMD should progressively increase from L1 to L4. Sites that do not follow this orderly progression should probably be eliminated from the analysis; however, the use of only one or two sites will reduce the accuracy of the test. It is important that identical sites be used for serial examinations.

In addition, a BMD value is reported for Ward's triangle, a region that reflects the cancellous bone found between the stress trabeculae in the femoral neck.

Traditionally, evaluation of the spine with DXA has been done in the posteroanterior direction. The measurement thus includes all tissues anterior and posterior to the vertebral bodies, and several artifacts can be introduced. Prevertebral vascular calcifications, discogenic sclerosis from degenerative disc disease, and osteophyte formation from facet osteoarthritis will all falsely raise the measured bone density.

In an effort to improve the accuracy of the DXA in the spine, lateral scanning techniques have been developed. These are performed with the patient supine (not in the lateral decubitus position), rotating the C-arm 90° to the side of the patient. Analysis thus excludes prevertebral vascular calcifications, end plate osteophytic spurs, and the posterior elements. This technique is associated with inherent errors. In the patient with scoliosis, differentiation between vertebra may be impossible. An overlap of the iliac wing with L4 is seen in 14% of patients, and the ribs overlap the L2 body in essentially all patients.

Quantitative ultrasound is a method that has only recently begun to receive widespread attention in the United States. This method usually evaluates the calcaneus, incorporating two ultrasound transducers that are positioned opposite each other (94).

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FINITE ELEMENT MODELING

Mathematical models of the spine are invaluable in (*a*) explaining the behavior of the spine in health and injury, (*b*) making predictions, and (*c*) suggesting theories or explanations for underlying diseases and treatment effectiveness. Developing these models by computer is necessary because of the technical difficulties of conducting in vivo experiments on humans. The unusual complexity of the spine structure demands a stepwise approach (i.e., at each step of the development); the prediction should be validated in terms of those parameters amenable to experimental measurement. Finite element analysis has been widely used in predicting the states of stress and disc bulges in the lumbar spine.

By far the most comprehensive finite element model was developed by Shirazi-Adl et al. (1–5) for L2–L3 motion segments. This model considered the detail geometry as well as the geometric and material nonlinearities associated with the material properties of the ground substance and the anular fibers of the disc. Using this model, the biomechanical responses were predicted under simple as well as combined loading conditions that would be encountered in physiologic activities. Ueno and Liu (6), using similar modeling technique and a commercially available software package (ANSYS), reported on the L4–L5 joint response under axial torsional load. Using the Shirazi-Adl et al. (1) concept of the anulus, Rao and Dumas (7) developed a simpler axisymmetric model of L5–S1 vertebra-disc-vertebra to conduct a parametric study on the material properties and biomechanical response of the spine under compressive loading. From these model studies, it is clear that the development of detailed finite element models representing the lumbar segments and adding studies of biomechanical treatment modalities (e.g., flexion-distraction) represents new and practical expansion of the efforts that will have immediate application in the clinical management of back pain. The following finite element study on an L2–L3 motion segment under traction load is a first step in that direction.

Model Development

The finite element model presented here was developed at The National College of Chiropractic with the assistance of a graduate student from the University of Illinois-Chicago. An L2–L3 vertebra-disc-vertebra unit was modeled under both compressive and traction loading. Because the loading was in one direction, the deformations here remained symmetrical about both the sagittal and midhorizontal planes. Therefore, only a quarter of the vertebra-disc-vertebra unit needed to be analyzed, thus reducing computation time and cost. Figures 1 and 2 show the finite element grid used in the modeling. ANSYS was the commercially available software used for this study. The software allowed for the modeling of the different materials representing the vertebra cortical shell, cancellous bone, end plates, anulus ground substance, anular fibers, and the nucleus pulposus. The model idealized the vertebrae, end plates, and ground substance as eight-noded, solid elements; anular fibers as two-noded cable elements; and the nucleus as eight-noded incompressible solid elements. The axial elements used to model the fibers of the anulus were arranged in eight layers, in a crisscross pattern, making an angle of about 30 degrees with respect to the horizontal plane of the disc. Thus, the model consisted of 330 eight-node solid elements, 48 incompressible eight-node elements, and 208 two-node cable elements totaling 586 elements and 532 nodes. The geometric dimensions were identical to those of the Shirazi-Adl et al. model (1).

Model Validation

The present model was validated by comparing the load-displacement responses with the results reported by Shirazi-Adl (1). The loading was implemented by means of uniform axial displacement in four increments of 0.2, 0.5, 0.8, and 1.1 mm, respectively. Geometric and material nonlinearity options available in the ANSYS NSTAR program were used for the study. Figures 3 and 4 show the present model response for axial displacement and lateral, anterior, and posterior disc bulge changes as a function of the compressive load. Comparison of the responses with the results reported by Shirazi-Adl shows good agreement between the two models.

Response of Model Under Traction Load

The model was studied by applying six incremental distractive displacements of 0.1, 0.3, 0.5, 0.7, 0.9, and 1.1 mm. Figure 5 shows the load-displacement curve under compression and tension. The results clearly demonstrate that the motion segment is more flexible in tension compared with compressive load. The graph also shows the nonlinear stiffening as the load is increased. Figure 6 shows the predicted disc necking under a tension load of 2300 N. Necking magnitude was found to be maximum at the anterior location, followed by those at lateral locations, and at the posterior location. Figure 7 shows the load-disc necking response in tension as well as the load-disc bulge response in compression. It demonstrates the stiffening behavior of the disc in terms of disc necking, which was similar to the observed response in axial displacement. The disc necking has a less stiffening effect in comparison with compression. An important and dramatic prediction on the disc necking can be seen clearly from Figure 7. At tension loads of 0 to 600 N, the posterior location possesses the greatest necking followed next in order by the anterior and lateral locations. When tension load ranges from about 600 to 1200 N, posterior disc necking is less compared with

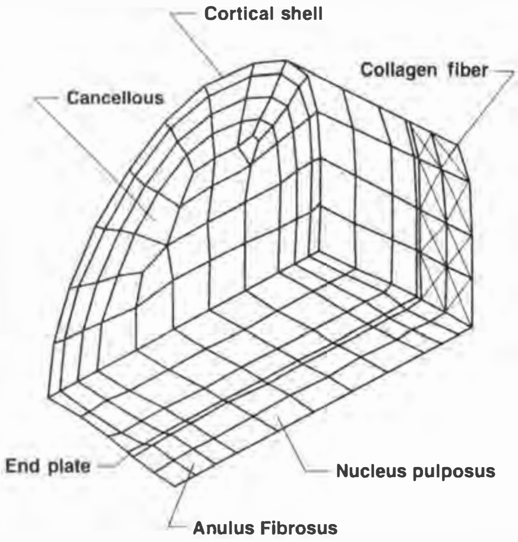

Figure 1. Finite element idealization of a vertebra-disc-vertebra unit.

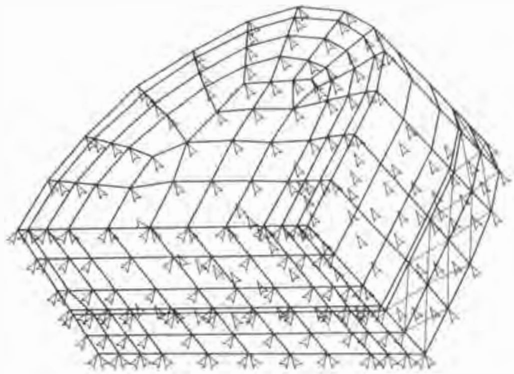

Figure 2. Boundary conditions and loading on the finite element model.

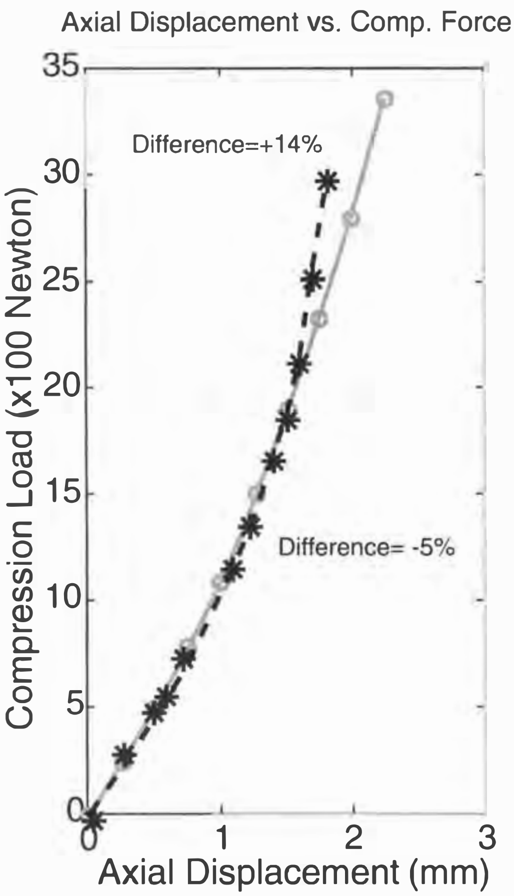

Figure 3. Axial displacement response of the finite element model under compressive load. 0–0, Shirazi-Adl; *— —*, present model.

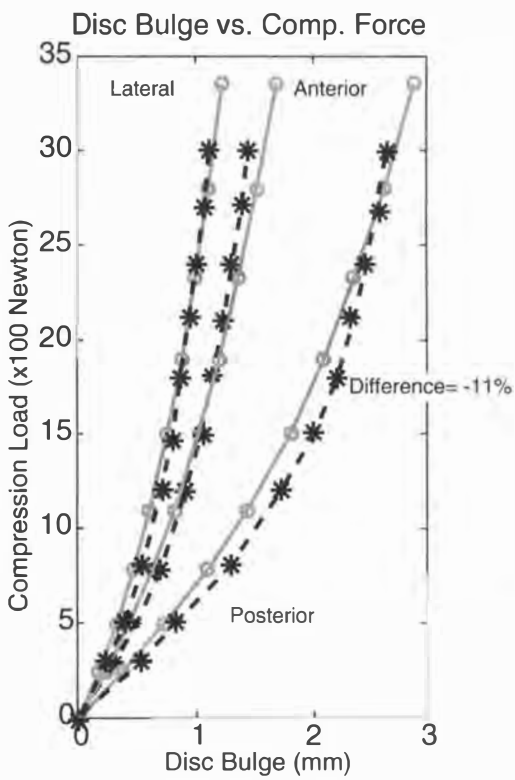

Figure 4. Disc bulge response of the finite element model under compressive load.

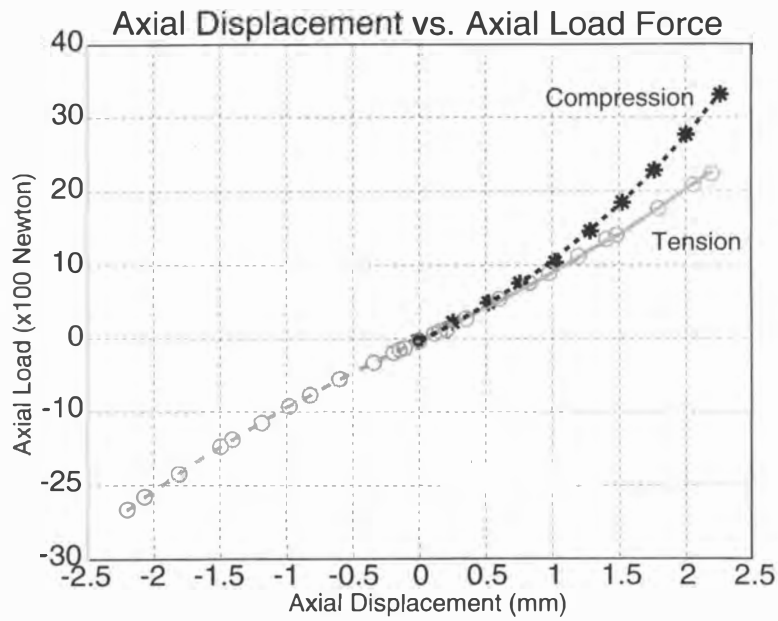

Figure 5. Comparison of the axial displacement response of the model under tension and compressive loads.

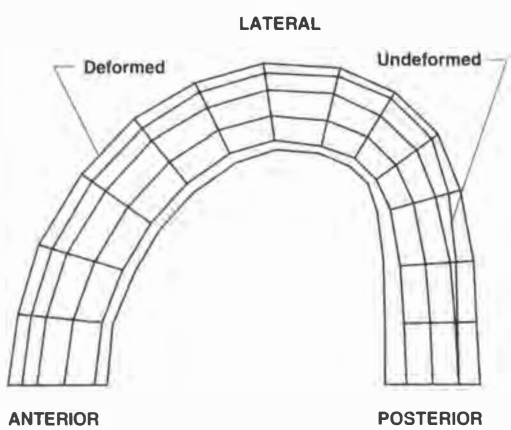

Figure 6. A cross-sectional view of disc necking under tension load of 2300 N.

the anterior; above 1200 N of tension load, it is less than both anterior and lateral disc necking values. The posterior disc necking has the most stiffening effect. This suggests that loads below 600 N of traction are more effective in expanding the canal space for neural elements. Greater than 600 N does not have any added benefit.

In summary, finite element modeling can be a valuable tool to predict the disc changes under distraction conditions. It allows less expensive and noninvasive experimentation to study the spine response under measured loads of flexion-distraction. Parametric variation and pathologies of the disc can be incorporated into the model. In the future, work will be done to address these issues.

INTERVERTEBRAL DISC PRESSURE CHANGES DURING DIFFERENT TABLE MOTIONS

The Cox flexion-distraction procedure involves different maneuvers of the spinal motion segments for treating low back pain. The intradiscal pressure changes during distractive manipulation under the flexion motions of the table were reported earlier in Chapter 8. This addendum to that chapter presents the results for the changes in the intradiscal pressures during flexion, extension, lateral flexion, and circumduction motions of the table.

Miniature pressure transducers (Model SPR-524, Millar Instruments, Houston) were used for this study. An unembalmed cadaver of a deceased 72-year-old man was used for this study. The cadaver was frozen at −20°C within 24 hours after death and thawed at room temperature before experimentation. Some paraspinal musculature was dissected to permit accurate insertion of the needle and pressure transducer. An epidural needle with stylette (17 gauge) was inserted into the nucleus of the disc (L3–L4). Then, the stylette was removed and the miniature pressure transducer inserted so that the pressure was exposed to the nucleus. The disc was pressurized with water using a continuous pipetting outfit connected by flexible tubing to a second needle in the disc. The intradiscal pressures were monitored during the table motions of flexion, extension, lateral flexion, and circumduction. Pressures were monitored during four cycles of table motions. Table 1 lists the mean values of the intradiscal pressures before the treatment cycle and in the extreme position of the table motion. Figure 8 shows the change in the intradiscal pressure variations as a function of treatment duration.

A decrease in intradiscal pressure was observed during the flexion motion of the table. Pressures increased during the

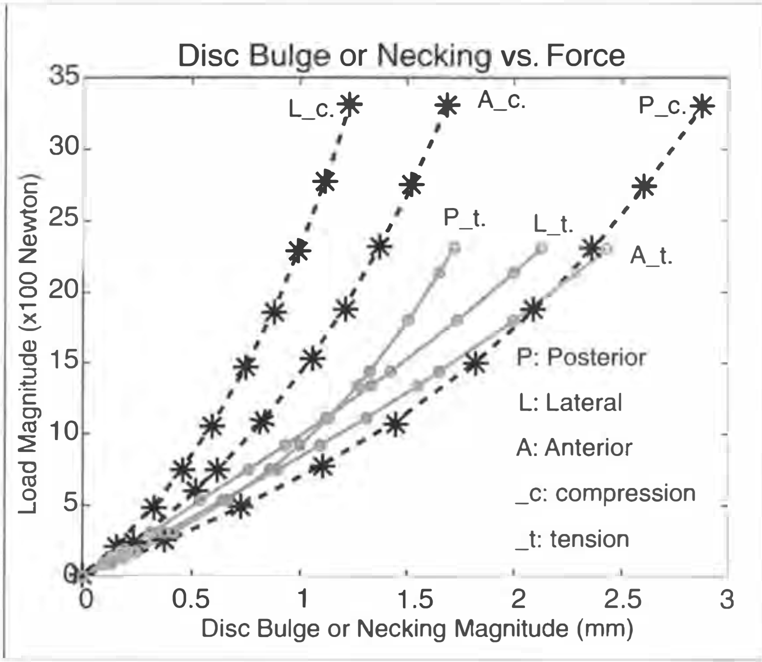

Figure 7. Comparison of disc necking under tension load versus disc bulge under compressive load.

Table 1

Mean Intradiscal Pressures (mm Hg) During Low Back Treatment Procedures (Joint L3–L4)

Table Motion	Pressure in Initial Prone Position	Pressure in Distracted Position	Change in Pressure
Flexion	228	−19.5	247.5
Extension	228	1250	1022
Right lateral flexion	226	747	521
Left lateral flexion	226	−151	377
Right lateral circumduction	240	530	290
Left lateral circumduction	240	−120	360

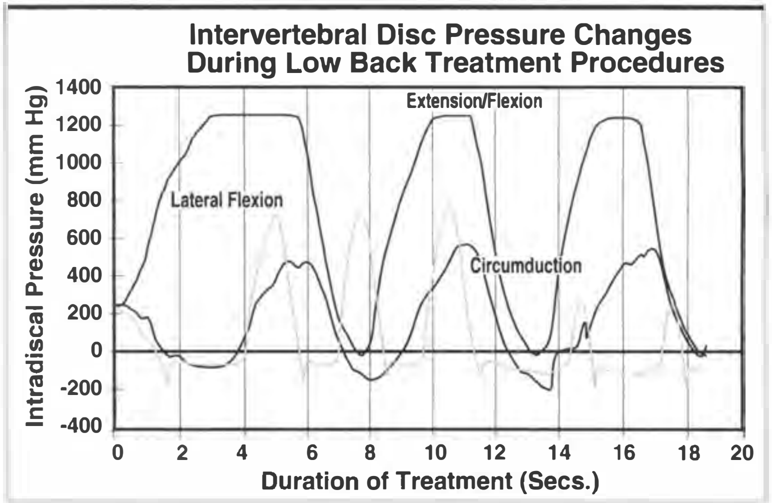

Figure 8. Intradiscal pressure changes during different table motions.

extension motion of the table, particulary during the right lateral motion, whereas the pressures decreased during the left lateral motions of the table. During circumduction, the pressures decreased during the left lateral and flexion motions, whereas they increased during right and flexion combined motions. In all of the motions, the pressures returned to their original values when the spine was brought back to the initial prone position. One reason for the increase and decrease during lateral motions was that the transducer was inserted somewhat right laterally from the center of the disc. The results clearly show that the pressures changed during different therapeutic motions of the table, with the greatest increase observed under extension. Although this study was limited to one cadaver, the results are interesting and additional studies with more cadavers and studies on animals can give further insight into the changes in the pressures at different regions of the spine.

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INDEX

Note: Page numbers in *italics* indicate illustrations; those followed by "t" indicate tables.

Abdominal aneurysm, *483*, 483–484, *484*
Abdominal aortic atherosclerosis, disc disease and, 25, *26*
Abdominal co-contraction, 667, *667*
Abdominal muscles, spinal stability and, 655, *656*
Abdominal pain, spinal origin of, 163
Abdominal pressure, spinal effects of, 40
Achilles reflex, 418t, *420*, *440*, 440–441
Acid phosphatase, 513
  in disc disease, 49, *50*
Acquired immunodeficiency syndrome (AIDS)
  acute lumbosacral polyradiculopathy in, 483
  laboratory evaluation in, 522
Acupressure
  for Cox distraction, 328, *329*
  for transitional segment, *242*
Acupuncture meridian tracing, for Cox distraction, 328
Acute back pain, 310, 377
Acute back sprain, 384, *385*
  Cox distraction for, 311t–313t, *322*, *323*
  diagnosis of, 384, *385*
Acute lumbosacral polyradiculopathy, in AIDS, 483
Adductor muscle, goading of, for Cox distraction, 328, *329*
Adolescents, disc degeneration in, 107
AIDS
  acute lumbosacral polyradiculopathy in, 483
  laboratory evaluation in, 522
Albuminuria, 510–511
Algometry, pressure, 452–454
Alkaline phosphatase, 511, 512t
Allopathic medicine, chiropractic care and, 7–8, 555–556
Amoss' sign, 439, *439*
Amyloid, in disc degeneration, 107
Analgesia, 40, 336–339, 553–554, 699. *See also* Pain management
  complications of, 553, 699
  epidural, 40, 553–554, 699
  facet joint injection for, 33
  for fibromyalgia, 256
Anemia
  in multiple myeloma, 519
  nonsteroidal anti-inflammatory drugs and, 338
Anesthesia, 40, 553–554, 699
  complications of, 553, 699
  spinal, 50, 553–554, 699
    complications of, 553
    for reflex sympathetic dystrophy, 160–161
Aneurysm
  abdominal, *483*, 483–484, *484*
  lateral sacral artery, 476
Aneurysmal bone cyst, 497–498, *498*
Ankle
  dorsiflexion of, 418t, *419*, 439, *439*
  extension of, 418t, *419*, 439, *439*
  plantar flexion of, 439, *439*
Ankle jerk reflex, 418t, *420*, *440*, 440–441
Ankylosing spondylitis, 466, *467*
  C-reactive protein in, 510
  HLA-B27 in, 516, 516t, 521
  laboratory evaluation in, 521–522
Antalgic lean, 56–62, *58–64*, 59, *61*, *63*
  assessment of, 430–431, *431*
  Cox distraction positioning for, 296, *296*
Anterior innominate procedures
  flexion-distraction, 229, *229*
  manipulative-adjustment, 229, *229*
Anterior longitudinal ligaments, nociceptors in, 33
  as pain source, 33, 695
Anterior sacroiliac ligament, 214–215, *216*, *217*
Antidepressants, 337–339
  for fibromyalgia, 256
Antihypertensives, with nonsteroidal anti-inflammatory drugs, 338
Antinuclear antibodies, *514*, 514–515, 515t
Anulus fibrosus, 51–52
  creep load on, 32
  dehydration and cracking of, 66, 383
    growth factors for, 551
  in disc degeneration, 108–109, 279–280
  disc prolapse and, 51, *52–54*, 52–56
  disc protrusion and, *52–54*, 52–56
  injury of, diagnosis of, 446
  movement of
    extension effects on, 278–279
    flexion effects on, 278–279
  nociceptors in, 32
  nucleus pulposus containment in, 69, *72*
  pain sensitivity of, 29–30, 31–33
  as pain source, 29–30, 31–33, 49–51, 82, 379–381
  posterolateral disruption of, 385–386, *386*
  proprioceptors in, 32
  rotational movement restriction and, 82–84, *83–85*
  sequestered, 387, *387*
    displaced, 387–388, *387–390*
  tears of, 31–32
    disc protrusion and, 51
    discography of, 396–405, *396–405*
    low back pain and, 49–51
    MRI of, 416
    pain and, 379–381
Apophyseal joints
  anatomy of, 79
  ligaments of, resistance to flexion by, 91
  load-bearing by, 78
  posture effects on, 78
  resistance of
    to compression, 92
    to flexion-extension, 91
    to shear forces, 91–92
Arteparon, for arthritis, 340
Arthritis. *See also* Osteoarthritis; Rheumatoid arthritis
  enteropathic, laboratory evaluation in, 521–522
  HIV-related, 522
  Lyme, 522
  psoriatic, laboratory evaluation in, 521–522
  reactive, laboratory evaluation in, 521–522
  ulcerative colitis and, 493–494
Arthroscopic discectomy, 550
Articular facets. *See* Facet(s)
Artificial disc replacement, 550–551
Aseptic necrosis of bone, steroid-induced, 104

Atherosclerosis, 693
  abdominal aortic, disc disease and, 25, *26*
Athletes, spondylolysis/spondylolisthesis in, 643–644
Atrophy
  calf measurement for, 440, *440*
  thigh measurement for, 440, *440*
Autoantibodies, in rheumatic disease, 513–514, *514*, 514t
Autoimmunity
  chemical radiculitis and, 143–148
  disc degeneration and, 49
Automated distraction adjustment, 305–310. *See also* Cox distraction technique, automated distraction adjustment in
Autotraction, 283, *283*
Axial loading test, 445, *446*
Axial rotation. *See* Rotation
Axoplasmic transport, 132–136

Back mouse, 476
Back sprain/strain, 384, *385*
  Cox distraction for, 311t–313t, *322*, *323*
  diagnosis of, 384, *385*
  vs. disc disruption, 31
Bacterial endocarditis, 478
  laboratory evaluation in, 522–523
Bacteriuria, 511
Baker's cyst, tibial nerve compression from, 483
Balance board, 672, *674*, 675
Balance sandals, *675*, 675–676
Ball bridge, 669, *669*
Basic fibroblast growth factor, for disc dehydration, 551
Bechterew's sign, 424, *429*
Bechterew's test, 424–430, *429*
Belt, lumbar, 334, *334*
Bence-Jones proteins, in multiple myeloma, 519
Bending studies, 56–64, *61–65*
Bertolotti's syndrome, 239, *239*, *244–248*, 245–249, 390–392, 642
Betamethasone, facet joint injection of, 33
Biceps femoris muscle, 222, 223
Big toe. *See* Great toe
Binder, lumbosacral flexible, 334, *334*
Bleeding, gastrointestinal, nonsteroidal anti-inflammatory drugs and, 337, 338
Body weight, low back pain and, 81–82
Bone, metastases to, 488–490, *489*, *490*
Bone cyst, aneurysmal, 497–498, *498*
Bone marrow, in disc degeneration, 105
Bone marrow study, in multiple myeloma, 519
Bone metastasis, 488–490, *489*, *490*
  alkaline phosphatase and, 511
  laboratory evaluation of, 520
  in melanoma, 478
  pain in, 377–378
  in prostate cancer, 478–479, *479*
Bonet's phenomenon, 436
Borrelia
Bowstring sign, 437, *437*
Brace. *See also* Orthotics
  CASH, 336, *336*
  Jewett, 334–336, *335*
  Taylor, 334, *335*
Brace stabilization, diagnostic, 539
Bracing
  for disc herniation, 333
  effects of on spinal biomechanics, 333
  indications for, 333–334
  orthoses in, 334–336, *334–336*
  principles of, 333
Braggard's maneuver, 436, *436*
Breast reduction, for pain relief, 497
Bridge track exercises, 669, *669–670*
Brown tumor of hyperparathyroidism, sciatica and, 481
Brudzinski's sign, 435, *435*
Burns' bench sign, 444, *445*
Bursitis
  obturator internus, 478
  piriformis, 117
Burst fractures, thoracolumbar, spinal stenosis and, 193
Burton's concepts of traction reduction, 564, *564*
Buttock pain, in piriformis syndrome, 114–117, *115*, *116*

Caffeine, low back pain and, 23, 681
Calcification
  candlewax, in Foresterier's disease, 464, *465*
  disc herniation, 474–476, 477
  thoracic disc, in children, 580
Calcitonin, for neurogenic claudication, 198
Calcitonin gene-related peptide, 145
Calcium levels, 512, 513t
Calcium pyrophosphate dihydrate crystal deposition, in spinal stenosis, 192
Calculus, staghorn, 463, *464*
Calf measurement, 440, *440*
Camptocormia, 497
Cancer
  back pain in, 377–378
  bone metastasis in, 488–490, *489*, *490*
    alkaline phosphatase and, 511
    laboratory evaluation of, 520
    in melanoma, 478
    pain in, 377–378
    in prostate cancer, 478–479, *479*, *480*
  erythrocyte sedimentation rate in, 510, 510t
  multiple myeloma, 483
    back pain in, 377–378
    laboratory evaluation in, 518–519, *520*, *521*
  non-Hodgkin's lymphoma
    of epidural space, 478
    methotrexate-related, 483
  osteosarcoma, vitamin D_3 for, 343
  prostate, prostate-specific antigen in, 513
Candlewax calcifications, in Foresterier's disease, 464, *465*
Capsular ligaments
  flexion resistance of, 91
  rotational resistance of, 95
Cardiac disease, low back pain and, 25
Cardiac surgery, sciatica and, 481
Cartilage
  chondroitin sulfate and, 340, 342–343
  glycosaminoglycans and, 340–343
  nonsteroidal anti-inflammatory drugs and, 337
CASH brace, 336, *336*
Cauda equina
  nerve root origin from, 62–64, 66*burgdorferi*, 522, *523*
  neuroanatomy of, 17, *18–20*
  pressure measurement in, 176, *176*
Cauda equina symptoms, as surgical indication, 274
Cauda equina syndrome, 10
  spinal stenosis and, 187, 195
Causalgia, posterior longitudinal ligament, 33
Central distraction testing, 293, *293*
Centralization phenomenon, 284
Cerebrospinal fluid, plasma protein leakage into, 158
Cerebrospinal fluid proteins, elevated, 448–449
Cervical pain, myofascitis and leg pain and, 449
Cervical spinal stenosis, with lumbar stenosis, spondylolisthesis and, 638, *639*

Cervical spine distraction manipulation, evolution of, 3
Cervical spine effects, on lumbar spine, 568
Chairback brace lumbosacral orthosis, *334*
Chemical radiculitis, 143–148
  vs. mechanical radiculopathy, 146–147, *147*
  sciatica and, 117
Chemical sensitization, low back pain and, 29, 30–31, 48, 49, *50*
Chemonucleolysis, indications for, 534
Child abuse, as risk factor, 682
Children
  disc herniation in, 378–379
  discitis in, 517
  low back pain in, 82, 378–379, 529
  sick role in, 681
  spondylolisthesis in, 613, 631–632
  thoracic disc calcification in, 580
Chiro-Manis table, 2
Chiropractic care. *See also* Specific techniques
  allopathic medicine and, 7–8, 555–556
  benefits of, 10–13, 554–555
  clinical expectations for, 562–564
  complications of, 9–10, 10t
  costs of, 8–9
  demographics of, 4, 5, 6t–7t
  DRGs and, 8t
  hands-on effect in, 556
  vs. hospitalization, 273
  vs. McKenzie treatments, 9, 555
  osteopathic manipulative therapy and, 7–8
  outcome of, 274
  as outpatient care, 528
  patient satisfaction with, 4
  physical therapy and, 5–7
  population using, 4, 5, 6t–7t
  in pregnancy, 496
  principles of, 274–275
  results of, literature update for, 697–698
  rising use and acceptance of, 4
  support for, 10–13
  vs. surgery, 273–274
Chiropractors
  ideal number of, 7
  medical physicians and, 7–8
  referrals to
    resistance to, 7
    wisdom of, 8
Chondroitin sulfate, for arthritis, 340, 342–343
Chondromalaciae facetae, 113, *113*
Chronic back pain. *See* Low back pain, chronic
Chronic compartment syndrome, 469
Chronic fatigue syndrome, vs. fibromyalgia, 254
Chymopapain injection, indications for, 534
Cigarette smoking
  chronic low back pain and, 681
  disc disease and, 379
  disc malnutrition and, 343
  intermittent claudication and, 180, 182
  low back pain and, 23–25, 379
Circumduction range of motion adjustment, 300–301, *301*
Claudication. *See* Intermittent claudication
Clinical instability, definition of, 449
Coccydynia, 602
Coccygodynia, 497
Cognitive therapy, for fibromyalgia, 256
Cold application, after Cox distraction, 330, *331*, 332
Collagen
  physical therapy effects on, 7
  for rheumatoid arthritis, 343
Colopathy, nonsteroidal anti-inflammatory drugs and, 338
Compartment syndrome, 468–469, *469*
Compensation effects, 553–554
Complementary medicine
  frequency of use of, 4–5
  payment for, 4–5
Complex regional pain syndrome, 160–161
Compression. *See also* Load
  apophyseal joint resistance to, 40, 73–74
  disc, 34–35, 51, 69–70, 71–74, 75, *75*
    resistance to, 78, *79*, 92
  facet, *593*, 593–596, *594*
  ischemia and, 148–149
  nerve fiber, 141–143, *142*
  nerve root. *See* Nerve root compression
Compression fractures, 75, *75*
  of L2 vertebral body, 498, *499*
  in metabolic disorders, 520
  in osteoporosis, 492–493
  spinal stenosis and, 201–203, *202*
Computed tomography (CT). *See also* Imaging
  advantages of, 409
  artifacts on, 411
  of disc herniation, 407, *407*, 412–413
  in disc protrusion measurement, 152–156, 153t, *153–156*
  with discography, 382, *384*
  of facet tropism, 47
  indications for, 407–408, 416
  in low back pain, 112
  vs. magnetic resonance imaging, 408, 416
  normal findings on, *410*
  principles of, 409–411
Computed tomography myelography, 408, 409
Congenital hip dislocation, 485
Congruency, 680–681
Conjoined nerve roots, 473, *473*, *474*
Connective tissue, physical therapy effects on, 7
Conservative treatment
  algorithm for, *580–581*
  duration of, 568
  imaging changes on, 557–558
Contained vs. noncontained disc, 52
Coping strategies, 680–681, 684, 685
Corporotransverse ligament, nerve root entrapment by, 157
Corticosteroids. *See* Steroids
Coupled motion, 83
Cox Distraction Adjusting, certification program of, 13
Cox distraction technique, 261, 273–376. *See also* Distraction adjustment; Flexion-distraction manipulation; Spinal manipulation
  acupressure for, 328, *329*
  acupuncture meridian tracing for, 328
  algorithms for, 288, *289–291*
  automated distraction adjustment in, 305–310
    benefits of, 305
    with bilateral hand contact of spine, 309, *309*
    eight-finger glide palpation in, 307, *308*
    with foramen magnum pump, 308, *309*
    with left and right lateral flexion, 307, *307*
    with left and right rotation, 307–308, *308*
    at preset flexion angle, 306–307, *307*
    for scoliosis, 309, *309*
    in thoracic spine, 308, *308*
    treatment parameters for, 305–306
    with vector thrust adjustment, 310, *310*
  autotraction in, 283, *283*
  benefits of, 275, 283
  biomechanics of, 280–284, *281–283*
  central distraction testing in, 293, *293*

Cox distraction technique—*Continued*
centralization phenomenon and, 284
contraindications to, 292–293
definition of, 275
for disc herniation, 280–284, 295–301, *295–301*
results of, 311t–313t, *320, 321*
with sciatica, 295–297, *296, 297*
without sciatica, *298–301,* 299–301
duration of, 273, 274, 288–290, 297, 325t
efficacy of
in disk reduction, 284
mechanisms of, 284–286
electrical stimulation after, 330–333, *330–333,* 331t
exercise rehabilitation and, 297
for facet syndrome, 298, *298*
results of, 311t–313t, *316, 317*
frequency of, 288, 296–297
goading for, 328, *329*
history of, 1–3
vs. hospitalization, 273
hot/cold application after, 330, *331*
imaging for, 290
increased disc space and, 284–285
indications for, 275, 288, *289–291*
intermittent, 283
intermittent traction in, 284
lateral distraction testing in, *293,* 293–294
lateral flexion in, 296, *296*
ligament loads in, 288
for lumbar spine sprain/strain, results of, 311t–313t, *322, 323*
mechanoreceptor activation with, 285–286
neutral starting point for, *294,* 295
nonresponse to, patient options for, 288–290
outcome measures for, 310–326, 311t–313t, *314–325*
pain production on, 293–294
palpatory contact point in, 294, *294*
patient compliance with, 288
patient selection for, 288, *289–291*
for pelvic pain/dysfunction, 285
positioning in, 291–292, *292, 293, 294,* 294–295
for antalgic lean, 296, *296*
positive galvanism after, *330,* 330–333, *331,* 331t
pre- and postdistraction adjustment care in, 328
range of motion adjustment in, 298–301
circumduction, 300–301, *301*
extension, 301, *301*
flexion, *298,* 298–301
rotation, 300, *300, 301*
re-evaluation during, 448
rehabilitation and, 288
research grants for, 12–13
response to, phases of, 297
sacroiliac joint distraction adjustment in, *304,* 304–305, *305*
for sciatica, *295,* 295–297, *296*
scoliosis distraction adjustment in, *303,* 303–304, *304*
side lying, 296, *296*
side lying circumduction adjustment in, 303, *303*
side lying extension adjustment in, 302, *302*
side lying flexion adjustment in, 301–302, *302*
side lying lateral flexion adjustment in, 302–303, *303*
for spinal stenosis, 200, 282–284
spinous process contact in, 298, *298*
for spondyloarthrosis, results of, 311t–313t, *314, 315*
for spondylolisthesis, 305, *305,* 641–643, *642, 643,* 645–649
results of, 311t–313t, *318, 319*
spondylolisthesis distraction adjustment in, 305, *305*
tetanic current application after, 333, *333*
for thoracic disc herniation, 582–583, *583*
tolerance testing in
of ability to withstand distraction movements, *293,* 293–294
of lumbar motion segment, 292–293
traction contraindications in, 292–293
for transitional segment, results of, 311t–313t, *322, 323*
trigger point therapy for, 328, *329*
Cox pain classification, 446–449
Cox's sign, *438,* 438–439
C-reactive protein, 510
in discitis, 510
in polymyalgia rheumatica, 521
Crossed femoral nerve stretch sign, 450
Cryotherapy
after Cox distraction, 330, *331,* 332
for spondylolisthesis, 644, *644*
CT. *See* Computed tomography (CT)
CT myelography, 408, 409
Cushing's syndrome, 520
Cyst
aneurysmal bone, 497–498, *498*
Baker's, tibial nerve compression from, 483
ganglion
intraneural, 478
of posterior longitudinal ligament, 480
lumbar synovial, 492, *492, 493*
sacral Tarlov, 471–473, *472*
Cystic meningioma, 481–483, *482*
Cystitis, interstitial, L5 nerve root compression and, 162
Cytokines, in low back pain, 145

Dallas classification, for discography, *381,* 381t, 396
Dead bug track, 669–670, *670*
Decompressive laminectomy, for spinal stenosis, 195–197
Deep pain, 144
Degenerative spondylolisthesis. *See* Spondylolisthesis, degenerative
Demographics, 527
Depression, 679, 684
drug therapy for, 337–339
Dermatome(s), 55, 57
L5, dysesthesia of, 418t, *419*
pinwheel examination for, 441, *441*
Dermatome mapping, 421
Diabetes mellitus
decompressive laminectomy in, 197
spondylolisthesis in, 617
Diabetic radiculopathy, 480
Diagnosis. *See also* Specific conditions and techniques
from clinical findings, 422
computed tomography in, 407–416. *See also* Computed tomography (CT)
correlation of findings in, 446–448
correlative, 448
Cox protocol for, case studies of, 454–462, *454–463*
criteria for, 418t, *419–421*
discography in, 392–406. *See also* Discography
history in, 423–424
magnetic resonance imaging in, 407–416. *See also* Magnetic resonance imaging (MRI)
motor assessment in, 418t, *419–420,* 420
pain assessment in, *423,* 423–424
pain onset in, 417–418
physical examination in, *419–420,* 424–446
pressure algometry in, 452–454
recommendations for, 422–423
thermography in, 452
Diagnosis related groups (DRGs), for osteopathic manipulative treatment, 8t

Diagnostic biomechanics, 382–384
Diagnostic imaging. *See* Imaging and specific techniques
Dialysis, spinal stenosis and, 195
Diffuse idiopathic skeletal hyperostosis, 464–466, *465*, *466*
Disability payments, return to work and, 553–554, 681
Disability scales, 534
Disc
  age-related changes in, 66, 70
  anterior, nociceptors in, 33
  articulation of, 69, *73*
  blood supply in, end plate receptors and, 25
  bulging, diagnosis of, 446
  cadaver transplant, 551
  centrode location in, 68–69, *68–71*
  circulation in, 48–49
  compression effects on, 34–35, 51, 69–70, 71–74, 75, *75*
  compression resistance of, 78, *79*, 92
  contained, 52
    vs. noncontained, 449
  distraction effects on, 282
  extension effects on, *276*, 276–277, *277*
  flexion effects on, 78–79, 276, *276*, *277*, 280
  flexion resistance of, 78
  innervation of, 26–27, *27*, 32
  L3-L4, osteomyelitis of, *484*, 484–485, *485*
  load bearing by, 34–35, 51, 71–74, 75, *75*
    during flexion-distraction manipulation, 264–271, *265–267*, *269–271*
    mechanics of, 78–79
    posture and, 78
  load-bearing components of, 35
  noncontained, 52
    vs. contained, 449
  nutrition in, 341–343
    end plate receptors and, 25
    exercise and, 342
    smoking and, 343
  osmotic principles of, 79–81
  as pain source, 21–22, 25–26, 28, 29, 31, 33–34, 36–37, 382–384, 388–392, *388–392*, 407, 566
  pain-sensitive structures in, 29–30, *30*
  pressure in. *See* Intradiscal pressure
  resistance of
    to compression, 77, 78, *78*, *79*, 92
    to flexion, 78
    to force, 77–78, *78*
    to rotation, 82–84, 95, *83–85*
    to shear force, 71–73, 77
  rotation of, 69–70, *73*, *74*, 75, 76, *80*
    resistance to, 82–84, *83–85*, 95
  rotational stress on, 593
  shear resistance of, 71–73, 77
  sitting effects on, 79, *80*
  torsional vs. compressive injury of, 97–100
  transplantation of, 551
  wandering, *387*, 387–388
  water content of, 51
  weightbearing load on, 34–35
Disc biomechanics, 51–56, *51–58*
  radiography of, 56–64, *61–65*
Disc calcification, thoracic, in children, 580
Disc degeneration
  abdominal aortic atherosclerosis and, 25, *26*
  in adolescents, 107
  age-related, 66, 70
  amyloid in, 107
  anulus fibrosus in, 108–109, 279–280
  biochemical changes in, 107
  biomechanical factors in, 34
  chemical sensitization and, 29, 30–31, 48, 49, *50*
  classification of, 381, *381*, 381t
  decreased nuclear pressure in, 112
  Discat for, 341
  disc-facet relationship in, 28, 33–34
  discogram findings in, *51*, 102t, 102–103
  end plate and bone marrow changes in, 105
  end plate failure in, 109
  facet changes in, 66, 67
  facet orientation and, 47
  imaging of, 411–413, *413*
  immunologic factors in, 45
  interleukin-1 in, 107
  joint laxity and, 105
  lactate levels in, 107
  leg pain and, 21–22, 417. *See also* Leg pain; Sciatica
  level of, determination of, *104*, 104–105
  lipofuscin in, 107
  literature update for, 693–695
  magnetic resonance imaging of, 109
  morphologic changes in, 107–108, *108*
  vs. muscle strain, 31
  nerve root compression in, cadaver studies of, 36
  normal x-ray of with abnormal MRI, 490, *491*
  nutritional factors in, 106, 340, 458–459
  obesity and, 25
  onset and course of, 381, *381*
  osteophytes in, 108–109
  pain in, 21–22, 388–392, *388–392*, *390–392*. *See also* Low back pain
  pathogenesis of, 565
  pelvic disease and, 417
  pressure vs. load in, 34
  proteoglycan loss in, 106, *106*, 107, 340
  rotational movement and, 34, 101
  spondylolysis and, 51. *See also* Spondylolysis
  spondylosis and, 33
  stages of, 67, 67
  subdiscal bone changes in, 109
  tall stature and, 25
  tissue regeneration in, 106
  vascular changes in, 108
  weightbearing and, 35–36
Disc disease. *See also* Disc degeneration; Disc prolapse
  biomechanical processes in, *50*
  diagnosis of. *See* Diagnosis
  grading of, 186–187, *187*
  level of, 55–56
  literature update for, 693–695
  motor changes in, 418t, *419–420*, 420
  nucleus pulposus motion in, 102
  posture and, 416
  sciatic scoliosis in, 449, *450*
  smoking and, 343, 379. *See also* Smoking
  spinal stenosis and, 179
  stages of, 67, 67
  vs. tumors, 423t
Disc extrusion, on discography, *51*
Disc herniation, *52–54*, 52–56, 67, 67
  acute, treatment of, 566
  age and, 529
  anular tears and, 51
  asymptomatic, 170, *171*
  bracing for, 333
  calcified, 474–476, *477*
  case studies of, 570–577, *570–578*

Disc herniation—*Continued*
in children, 378–379
incidence of, 530
treatment of, 530–531
classification of, by intravertebral location, *451*
computed tomography of, 152–156, 153t, *153–156*, 407, *407*, 412–413. *See also* Computed tomography (CT)
Cox distraction for, 280–284, 295–301, *295–301*. *See also* Cox distraction technique
results of, 311t–313t, *320*, *321*
definition of, 416, 417
diagnosis of, 447
case studies of, 454–461, *454–461*
vs. disc protrusion, 418t
discography of, *51*, 396–401, *396–401*, 405, 406
dorsal root ganglion compression by, sciatica and, 149–150
extension effects on, *276*, 276–277, *277*
extraforaminal, 451–452, *453*, *454*, 458–459, *460*
extreme lateral, 535
far lateral (foraminal), *450*, 450–454, *451*, 452t, *453*
flexion effects on, 276, *276*, 276–277, *277*
gas-containing, 480
hypervascularity in, 108–109
intradiscal pressure and, 280
intradural, 452
lateral vs. medial, sciatic scoliosis and, 449, *450*
leg length inequality and, 118
leg pain and, 417
low back pain and, 30–31, *31*
magnetic resonance imaging of, 406–416. *See also* Magnetic resonance imaging (MRI)
natural course of, 532–534
nerve fiber compression in, 141–143, *142*
nerve root compression in, 149
neuroanatomic findings in, 17–23
outcome predictors in, 529–530
pathomechanism of, 381–382, *381–383*
posture in, 56–58, *58*
principles of, 416–417, 417
vs. prolapse, 418t
recurrent, vs. scar tissue, 449, 536
reduction of
clinical correlates of, 558, 560–562, *560–562*
Cox distraction for. *See* Cox distraction technique
flexion-distraction manipulation for. *See* Flexion-distraction manipulation
imaging of, *557*, 557–558, *558*, 560–562, *560–562*
mechanisms of, 559
pain relief from, 150–152, *150–152*
with spinal manipulation, 555
symptom relief and, 284
Schmorl's nodes in, 109
scrotal pain and, 21
sequestration in, 109
size of
assessment of, 560
clinical correlates of, 557–560
spinal stenosis and, 186, *188*, 188–189, *190*, *198*, *199*, 199–200
in thoracic spine, 198
with spondylolisthesis, 631
spontaneous regression of, *500*, 501, *501*
surgery for. *See also* Surgery
thoracic
in children, 582
diagnosis of, 449, 581–582
incidence of, 578–580
symptoms of, 580–581
treatment of, 582–583
3-month healing period for, 297
transdural, 561–562, *562*
transitional segment and, 239, *244–246*, 245
treatment options for, 261
treatment-related changes in, imaging of, 557–558
upper lumbar, diagnosis of, 449–450
vertebrogenic symptoms of, 32, *32*
Disc implants, artificial, 550–551
Disc infection
erythrocyte sedimentation rate in, 510, 510t
laboratory evaluation in, *517*, 517–518
Disc injury
from low back exercises, 101
rotational, 41
vs. compressive, 41, 97–100, 100t
Disc metabolism, flexion effects on, 277
Disc prolapse, *52–54*, 52–56. *See also* Disc herniation
chemical irritation of nerve root from, 49
definition of, 384, 417
principles of, 417
sagittal facets and, 43–47
stages of, 36, *36*
Disc protrusion. *See* Disc herniation
Disc resorption, mechanisms of, 559
Disc surgery, indications for, 407
Disc transfer, 551
Discat, for disc degeneration, 341
Discectomy. *See also* Surgery
arthroscopic, 550
complications of, 535–536
disc contour after, 559
laser, 535
microdiscectomy, results of, 537
percutaneous
indications for, 534
MRI findings on, 559
results of, 537
results of, 535
scar tissue from
pain and, 559
vs. recurrent disease, 449
Discitis
erythrocyte sedimentation rate in, 510, 510t
laboratory evaluation in, *517*, 517–518
Discogenic spondylosis, 66
Discography, 392–406. *See also* Imaging
abnormal findings on, 51, *51*, 396–401, *396–405*
age and, 51, 52t
case studies of, 402–405, *403–405*
complications of, 405
with computed tomography, 382, *384*
Dallas classification for, *381*, 381t, 396
for extraforaminal disc fragmentation, 394
indications for, 405–406
vs. MRI, 392–394, *393*, *394*
normal findings on, 396, *396*, *399*
pain on, 405
pain relief from, 394
results of, classification of, *381*
in spondylolisthesis, 629, *630*
Dislocation, congenital hip, 485
Distraction adjustment. *See also* Cox distraction technique; Flexion-distraction manipulation
for acute herniation, 566
biomechanical effects of, 564–566, *564–566*
cauda equina syndrome and, 10

certification in, 3
cervical, evolution of, 3
complications of, 9–10, 10t
contraindications to, 567
costs of, 9–10
disc effects of, 282
effectiveness
government endorsement of, 12–13
history of, 1–3
indications for, 10–11, 288, *289–291*
literature on, 3-4
maturation of, 3
vs. mobilization, 5–6
purposes of, 566–567
research grants for, 12–13
rule of three for, 567
safety of, 11
screening for, 288, *289–291*
for spondylolisthesis, 305, *305*, 641–643, *642*, *643*, 645–649
theories of, 564–566, *564–566*
training in, 13
for visceral conditions, 10, 10t
Diurnal height, loss of, low back pain and, 25
Divorce, 683, 684
Doctor-patient interaction, 683–684
Dorsal fat pad, flexion-extension effects on, 89–90, *90*, *92*, *93*
Dorsal nerve roots. *See* Nerve root(s)
Dorsal ramus damage, failed back syndrome and, 536
Dorsal ramus entrapment, iliac crest and T11-L1 pain and, 28–29
Dorsal ramus irritation, sources of, 27–28
Dorsal root ganglia
anatomy and physiology of, 131–132, *132–134*, *132–136*
cells of, 178
changes in, radiculopathy and thermal hyperalgesia and, 132–143
chemical irritation of, 143–148
circulation and protein synthesis in, 132–136
in claudication, 140
compression of, 147–150
diagnosis of, 449
sciatica and, 149–150
in groin pain, 140
hypoxia in, 148
in low back pain, 132–143, 138–141, 286–288, *287*
pain sensitivity of, 286–288
positions of, 131–132, *132–134*
in sciatica, 140
size and location of, 286
in spinal stenosis, 178
substance P in, 140–141, 145–146, 150
types of, *134*, *135*
vulnerability of to injury, 34
Dorsal root gangliectomy, for sciatica, 150
Dorsal sacroiliac ligament, 214–215, *216*, *217*
Dorsalis pedal artery, assessment of, 442, *443*
Dorsiflexion
of foot, 418t, *419*, 439, *439*
of great toe, 418t, *419*, 439, *439*
Double crush syndrome, 159–160
DRGs, for osteopathic manipulative treatment, 8t
Drug therapy. *See also* Specific drugs and drug families
complications of, 336–339
effectiveness of, 336–337
in elderly, 339
indications for, 336–337
Dual dermatome treatment, for sciatica, 568, 569
Duodenal ulcers
nonsteroidal anti-inflammatory drugs and, 337, 338
spinal manipulation for, 554
Dura mater, in low back pain, 29, 695
Dural sac, nerve root origin from, 62–64, 66
Dutchman roll, 241, *242*
Dysplastic spondylolisthesis, 611. *See also* Spondylolisthesis

Edema, intraneural, in nerve root compression, 140f, 3–4, 10–13
Eight finger glide palpation, in Cox distraction, 307, *308*
Eisenstein's measurements, 180, 607, 637, *637*
Elderly, drug therapy in, 339
Electrical stimulation, after Cox distraction, 330–333, *330–333*, 331t
Electroacupuncture, for fibromyalgia, 256
Electromyographic feedback, for fibromyalgia, 256
Electromyographic studies, of flexion-distraction manipulation, 263–264, *264*, *265*
Electromyography, vs. nerve root needle stimulation, 421–422
Ely's heel-to-buttock sign, 443, *443*
Embolism, nucleus pulposus, postoperative, 536
Employment, return to, 553–554, 681–682
Employment-related factors, in low back pain, 22, 81, 529, 553–554, 681
End plate(s)
in disc degeneration, 105
in low back pain, 107
End plate degeneration, imaging of, 413
End plate receptors, disc nutrition and, 25
Endocarditis, infective, 478
laboratory evaluation in, 522–523
Endometriosis, of sciatic nerve, 480–481
End-stage renal disease, nonsteroidal anti-inflammatory drugs and, 338
Endurance tests, 659–662
Enteropathic arthritis, laboratory evaluation in, 521–522
Enteropathy, nonsteroidal anti-inflammatory drugs and, 338
Eosinophilia-myalgia syndrome, 476–478
Ependymoma, 463, *463*
Epidural analgesia/anesthesia
complications of, 553
for low back pain, 40
Epidural fat graft, nerve root compression from, 536
Epidural fibrosus, 536
Epidural hematoma, 469, *470*, *471*
Epidural lipomatosis
idiopathic, 481
steroid-induced, 339
Epidural pressure, in spinal stenosis, 178
Epidural space, non-Hodgkin's lymphoma of, 478
Epidural steroid injections, 40, 551–553, 699
Epstein-Barr virus infection, lumbar radiculopathy and, 481
Erector spinae muscles, 215–216, *218*, 222–223
spinal stability and, 654–655, *655*
Ergonomics, of proper lifting, 77
Erythrocyte sedimentation rate (ESR), 510, 510t
in disc infections, 510, 510t, 517
in multiple myeloma, 519
in osteomyelitis, 510, 510t
in polymyalgia rheumatica, 521
Esophageal injury, nonsteroidal anti-inflammatory drugs and, 338
Eversion, of foot, assessment of, 418t, *419*
Exercise. *See also* Rehabilitation
disc nutrition and, 342
in intermittent claudication, 182
nutrition in, 341–342
Exercise tracks, 668–676
Exercises, 653–676. *See also* Rehabilitation
bridge track, 669, *669–670*
with Cox distraction, 297
for fibromyalgia, 255
flexion-extension, 117–118

Exercises—*Continued*
for instability, 105
low back, disc injury from, 101
with manipulation, 654
overview of, 653–654
peeling back in, 669
pelvic stabilization, costs of, 528
progression of, 669, 669t
for sacroiliac joint pain, 230
in spinal stabilization training, 665–676
for spondylolisthesis, *644*, 644–645, *645*
Extension. *See also* Flexion-extension; Lordosis
disc and ligament support in, 101
effects of
on disc herniation, *276*, 276–277, *277*
on facet, 596
on ligamentum flavum, 277
on lumbar spine, 85–90, *87*, *276*, 276–277
on nucleus pulposus movement, 278–279
in spinal stenosis, *175*, *176*
Extension manipulation. *See also* Flexion-extension distraction
for retrolisthesis subluxation, 606–608, *606–608*
Extension range of motion adjustment, 301, *301*
Extension-distraction manipulation, effects of, 275–276, *276*
External fixation, diagnostic, 539
Extraforaminal herniation, 451–452, *453*, *454*, 458, *459*

Facectomy, unilateral, facet load and, 34
Facet(s)
analgesic injection of, 33
arthritis of, 42–43
arthrosis of, 67, 67
asymmetric, *41*, 41–48, 42t, *43–46*. *See also* Facet tropism
compressive forces on, 591–593, *592*, 592t
coronal facings of, 41–43, 42t, *43–46*. *See also* Facet tropism
in degenerative spondylolisthesis, 634, *635*
in disc degeneration, 66, 67
distraction of, beneficial effects of, 556–557
extension effects on, 596
flexion effects on, 78–79, 596
flexion-distraction effects on, 556–557
fractures of, 464, *464*
in hyperextension-rotation injury, 114
inclination angle of, 73
innervation of, 110
load effects on, 34–35, 73, 593, *593*, *594*, 596
in low back pain, 25–26, 28, 29, 33–34, 109–112, *110*, *111*
in lumbar spine stability, 92–94
mechanoreceptors in, 110–112, 286
normal movement of, 605
orientation of, 41–43, 42t, *42–46*, *44*
facet orientation circle and, *47*, 47–48, *48*
measurement of, 43, *45*, *47*, 47–48, *48*
radiographic assessment of, 47
pain patterns in, 448–449, 602t, 602–603, *603*
pain sensitivity of, 593–596
pressure effects on, 34
range of motion of, 605
resistance of, to rotation, 95
rotational stress on, 593
sagittal facings of, 41–43, 42t, *43–46*. *See also* Facet tropism
shear resistance of, 71–73
single-photon emission computed tomography of, 113–114, *113*, *114*
stability of, 599–601, *599–601*
steroid injections in, 603
subluxation of, 593
Hadley S curve and, 593, *594*, *595*
spinal stenosis and, 186, *189*
symmetric, 43, *45*
Facet orientation circle, *47*, 47–48, *48*
Facet pain, distribution of, 448–449, 602t, 602–603, *603*
vs. disc pain, 448–449
Facet syndrome, 30, 286, 591–608
anatomic factors in, 601–602
ancillary care for, 605, *606*
biomechanics of, 591–596, 601–602
case study of, 608
Cox distraction for, 298, *298*
results of, 311t–313t, *316*, *317*
diagnosis of, 447
Hadley S curve in, 593–596, *594*, *595*
manipulative care in, *604*, 604–606
pain patterns in, 602t, 602–603, *603*
radiofrequency denervation for, 604
radiographic findings in, 596–599, *596–601*
referred pain in, 602t, 602–603, *603*
retrolisthesis subluxation and, 606–608, *606–608*
spinal stenosis and, 186, *189*
Facet tropism, *41*, 41–48, 42t, *43–46*
atherosclerosis and, 693
disc degeneration and, 47
disc prolapse and, 43–47
facet orientation circle for, *47*, 47–48
prevalence of, 47
radiographic assessment of, 47
Facet-lamina syndrome, 591
Factitious disorders. *See* Malingering
Failed back surgery syndrome (FBSS)
algorithm for, *540*
causes of, 536
clinical presentations of, 540–550, *541–543*, *545–550*
spinal cord stimulation for, 539
Fajersztajn's sign, 436–437, *437*
Far lateral disc herniation, 450–454, *451*, 452t, *453*
Far out syndrome, 458
Fascia, thoracolumbar, innervation of, 33
Fat pad, dorsal, flexion-extension effects on, 89–90, *90*, *92*, *93*
Fatigue, in fibromyalgia, 254–255
Fear-avoidance behavior, 685
Femoral artery, assessment of, 442, *442*
Femoral nerve, anatomy of, 21
Femoral neuropathy, abdominal aneurysm and, 484
Femur. *See also* Hip
osteomyelitis of, 487–488, *488*
Fibromyalgia, 251–257
chiropractic care for, 255–256
vs. chronic fatigue syndrome, 254
clinical manifestations of, 252, 252t, 253t
diagnosis of, 251–252, 252t, 253t
differential diagnosis of, 253–254, 254t
drug therapy for, 256
etiology of, 254–255
exercise therapy for, 255–257
history of, 251
incidence of, 251
management of, 255–257
vs. myofascial pain syndrome, 254, 254t
pathophysiology of, 254–255
prognosis in, 257
psychologic aspects of, 256, 684
tender points in, 252, *252*
Fibrous capsule, in low back pain, 109–110
Financial aspects, of low back pain, 527–528
Finite element modeling, 707–711

First onset back pain, 377
Flank pain, spinal origin of, 163
Flare-up onset back pain, 377
Flexed hip test, 445, *446*
Flexibility test, 659, *660*
Flexibility training, 665, *666*
Flexion
effects of
on anulus fibrosus movement, 278–279
on disc herniation, *276*, 276–277, *277*
on disc metabolism, 277
disc strength and, 280
on facets, 78–79, 596
on foraminal size, 277
on ligamentum flavum, 277
on lumbar spine, 85–90, *87*, *276*, 276–277
on nerve root compression, 277
on nucleus pulposus movement, 278–279
on spinal canal diameter, 277
lateral
disc protrusion and, 56–62, *58–64*
right vs. left-sided, 102
vs. rotation, *97–99*, 97–100, 100t
segmental site and degree of, *238*
Flexion exercises, for spondylolisthesis, *644*, 644–645, *645*
Flexion range of motion adjustment, *298*, 298–299
Flexion roll, Dutchman, 241, *242*
Flexion-distraction manipulation. *See also* Distraction adjustment
analgesic effects of, 286–288
biomechanics of, 85–90, *87*, 261–271, 280–284, *281–283*, 556–557
clinical expectations for, 562–564
contraindications to, 292–293
vs. conventional medical treatment, 555–556
Cox method. *See* Cox distraction technique
for duodenal ulcers, 554
electromyographic studies of, 263–264, *264*, *265*
facet effects of, 556–557
with flexion-extension exercises, 117–118
functional basis for, 85–86
hands-on effect in, 556
history of, 261
indications for, 288, *289–291*
intradiscal pressure in, 264–271, *265–267*, 267t, 268t, *269–271*, 278
ligament loads in, 268–271, *269–271*, 288
vs. McKenzie treatment, 555
mechanoreceptor activation with, 285–286
pain production on, 293–294
for pelvic pain/dysfunction, 163t, 163–164
for pelvic pain/dysfunction, 285
radiographic studies of, 262–263
re-evaluation during, 448
research grants for, 12–13
for sacroiliac joint pain, 229, *230*
screening for, 288, *289–291*
spinal effects of, 85–90, *87*, 275, *275*, 276–278
spinal reflexes and, 286
for spinal stenosis, 117, 198
for testalgia, 163
for transitional segment, 241–243, *242*
vertebral canal diameter and, 277–278
vertebral motion during, 262–263
Flexion-distraction table, mobility of, 263, *263*
Flexion-extension
effects of
on disc, 78–79
on dorsal fat pads, 89–90, *90*, *92*, *93*
on lumbar spine, 85–90, *87*
nucleus pulposus motion in, 101–105, *103*, *104*
resistance to
by apophyseal joints, 91
by discs, 78
by ligaments, 91
in spinal stenosis, *175*, *176*
Flexion-extension exercises, 117–118
Flip test, 444, *445*
Floor bridge, 669, 669
Fluid ingestion, pain and, 384–385, *385*
Foot
dorsiflexion of, assessment of, 418t, *419*, 439, *439*
eversion of, assessment of, 418t, *419*
plantar flexion of, assessment of, 418t, *420*
Foramen, L5-S1, developmentally enlarged, 462, *463*
Foramen magnum pump, in Cox distraction, with automated distraction adjustment, 308, *309*
Foraminal herniation, 450–454, *451*, 452t, *453*
Foraminal size, flexion effects on, 277
Foresterier's disease, 464–466, *465*, *466*
Fracture(s)
alkaline phosphatase and, 511, 512t
burst, spinal stenosis and, 193
compression, 75, 75
of L2 vertebral body, 498, *499*
in osteoporosis, 492–493
spinal stenosis and, 201–203, *202*
facet, 464, *464*
in hyperextension-rotation injury, 114
Harrington rod, 488, *489*
pars interarticularis, in spondylolisthesis, *613*, 614, *614*. *See also* Spondylolisthesis
posterior apophyseal ring, 481, *482*
sacral insufficiency, 493, *493*
spinal stenosis and, 193, 201–203, *202*
spondylolysis and, 611–613, *613*
stress, metatarsal, 487, *488*
Functional spinal unit, injury of, stages of, 35–36
Functional testing
qualifiable, 659t, 662–665
quantifiable, 659t, 659–662
Furcal nerve, in lumbosacral radiculopathy, 158

Gadolinium-enhanced magnetic resonance imaging, 413–416, *415*, *416*
Gaenslen's sign, 438, *438*
Gaenslen's test, for sacroiliac pain, 226
Gait assessment, 430, *431*
Galvanism
application of, guidelines for, 331t
after Cox distraction, *330*, 330–333, *331*, 331t
definition of, 330
effects of, 331t
electrode application in, 332
for low back pain, 332
mechanism of action of, 330–331, 331t
milliamperage for, 331–332
polarity in, 332
results of, 332
for spondylolisthesis, 644, *644*
Ganglion cyst
intraneural, 478
of posterior longitudinal ligament, 480
Gastric ulcers, nonsteroidal anti-inflammatory drugs and, 337, 338
Gastrointestinal bleeding, nonsteroidal anti-inflammatory drugs and, 337, 338
Gastrointestinal dysfunction, nonsteroidal anti-inflammatory drugs and, 338

Genetic factors, in low back pain, 22
Genitofemoral nerve, anatomy of, 17
Genitourinary disease, disc disease and, 417
George's line, *606*, 607
G-glutamyl transferase, 511
Glucosamine sulfate, for arthritis, 342
Glucosuria, 511
Gluteal skyline sign, 418t, *420*
Gluteus maximus muscle, 215–216, *218*, 222, 223
  goading of, for Cox distraction, 328, *329*
  pain trigger points and referral patterns in, *228*
Gluteus medius muscle
  goading of, for Cox distraction, 328, *329*
  pain trigger points and referral patterns in, *228*
Gluteus minimus muscle
  goading of, for Cox distraction, 328, *329*
  pain trigger points and referral patterns in, *228*
Gluteus muscle testing, 418t, *420*
Glycosaminoglycans, sulphated, for arthritis, 340–341
Goading, for Cox distraction, 328, *329*
Goniometric measurements, 431–434, *433*, *434*
Gout, 512
Gracilis muscle, goading of, for Cox distraction, 328, *329*
Great toe
  dorsiflexion of, assessment of, 418t, *419*, 439, *439*
  plantar flexion of, 440, *440*
    assessment of, 418t, *421*
Growth factors, for disc dehydration, 551

Hadley S curve, 593, *594*, 595
Hamstring bridge, 669, *670*
Hamstring muscle, scarring of, sciatic nerve entrapment by, 483
Hamstring muscle length, spondylolisthesis and, 618
Hamstring muscle reflex, 442, *442*
Hamstring muscle testing, 418t, *420*
Hamstring postisometric relaxation, 666
Hamstring self-stretch, 666
Hamstring stretch, for spondylolisthesis, 644, *644*
Hands-on effect, 556
Harrington rod fracture, 488, *489*
Headache, with chronic back pain, 492
Healing process, phases of, 297
Heat application, after Cox distraction, 330, *331*, 332
Heel walk, 434, *434*
Height, low back pain and, 81–82
Helicobacter pylori infection, nonsteroidal anti-inflammatory drugs and, 339
Hemangiomas, 466, *466*
Hematoma
  epidural, 469, *470*, *471*
  ligamentum flavum, 470
  psoas muscle, 478
Hematuria, 511
Hepatic dysfunction, nonsteroidal anti-inflammatory drugs and, 339
Herpes zoster radiculopathy, 480
Hexopal (mesoinositol hexanicotinate), for spinal stenosis, 196
Hip
  avascular necrosis of, 486–487, *487*
  congenital dislocation of, 485
  osteomyelitis of, 487–488, *488*
  transient osteoporosis of, 497
Hip abduction test, 662–664, *664*
Hip extension test, 662, *663*
Hip osteoarthritis, leg length inequality and, 118
History taking, 423–424
HLA system, 516, *516*, 516t
HLA-B27, 516, *516*, 516t, 521
  in ankylosing spondylitis, 516, *516*, 516t, 521
Homeopathy, for fibromyalgia, 256
Human immunodeficiency virus (HIV) infection, laboratory evaluation in, 522
Human leukocyte antigen (HLA) system, 516, *516*, 516t
Hydrocollator, after Cox distraction, 330, *331*, 332
Hypercalcemia, 512, 513t
Hypercortisolism, 520
Hyperextension-rotation injury, facet fracture as, 114
Hypergammaglobulinemia, in multiple myeloma, 519
Hyperparathyroidism, 512
  back pain and, 520
Hyperphosphatemia, 513
Hypertension
  intraosseous, low back pain and, 37, 176–177
  nonsteroidal anti-inflammatory drugs and, 338
Hyperuricemia, 512
Hypocalcemia, 512, 513t
Hypophosphatemia, 513

Iatrogenic pain, diagnosis of, 447
Idiopathic epidural lipomatosis, 481
IgA, in disc disease, 45
IgG
  in disc disease, 45
  in low back pain, 145
IgM
  in disc disease, 45
  in low back pain, 145
Iliac crest pain, L1-L2 dorsal ramus entrapment and, 28–29
Iliac tuberosity, disfigurement of, 222, 223
Iliocostal pain, 496–497
Iliohypogastric nerve, anatomy of, 17
Ilioinguinal nerve, anatomy of, 17
Iliolumbar artery, 216, *218*
Iliolumbar ligament, 215, *216*
Iliopsoas muscle, 215–216, *218*
Illness behavior, 681
Imaging. *See also* Specific techniques
  artifacts on, 411
  for Cox distraction, 290
  of disc reduction, *557*, 557–558, *558*, 560–562, *560–562*
  indications for, 407–408, 531–532
  for osteomyelitis, 518
  for thoracic herniation, 450
  timing of, 406–407
  of treatment effects, *557*, 557–558, *558*
Immobilization, low back pain and, 113
Immunoglobulins
  in disc disease, 45
  in low back pain, 145
Immunologic factors, in disc disease, 45
Incontinence, urinary, low back pain and, 162–163
Infections, spinal, laboratory evaluation in, 510, 510t, *517*, 517–518
Infective endocarditis, 478
  laboratory evaluation in, 522–523
Inflammatory bowel disease, spondyloarthropathies and, 521
Inguinal pain, source of, 28
Insurance effects, 554
Interleukin-1, in disc degeneration, 107
Intermittent claudication
  in athletes, 469
  calcitonin for, 198
  definition of, 180
  degenerative spondylolisthesis and, 193, *193*, *194*
  differential diagnosis of, 180, *180*, 181
  Doppler testing in, 181
  mechanisms of, 181

mesoinositol hexanicotinate for, 196
nerve root ischemia and, 181
neurogenic vs. ischemic, 180
pentoxifylline for, 196
physical examination in, 181
risk factors for, 180
in spinal stenosis, 172, 178, *180*, 180–182
atypical, 184
treadmill test in, 182
treatment of, 182
Internal iliac artery, 216, *218*
Internal oblique abdominis muscle, spinal stability and, 655, 656
Interneural inflammation, low back pain and, 287–288
Interspinous ligaments, resistance of
to flexion, 91
to rotation, 95
Interstitial cystitis, L5 nerve root compression and, 162
Intervertebral disc. *See under* Disc
Intervertebral osteochondrosis, 102–104
Intestinal injury, nonsteroidal anti-inflammatory drugs and, 338
Intra-abdominal pressure, spinal effects of, 40
Intradiscal gas, 480
Intradiscal pressure
above fusion level, 538–539
changes in, 64, 66
disc herniation and, 280
in flexion-distraction manipulation, 264–271, *265–267*, 267t, 268t, *269–271*, 278
pain and, 382–383
Intradural herniation, 452
Intraneural edema, in nerve root compression, 149
Intraneural ganglion cyst, 478
Intraosseous pressure, in low back pain, 37, 176–177
Intravenous immunoglobulin, for nerve root pain, 550
Ischemia, nerve root, 148–149, *149*
Isometric ball squeeze, 669, 670
Isthmic spondylolisthesis, 611, *612*. *See also* Spondylolisthesis

Jewett brace, 334–336, *335*
Job, return to, 529, 553–554, 681–682
Job satisfaction, 529
Job-related factors, in low back pain, 529, 553–554, 681
Joint pain, pathophysiology of, 285–286
Joint-muscle relationships, 665t

Kemp's sign, 431, *432*
Kidney stone, 463, *464*
Kyphosis, low back pain and, 81–82

L1–L2 dorsal ramus entrapment, iliac crest and T11–L1 pain and, 28–29
L5 dermatomes
dysesthesia of, 418t, *419*
mapping of, 421
pinwheel examination for, 441, *441*
L5 nerve, sickle-shaped ligament compression of, 478
L5–S1 joint. *See also* Transitional segment
vulnerability of, 601–602
Laboratory evaluation, 509–523
acid phosphatase in, 513
in AIDS, 522
alkaline phosphatase in, 511, *512*, 512t
antinuclear antibodies in, *514*, 514–515, *515*, 515t
calcium in, 512, 513t
C-reactive protein in, 510
erythrocyte sedimentation rate in, 510, 510t
in infective endocarditis, 522–523
in lumbar spine and sacroiliac infections, *517*, 517–518, *518*
in Lyme disease, 522, *523*
in metabolic disorders, 520, *521*
in metastatic cancer, 519
in multiple myeloma, *517–519*, 518–519, 520t
nonspecific tests in, 509, 510–513
in osteoporosis, 516–517
in Paget's disease, 522
phosphorus in, 513
in polymyalgia rheumatica, 521
in polymyositis, 522
prostate specific antigen in, 513
rheumatoid factors in, 513–514, *514*, 514t
specific tests in, 509, 513–516
in spondyloarthropathies, 520–521
uric acid in, 512, 513t
urinalysis in, 510–511
Lactate levels, in disc degeneration, 107
Laminectomy. *See also* Surgery
lumbar decompressive, for spinal stenosis, 195–197
trumpet, for spinal stenosis, 197
Laser discectomy, 535
Lateral bending studies, 56–64, *61–65*
Lateral cutaneous nerve, anatomy of, 21
Lateral distraction testing, *293*, 293–294
Lateral flexion
disc protrusion and, 56–62, *58–64*
right vs. left-sided, 102
vs. rotation, 97–99, *97–100*, 100t
Lateral flexion range of motion adjustment, 299, *299*
Lateral sacral artery, 216, *218*
Lateral sacral artery aneurysm, 476
Latissimus dorsi muscle, 222, 223
Lawsuits, compensation in, 554
Layer syndrome, 658
Lean, antalgic, 56–62, *58–64*, 59, *61*, *63*
assessment of, 430–431, *431*
Cox distraction positioning for, 296, *296*
Leg length inequality, 118–121
correction of, 119–121, *120*
evaluation of, visual vs. radiographic, 118–119, 121t
hip osteoarthritis and, 118
low back pain and, 82, 449
Leg pain. *See also* Sciatica
claudication. *See* Intermittent claudication
disc degeneration and, 417
disc disease and, 21–22
distribution of, 602t, 602–603, *603*
in spinal stenosis, 177, 178
Legal awards, 554
Lewin's standing sign, 430, *430*
Lewit's functional chains, 665, 665t
Libman's sign, 444, *445*
Lidocaine
facet joint injection of, 33
for reflex sympathetic dystrophy, 160–161
Lifting
correct, ergonomics of, 77
posture for, 280
Ligaments. *See also* Specific ligaments
loads on, in flexion-distraction manipulation, 268–271, *269–271*, 288
in lumbar spine stability, 92–94
nerve root entrapment/fixation by, 157
resistance of
to flexion, 91
to rotation, 95

Ligamentum flavum
  flexion/extension effects on, 277
  in spinal stenosis, *102*, 192, 200, *201*, *203*, 203–204, *204*
Ligamentum flavum hematoma, 470
Limbus vertebrae, *461*, 461–462, *462*
Lindner's sign, 424–430, *429*, 435, *435*
  straight leg raising sign and, 436
Lipofuscin, in disc degeneration, 107
Lipomatosis, epidural, steroid-induced, 339
Literature update, 689–703
Liver function tests, for nonsteroidal anti-inflammatory drugs, 339
Load
  in disc disease, 35–36
  effects of on facet vs. joint, 34–35
Load bearing. *See also* Compression
  in disc disease, 35–36
  by discs, 34–35, 51, 71–74, 75, *75*
    mechanics of, 78–79
  by facets, 34–35, 71–73, 78, 593–596, *593–596*
Local pain, 377
Locus of control, 683
Longissimus thoracis muscle, pain trigger points and referral patterns in, *228*
Longitudinal ligaments. *See* Anterior longitudinal ligament; Posterior longitudinal ligament
Lordosis. *See also* Extension
  assessment of, 431, *432*
  low back pain and, 81–82, 280
  lumbar spine effects of, 40
Lovett reverse sciatic scoliosis, 56, 62, 65
Low back exercises, disc injury from, 101
Low back pain. *See also* Pain
  acute, 310, 377
  in acute back sprain, 384, *385*
  afferent pathways of, 28, *28*
  in AIDS, 522
  anatomic factors in, 81–82
  anulus fibrosus in, 29–30
  anulus fibrosus in, 29–30, 31–33, 49–51, 82, 379–381
  atherosclerosis and, 693
  biomechanics of, literature update for, 689–691
  bracing for, 333–336, *334–336*
  bulging disc and, 386, *386*, *387*
  caffeine and, 23, 681
  causes of, 379–382
    differential diagnosis of, 379, 380t
  chemical sensitization and, 29, 30–31, 48, 49, *50*
  in children, 82, 529
  in chondromalaciae facetae, 113, *113*
  chronic, 310, 377
    psychological factors in, 679–686. *See also* Psychological issues
    socioeconomic risk factors for, 680
  classification of, 377, 381t, 384–392
  communication of, 679
  computed tomography in, 112
  Cox classification of, 446–449
  in Cushing's syndrome, 520
  cytokines in, 145
  Dallas Discogram classification of, 381t
  demographics of, 81–82, 378, 527
  depression and, 679, 684
  diagnosability of, 29
  diagnosis of, 377–502
  disc and facet biomechanics in, 25–26
  disc anular irritation and, 49–51
  disc as source of, 21–22, 36–37, 382–384, 388–392, *388–392*, 407, 566
  disc protrusion and, 30–31, *31*, 36–37
  disc-facet relationship in, 25–26, 28, 29, 33–34
  displaced fragment and, 387–388, *387–390*
  distribution of, 448
    vs. facet pain distribution, 448–449
  dorsal root ganglia in, 138–141, 286–288, *287*
  drug therapy for, 336–339
  dura mater in, 29, 695
  duration of, 310, 377, 529
  end plates in, 107
  epidural anesthesia for, 40
  facet joint injection for, 33
  facet joints in, 109–112, *110*, *111*
  familial factors in, 22
  fascia in, 33
  financial impact of, 527–528
  first onset, 377
  flare-up, 377
  fluid ingestion and, 384–385, *385*
  galvanism for, 330–333, *331*, *332*
  genetics of, 22, 378
  heart disease and, 25
  history of growth period pain and, 22
  hyperparathyroidism and, 520
  iatrogenic, diagnosis of, 447
  immobilization and, 113
  immunoglobulins in, 145
  incidence of, 695–696
    in children, 378
  in infective endocarditis, 522–523
  interneural inflammation and, 287–288
  intradisc, 31
  intraosseous pressure in, 37, 176–177
  kyphosis and, 81–82
  L1–L2 dorsal ramus entrapment and, 28–29
  leg length inequality and, 82, 449
  ligaments in, 33, 695
  local, 377
  localization of, 28
  lordosis and, 81–82, 280
  loss of diurnal height and, 25
  lumbar zygapophysial joint pain and, 29
  in Lyme disease, 522
  malignancy-induced, 377–378
  mechanisms of, 25–29, 36–37
  mechanoreceptors in, 110–112
  in metabolic disorders, 520
  muscular dysfunction and, 655–657
  nerve root compression and, 287–288
  nociceptors in, 144, 605
  nonvertebral causes of, 463–501
  occupational factors in, 22, 81
  organic diseases causing, 463–501
  organic idiopathic, 392
  osteomalacia and, 520
  in Paget's disease, 522
  pain receptors in, 144
  pathologic sequence in, 182
  perception of, 147
  phospholipase A_2 in, 145
  in polymyalgia rheumatica, 520
  in polymyositis, 522
  posterolateral anulus disruption and, 385–386, *386*
  postoperative
    clinical correlates of, 559
    radiographic correlates of, 559
    scar tissue and, 559
  in pregnancy, 22–23, 496
  prevention of, 550

previous back injury and, 23
prostaglandins in, 145
provocation of, 112
psychological issues in, 679–686. *See also* Psychological issues
radicular, 377
recurrent, 377
job satisfaction and, 529
postoperative. *See* Failed back surgery syndrome (FBSS)
referred, 28, 156, 377, 602t, 602–603, *603*
anterior herniation and, 461, *462*
risk factors for, 22–23
nonorganic, 529, 680, 682
sacroiliac joint in, 209, 223–231. *See also* Sacroiliac joint pain
sciatica and, 383–384. *See also* Sciatica
sequestered fragment and, 387, *387*
sex and, 112–113
smoking and, 23–25, 379
sources of, 25–34, *27*
spinal fixation and, 286
in spinal stenosis, 179
in spondylolisthesis, 615–616, *616*, 617
subacute, 310
substance P in, 140–141, 145–146
surgery for. *See* Surgery
indications for, 274
transient, 377
transitional segment in, 237
traumatic-onset, 25
trunk length in, 40
trunk velocity and, 90–91
urinary incontinence and, 162–163
vertebral bodies in, 107
vibration effects in, 22, 109
weightlessness and, 25
without sciatica, diagnosis of, 383–384
zygapophysial osteoarthritis and, 36–37, *37*, *38*
Low back wellness school, 297
Lower crossed syndrome, 658
Lower thoracic iliocostalis muscle, pain trigger points and referral patterns in, *228*
Lumbar decompressive laminectomy, for spinal stenosis, 195–197
Lumbar epidural analgesia/anesthesia, complications of, 553
Lumbar fusion. *See* Spinal fusion
Lumbar intervertebral disc syndrome, pathophysiology of, 27–28
Lumbar lordosis
assessment of, 431, *432*
low back pain and, 81–82, 280
lumbar spine effects of, 40
Lumbar mechanics, 68–74
centrode location and, 68–69, *68–71*
rotation, 69–70, *73*, *74*
summary of, 70–71
Lumbar motion, physiologic and abnormal, 68–74
Lumbar muscles, in flexion-distraction manipulation, electromyographic studies of, 263–264, *264*, *265*
Lumbar radiculopathy. *See* Radiculopathy
Lumbar rib, imaging of, 462, *462*
Lumbar spine
age-related changes in, 66, 70
anterior vs. posterior elements of, 70
biomechanics of, 17–23
cervical spine effects on, 568
dissection of, 577–578, 579
diurnal stress variations on, 40–41
embryonic development of, 600–601
extension effects on, 85–90, *87*, *276*, 276–277, *277*
flexion effects on, 85–90, *87*, 276, *276*, *277*
motion dynamics and aberrancies of, 84–101
posture effects on, 78
rotational and lateral flexion capabilities of, 97
stability of
ligaments and facets in, 92–94
Van Akkerveeken's measurement lines and, 599–601, *599–601*
suspension effects on, 81
Lumbar spine instability
acute vs. chronic, 105
lumbar vertebral translation in, 105
radiographic findings in, 105
Lumbar spine sprain/strain, 384, *385*
Cox manipulation for, results of, 311t–313t, *322*, *323*
diagnosis of, 384, *385*
vs. disc disruption, 31
Lumbar support orthosis, 334, *334*
for transitional segment, *242*
Lumbar sympathetic afferents, in pain transmission, 28, *28*
Lumbar synovial cyst, 492, *492*, *493*
Lumbar vertebra(e), L5
locking mechanism of, 611, *612*
pseudosacralization of, 237, *238*, 413, *414*
sacralization of. *See* Transitional segment
Lumbar vertebral translation, 105
Lumbar zygapophysial joint pain, 29
Lumbopelvic control, pelvic tilt for, 667, *667*
Lumbosacral brace, semirigid, 334, *334*
Lumbosacral corset, 334, *334*
Lumbosacral ligament
compression of L5 nerve by, 478
nerve root entrapment/fixation by, 157
Lumbosacral list, assessment of, 430–431, *431*
Lumbosacral plexus, anatomy of, 17–23
Lumbosacral radiculopathy, furcal nerve in, 158
Lumbosacral support, for spondylolisthesis, 644, *644*
Lumbosacral transitional vertebrae, 237–249. *See* Transitional segment
in low back pain, 237
Lyme disease, laboratory evaluation in, 522, *523*
Lymphoma
non-Hodgkin's
of epidural space, 478
methotrexate-related, 483

Magnetic resonance imaging (MRI). *See also* Imaging
abnormal, with normal x-ray, 490, *491*
advantages of, 408–413
artifacts on, 411
vs. computed tomography, 408, 416
contraindications to, 411
vs. CT myelography, 408
in disc degeneration, 109
of disc herniation, 406
disc hyperdensity on, 413
vs. discography, 392–394, *393*, *394*
gadolinium-enhanced, 413–416, *415*, *416*
indications for, 406, 407–408
vs. myelography, 408
normal findings on, *412*
pain source and, 413
postoperative, 536
in pregnancy, 496
principles of, 409–411
specificity and sensitivity of, 406
in spondylolisthesis, 627–629, *628*, 629t, *630*
T-1 weighted images in, 410–411
T-2 weighted images in, 411

Magnetic resonance imaging (MRI)—*Continued*
timing of, 406
Major histocompatibility complex, 516, *516*, 516t
Malic acid, for fibromyalgia, 256
Malignancy-induced back pain, 377–378. *See also* Cancer
Malignant melanoma, spinal metastases in, 478
Malingering
diagnosis of, 444–446, *445*, *446*
signs of, 424
Malnutrition, spinal stenosis and, 179–180
Manipulative-adjustment procedures, for sacroiliac joint pain, 227–229, *229*
Mannkopf's sign, 444, *445*
Marital discord, 683, 684
Marrow, in disc degeneration, 105
McKenzie treatments
vs. chiropractic care, 9
results of, 555
McManis, John, 1, 274
McManis table, 1–2
Mechanoreceptors
activation of, flexion-distraction manipulation and, 285–286
in facet joints, 110–112, 286
in low back pain, 110–112
Melanoma, spinal metastases in, 478
Meningioma, cystic, 481–483, *482*
Meralgia paresthetica, 490–492
Mesh cage implant, 551
Mesoinositol hexanicotinate (Hexopal), for spinal stenosis, 196
Metabolic disorders, back pain in, 520
Metastasis, bone, 488–490, *489*, *490*
alkaline phosphatase and, 511
laboratory evaluation of, 520
in melanoma, 478
pain in, 377–378
in prostate cancer, 478–479, *479*
Metatarsal stress fracture, 487, *488*
Methotrexate, for rheumatoid arthritis, lymphoma and, 483
Microdiscectomy, results of, 537
Milgram's sign, 440, *440*
Minor's sign, 424, *429*
Mobilization, vs. distraction manipulation, 5–6
Monoclonal gammopathy, in multiple myeloma, 519
Moses' sign, 442, *443*
Motion studies, of nucleus pulposus in flexion-extension, *101*, 101–105, 102t, *103*, *104*
Motor changes, in disc disease, 418t, *419–420*, 420
Movement pattern assessment, for rehabilitation program, 662
MRI. *See* Magnetic resonance imaging (MRI)
Multifidi muscles, 215–216, *218*
in disc degeneration, 110
pain trigger points and referral patterns in, *228*
postoperative changes in, 536
spinal stability and, 654–655, *655*
Multiple laminotomy, for spinal stenosis, 197
Multiple myeloma, 483
back pain in, 377–378
laboratory evaluation in, 518–519, *520*, *521*
Muscle(s). *See also* Paraspinal muscles and specific muscles
Muscle balance, evaluation of, 657t, 657–659
Muscle imbalance syndromes, 658
Muscle relaxants, 337
Muscle strain. *See also* Low back sprain/strain
vs. disc disruption, 31
Muscle weakness, assessment of, 418t, *419*, *420*, 421
Muscle-joint relationships, 665t
Muscular dystrophy, 468, *468*, *469*
Myelography. *See also* Imaging
computed tomography, 408, 409
in degenerative spondylolisthesis, 638, *640*
limitations of, 417
vs. magnetic resonance imaging, 408
in spinal stenosis, 175–176
Myeloma, 483
back pain in, 377–378
laboratory evaluation in, 518–519, *520*, *521*
Myofascial pain syndrome
vs. fibromyalgia, 254, 254t
trigger points and referral patterns in, 227, *228*
Myofascial release techniques, 665, 666
Myofascitis, cervical pain and, 449

Nachlas' knee flexion sign, 442–443, *443*
Nephrocalcinosis, 497, *497*
Neri's bowing sign, 430, *430*
Nerve(s). *See also* Specific nerves
edema of, in nerve root compression, 149
innervating discs, 28
irritation of, sources of, 27–28
Nerve block, for low back pain, 40
Nerve fibers, compression of, 141–143, *142*
Nerve regeneration, 161
Nerve root(s)
anatomy and physiology of, 137, *170*, 170–172, *171*
anomalous anastomosis of, 422
blood supply of, 136–137, *138*, *148*, 148–149
compression ischemia of, *148*, 148–149
conjoined, 473, *473*, *474*
dermatomes for, 55–56, *57*
innervation of, 55–56, *57*
needle stimulation of, vs. electromyography, 421–422
vs. peripheral nerves, 136
symptomatic, MRI identification of, 416
Nerve root compression, 141–143, *142*
in disc degeneration, cadaver studies of, 36
in disc herniation vs. spinal stenosis, 149
epidural fat graft–induced, 536
flexion effects on, 277
in foraminal narrowing and subluxation, 17, *18–20*
intraneural edema and, 149
ischemia in, 171
L5, interstitial cystitis and, 162
literature update for, 691–693
mesh cage implant for, 551
pain mechanisms in, 172, 287–288
pathophysiology of, 156–159, 170–172
pelvic pain and, 162–164
spinal stenosis and, 170–172
in spondylolisthesis, 616–617
vulnerability to, 34
Nerve root entrapment
ligamentous, 157
pathogenesis of, 67, 67, *68*
in spinal stenosis, 173–174
Nerve root fixation, ligamentous, 157
Nerve root irritation, causes of, 150, 691–693
Nerve root ischemia, intermittent claudication and, 181
Nerve root origin, from cauda equina, 62–64, 66
Nerve sheath tumor, 481
vs. extraforaminal herniation, 451–452
Neugebauer's method, 564
Neuralgia, obturator nerve, 454
Neurilemoma, 481
of sciatic nerve, 468

Nociceptors, 30–31
 in anterior disc, 33
 in anterior longitudinal ligaments, 33
 in anulus fibrosus, 32–33
 in inflamed vs. normal joints, 159
 in low back pain, 144, 605
Noncompliance, 288
Non-Hodgkin's lymphoma
 of epidural space, 478
 methotrexate-related, 483
Nonorganic pain drawings, 685
Nonsteroidal anti-inflammatory drugs (NSAIDs)
 classification of, 336–337
 efficacy of, 339
 in elderly, 339
 for fibromyalgia, 256
 indications for, 336
 mechanism of action of, 336
 side effects of, 337–339, 699
Nuclear pressure, decreased, in disc degeneration, 112
Nucleus pulposus
 bulging of, diagnosis of, 447
 elasticity of, *278*, 278–279, 285
 fluid ingestion in, pain and, 384–385, *385*
 herniation of. *See* Disc herniation
 hydrophilia in, 341
 inflammatory response to, 29, 146. *See also* Chemical radiculitis
 intradisc location of, 68–69, *68–74*
 leakage of. *See also* Disc prolapse; Disc protrusion
  discography of, 396–405, *396–405*
 lesions of, in disc degeneration, 108
 movement of
  extension effects on, *101*, 101–105, *103*, *104*, 278–279
  flexion effects on, *101*, 101–105, *103*, *104*, 278–279
  in normal vs. abnormal disc, 279
 pressure changes in, 64, 66
 sequestered, 387, *387*, 461, *461*
  displaced, 387–388, *387–390*
Nucleus pulposus emboli, postoperative, 536
Nutrition, 340–343
 arthritis and, 340
 disc, 341–343
  end plate receptors and, 25
  exercise and, 342
  smoking and, 343
 disc degeneration and, 106, 340, 458–459
 in home care, 340–342
 spinal stenosis and, 179–180
 training in, 8

Obesity
 disc disease and, 25
 low back pain and, 81–82
Obturator internus bursitis, 478
Obturator nerve, anatomy of, 21
Obturator nerve neuralgia, 454
Occupational factors, in low back pain, 22, 81, 529, 553–554, 681
Oophorectomy, spondylolisthesis and, 617
Opioids, 337
Oral tolerization, for rheumatoid arthritis, 343
Organic idiopathic spine pain, 392
Orthotics, 333–336, *334–336*. *See also* Bracing
 diagnostic, 539
 for disc herniation, 333–336, *334–336*
 lumbar support, 334, *334*
  for transitional segment, *242*
 for pes planus, 605, 606
 for transitional segment, *242*
 types of, 334–336, *334–336*
Osteitis condensans ilii, 225
Osteitis deformans, laboratory evaluation in, 522
Osteoarthritis
 Arteparon for, 340
 chondroitin sulfate for, 340, 342–343
 disc changes in, 106–108
 facet, 42–43
 glucosamine sulfate for, 342
 hip, leg length inequality and, 118
 nutritional therapy for, 340–344
 proteoglycan loss and, 340
 of zygapophysial joint, 37, *37*, *38*
Osteoarthrosis, 66
Osteochondrosis, intervertebral, 102–104
Osteomalacia, laboratory evaluation of, 520
Osteomyelitis
 of femur, 487–488, *488*
 of L3–L4 disc, *484*, 484–485, *485*
 laboratory evaluation in, 517–518
Osteopathic lesion, definition of, 275
Osteopathic manipulative therapy, 7–8
Osteopathy, principles of, 274–275
Osteopenia, causes of, 520, 521t
Osteophytes
 in disc degeneration, 108–109
 sacroiliac joint, 214, *215*
 spinal stenosis and, 186, *189*
 sympathetic nerve trunk compression by, 141
Osteoporosis
 compression fracture in, 492–493
 laboratory evaluation in, 516–517
 literature update for, 700–703
 transient, of hip, 497
Osteosarcoma, vitamin D_3 for, 343
Oswestry Disability Scale, 534, 659
Outpatient care
 benefits of, 528
 chiropractic care as, 528

Paget's disease, 463, *464*
 alkaline phosphatase in, 511, 512t
 laboratory evaluation in, 522
Pain
 abdominal, spinal origin of, 163
 buttock, in piriformis syndrome, 114–117, *115*, *116*
 cervical, myofascitis and leg pain and, 449
 communication of, 679
 deep, 144
 facet, patterns of, 602t, 602–603, *603*
 flank, spinal origin of, 163
 iliac crest, L1–L2 dorsal ramus entrapment and, 28–29
 iliocostal, 496–497
 inguinal, source of, 28
 joint, pathophysiology of, 285–286
 leg. *See* Leg pain; Sciatica
 low back. *See* Low back pain
 in multiple myeloma, 518–519
 myotomal vs. sclerotomal vs. dermatomal, 157
 in nerve root compression, 172
 nociceptors for, 30–31, *31*
 on palpation, 431, *432*
 pelvic
  Cox distraction for, 285
  flexion-distraction manipulation for, 163t, 163–164, 285
  nerve compression and, 162–164

Pain—*Continued*
in pelvic pain and organic dysfunction (PPOD) syndrome, 163t, 163–164
in pregnancy, 22–23
perception of, 147–148, *148*
radicular, pathogenesis of, 150
referred, 156, 602t, 602–603, *603*
convergence-facilitation theory of, 158
convergence-projection theory of, 158–159
sacroiliac joint, 223–231. *See also* Sacroiliac joint pain
scrotal, 422
in disc compression of S2/S3 nerve roots, 21
somatic, 144
subjective assessment of, 423
substance P and, 140–141, 145–146, 150, 286
testicular, thoracolumbar dysfunction and, 163
Pain drawing, patient, *423*, 423–424
Pain management. *See also* Analgesia
antidepressants in, 337
facet joint injection in, 33
muscle relaxants in, 337
nonsteroidal anti-inflammatory drugs in, 336, 337–339
opioids in, 337
steroids in, 336
Pain receptors, 30–31
in anterior disc, 33
in anterior longitudinal ligaments, 33
in anulus fibrosus, 32–33
in inflamed vs. normal joints, 159
in low back pain, 144
Pain relief, extent of herniation reduction and, 150–152, *150–152*
Palpation, pain on, 431, *432*
Panjabi spinal stability model, 654, 654t
Paralysis, postoperative, 536
Paraplegia, postoperative, 536
Paraspinal muscles. *See also* Muscle(s) and specific muscles
balance of, evaluation of, 657t, 657–659
in disc degeneration, 110
inhibition prone, 657, 657t
low back pain and, 655–657
spinal stability and, 654t, 654–655, *655*, *656*
stereotypic responses of, 657, 657t
tightness prone, 657, 657t
Parathyroid hormone, 512, 520
Pars interarticularis, elongated, pseudospondylolisthesis and, 643
Pars interarticularis defect, in spondylolisthesis, *613*, 614, *614*. *See also* Spondylolisthesis
Patellar reflex testing, 418t, *419*, 441, *441*
Patient pain drawing, *423*, 423–424
Patient-doctor interaction, 683–684
Patrick's sign, 438, *438*
Patrick's test, for sacroiliac pain, 226
Pedicle screw fixation
after failed surgery, 544–550, *545–550*
literature update for, 698–699
nerve root irritation in, 543–544
Pelvic crossed syndrome, 658
Pelvic disease, disc degeneration and, 417
Pelvic muscles
pain trigger points and referral patterns for, 227, *228*
sacroiliac joint and, 215–216, *218*
Pelvic pain
Cox distraction for, 285
flexion-distraction manipulation for, 285
nerve compression and, 162–164
in pregnancy, 22–23
Pelvic pain and organic dysfunction (PPOD) syndrome, flexion-distraction manipulation, 163t, 163–164
Pelvic stabilization exercises, costs of, 528
Pelvic tilt, 667, *667*
Pentoxifylline, for spinal stenosis, 196
Peptic ulcers
nonsteroidal anti-inflammatory drugs and, 337, 338
spinal manipulation for, 554
Percussion, 431, *432*
Percutaneous discectomy. *See also* Discectomy
indications for, 534, 535
results of, 535, 537
MRI findings of, 559
Peripheral nerves. *See also under* Nerve(s)
anatomy of, 137, *138*
vs. nerve roots, 136
Perna canaliculus extract, for arthritis, 340
Peroneal muscle testing, 439–440
Pes planus, orthotics for, 605, 606
Phospholipase A_2, in low back pain, 145
Phosphorus levels, 513
Physical examination, 424–446
dermatome testing in, 441, *441*
form for, *425–428*
goniometric measurements in, 431–434, *433*, *434*
lower limb circulation in, 442
palpation in, 431, *432*
percussion in, 431, *432*
in prone position, 442–444
reflex testing in, 418t, *419*, *420*, 440–441, *440–442*, 442
in sitting posture, 424–430, *429*
in standing position, 430–434, *430–434*
in supine position, 435–442
during treatment, 448
Physical therapy
chiropractic and, 5–7
effects of on connective tissue, 7
for reflex sympathetic dystrophy, 161
Physioball routines, 669–672, *669–673*
Pinwheel examination, 441, *441*
Piriformis bursitis, 117
Piriformis muscle, 215–216, *218*
pain trigger points and referral patterns in, *228*
Piriformis postisometric relaxation, 666
Piriformis self-stretch, 666
Piriformis syndrome, 114–117, *115*, *116*, 454
chemical irritation and, 144
Placebo effect, 10, 686
Plantar flexion
of ankle, 439, *439*
of foot, assessment of, 418t, *420*
of great toe, 440, *440*
assessment of, 418t, *421*
Plantar flexion test, 445, *445*
Plasma protein, leakage of into cerebrospinal fluid, 158
Polymyalgia rheumatica, 479–480
laboratory evaluation in, 521
Polymyositis, laboratory evaluation in, 522
Popliteal artery, assessment of, 442, *442*
Popliteal fossa pressure, 444, *444*
Positioning, for Cox distraction, 291–292, *292*, *293*, *294*, 294–295
Positive galvanism, after Cox distraction, *330*, 330–333, *331*, 331t
Posterior apophyseal ring fracture, 481, *482*
Posterior innominate procedure
flexion-distraction, 229, *229*
manipulative-adjustment, 229, *229*
Posterior longitudinal ligament

ganglion cyst of, 480
innervation of, 33
loads on, in flexion-distraction manipulation, 268–271, *269–271*
ossification of, spinal stenosis and, 195
as pain source, 33, 695
Posterior longitudinal ligament causalgia, 33
Posterior sacroiliac ligament, 214–215, *216, 217*
Posterior tibial artery, assessment of, 442, *443*
Postural analysis, 662, 663t
Posture
antalgic, 56–62, *58–64*, 59, *61, 63*
assessment of, 430–431, *431*
Cox distraction positioning for, 296, *296*
disc disease and, 416
in disc protrusion, 56–58, *58*
for lifting, 280
lumbar spine effects of, 40, 78
muscle dysfunction and, 657, 663t
in sciatica, 53–55, *55*
in spinal stenosis, 175, *175, 176*
Prednisone, 336, 337
Pre-employment radiography, 529
Pregnancy
back pain in, 22–23, 496
chiropractic care in, 496
MRI in, 496
sacroiliac joint mobility in, 219–220, *220*
spondylolisthesis in, 618
Pressure algometry, 452–454
Pressure goading, for Cox distraction, 328, *329*
Primary nerve sheath tumor, 481
Prone knee flexion test, *443*, 443–444
Proprioceptors, in anulus fibrosus, 32
Prostaglandins, in low back pain, 145
Prostate cancer, 478–479, *479, 480*
Prostate-specific antigen, 513
Protein-polysaccharide synthesis, abnormal, in disc disease, 49, *50*
Proteins, axoplasmic transport of, 132–136
Proteoglycans
cartilage and, 340–343
loss of, in disc degeneration, 106, *106*, 107, 340
sulfated, 342–343
synthesis of, 342
Provocation tests, 112
for sacroiliac joint pain, 226
Pseudogout, spinal stenosis and, 195
Pseudosacralization
of L5, 237, *238*
imaging of, 413, *414*
with pseudospondylolisthesis, 641, *641*
Pseudospondylolisthesis, 632
case studies of, 637, *637, 638*, 641, *641*
elongated pars interarticularis and, 643
pseudosacralization with, 641, *641*
Psoas muscle hematoma, 478
Psoas postisometric relaxation, 665, 666
Psoas self-stretch, 666
Psoriatic arthritis, laboratory evaluation in, 521–522
Psychological issues, 679–686
childhood trauma, 682
congruency, 680–681, 684
coping strategies, 680–681, 684, 685
depression, 679, 682, 684
diagnosis and identification of problems, 685
doctor-patient interaction, 683–684
fear-avoidance behavior, 685
illness behavior, 681
locus of control, 683
marital discord, 683, 684
occupational, 681
patient profile, 680, 682–683
placebo effect, 686
quality of life, 685
sexual abuse, 682
sick role, 681, 682, 683–684
somatization, 680
spousal support, 682, 683
treatment-related, 685–686
unemployment, 681
Psychotherapy, for fibromyalgia, 256
Pubis symphysis, mobility of, 220
Pudendal nerve, anatomy of, 21
Pudendal plexus, 417
Pulses, assessment of, 442, *442, 443*
Pyuria, 511

Quadratus lumborum muscle
pain trigger points and referral patterns in, *228*
spinal stability and, 655, 657
Quadriceps muscle testing, 418t, *419*
Quadriplegia, postoperative, 536
Quadruped track, 670, *671*
Quality of life, 685
Quebec Back Pain Disability Scale, 534

Radicular pain, 377
pathogenesis of, 150
Radiculitis, chemical, 143–148
Radiculopathy
chemical, 143–148
vs. mechanical, 146–147, *147*
diabetic, 480
dorsal route ganglia and, 132–143
Epstein-Barr virus infection and, 481
herpes zoster, 480
lower-extremity, discogenic, 394–396, *395*
Radiofrequency facet denervation, 604
Radiography, 56–64. *See also* Imaging
bending studies, 56–64, *61–65*
in facet syndrome, 596–599, *596–601*
indications for, 531–532
in leg length discrepancy evaluation, 118–119, 121t
of Lovett reverse scoliosis, 62, 65
pre-employment, 529
in sacroiliac joint pain, 227
of spinal stenosis, *183*, 183–185, *184*
in spondylolisthesis, 618–619, *619–621*, 629
Range of motion, assessment of, 434, *434*, 529
Range of motion adjustment
circumduction, 300–301, *301*
in Cox distraction, 298–301, *298–301*
extension, 301, *301*
flexion, *298*, 298–299
lateral flexion, 299, *299*
rotation, 300, *300, 301*
Reactive arthritis, laboratory evaluation in, 521–522
Rectus abdominis muscle, 216, *218*
spinal stability and, 655, 656
Recurrent meningeal nerve. *See* Sinuvertebral nerve
Referrals
resistance to, 7
wisdom of, 8

Referred pain, 28, 158–159, 377
  anterior herniation and, 461, *462*
  convergence-facilitation theory of, 158
  convergence-projection theory of, 158–159
Reflex(es)
  ankle jerk, 418t, *420, 440,* 440–441
  hamstring muscle, 442, *442*
  patellar, 418t, *419,* 441, *441*
  spinal, spinal fixation and, 286
Reflex sympathetic dystrophy, 160–161
  postoperative, 535–536
  sciatic radiculopathy and, 149
  spinal stenosis and, 178–179
Rehabilitation, 653–676. *See also* Exercises
  exercise prescription for, 655–676
  exercise tracks in, 668–676
  flexibility training in, 665, 666
  manipulation in, 655, 655t
  myofascial release in, 665t
  patient assessment for, 659–665
    functional testing in, 659t, 659–665
    movement pattern analysis in, 662
    patient-generated outcome tools in, 659
    postural analysis in, 662, 663t
  spinal stabilization training in, 665–676
  treatment continuum in, 665t
Reiter's syndrome, laboratory evaluation in, 521–522
Relaxation techniques, for fibromyalgia, 256
Renal calculus, staghorn, 463, *464*
Renal dialysis, spinal stenosis and, 195
Renal disease, nonsteroidal anti-inflammatory drugs and, 338
Repetitive squat test, 659–660, 660
Repetitive trunk curl, 660, *661*
Retrolisthesis, 634, *636*
Retrolisthesis subluxation, 606–608, *606–608*
Return to work, 553–554, 681–682
Reverse spondylolisthesis, 634, *636*
Rheumatoid arthritis, 483
  methotrexate for, lymphoma and, 483
  oral tolerization for, 343
  rheumatoid factor in, 513–514, *514,* 514t
  tests for, 483
  type II collagen and, 343
Rheumatoid factors, 513–514, *514,* 514t
  tests for, 483
Rib, lumbar, imaging of, 462, *462*
Ro, Chae Song, 235, 577
Rocker board, 672, *674–675,* 675
Rodman, John C., 1
Rotation
  anatomic limits on, 82–84, *83–85*
  with disc degeneration, 101
  disc injury from, 41, 75, *80,* 593
  vs. lateral flexion, 97–99, 97–100, 100t
  level of, 95
  resistance to
    axis of rotation and, 82–83, *83–85*
    by discs, 95
    by facets, 95
    by ligaments, 95
  upright vs. recumbent, 95, 95–97, *96*
Rotation mechanics, 69–70, *73, 74,* 75, *76, 80,* 593
Rotation range of motion adjustment, 300, *300, 301*
Rotation test, shoulder/pelvis, 446, *446*
Rule of three, 567

S1 dermatomes
  mapping of, 421
  pinwheel examination for, 441, *441*
Sacral artery aneurysm, 476
Sacral insufficiency fractures, 493, *493*
Sacral Tarlov cyst, 471–473, *472*
Sacroiliac joint, 209–231
  anatomy of, 209–219, *210–219*
  arterial supply to, 216, *218*
  biomechanics of, 219–223, *220*
  extrinsic ligaments of, 215, *216*
  hypermobility of, 224
  innervation of, 216–219, *219*
  instantaneous axis of rotation of, 221
  intrinsic ligaments of, 214–215, *216, 217*
  kinematics of, 219–220
  kinetics of, 221–223, *222, 223*
  load resistance of, 221
  in low back pain, 209
  marginal osteophytes of, 214, *215*
  mobility of, 219–220, *220*
    alterations in, 223
  mobilization of, 229
  morphology of, 209
  muscles surrounding, 215–216, *218*
    pain trigger points and referral patterns for, 227, *228*
  phylogenetic differences in, 211–212, *214*
  postnatal development of, 212–214, *215*
  self-bracing mechanism of, 221–223, 224, *224*
Sacroiliac joint distraction adjustment, *304,* 304–305, *305*
Sacroiliac joint infections, laboratory evaluation of, 517–518
Sacroiliac joint pain, 223–231
  causes of
    inflammatory, 225
    mechanical, *224,* 224–225
  diagnosis of, 226–227
  differential diagnosis of, 227, *228*
  exercise procedures for, 230
  flexion-distraction procedures for, 229, *230*
  functional restoration programs for, 230t, 230–231
  imaging in, 227
  management of, 227–231
  manipulative-adjustment procedures for, 227–229, *229*
  mapping of, *225,* 225–226
  mobilization for, 229
  pathogenesis of, 223–225
  physical examination in, 226–227
  presentation of, *225,* 225–226
  prevalence of, 223
  rehabilitation in, 230–231
  soft tissue injuries and, 227
Sacroiliac shear test, 226
Sacrospinous ligament, 215, *216*
Sacrotuberous ligament, 215, *216*
Sarcoidosis, 478
Sartorius muscle, 216, *218*
Scalloping, in spinal stenosis, 172–173
Scar tissue
  failed back syndrome and, 536
  pain correlation with, 559
  vs. recurrent disc herniation, 449, 536
Schmorl's nodes, 66
  in disc herniation, 109
Sciatic nerve
  anatomy of, 21
  endometriosis of, 480–481
  neurilemoma of, 468
Sciatic nerve entrapment, hamstring muscle scarring and, 483
Sciatic radiculopathy, relief of, by disc reduction, 150–152, *150–152*
Sciatic scoliosis, 53, *55, 56*

in lateral vs. medial disc protrusion, 449, *450*
Lovett reverse, *56*, 62, 65
Sciatica
brown tumor of hyperparathyroidism and, 481
bulging disc and, *386*, 386–387, *387*
cardiac surgery and, 481
causes of, differential diagnosis of, 379, 380t
chemical irritation in, 144
chemical radiculitis and, 117
clinical manifestations of, 53–55, *55*
Cox distraction for, *295*, 295–297, *296*, *297*
definition of, 377
diagnosis of, 53–55, *55*, *56*
disc herniation size and, 560
dorsal root gangliectomy for, 150
dorsal root ganglion compression and, 149–150
dual dermatome treatment for, 568, *569*
epidural anesthesia for, 40
natural course of, 533
nerve root compression and, 17, *18–20*
organic diseases causing, 463–474
pain distribution in, 55–56, *57*, 602t, 602–603, *603*
pain pathogenesis in, 147, 150
pathogenesis of, 383
pathologic change in sciatic foramen and, 449
pelvic disease and, 417
piriformis bursitis-induced, 117
piriformis syndrome and, 454
posterolateral anulus disruption and, 385–386, *386*
referred, 385–386, *386*
reflex sympathetic dystrophy and, 149
sequestered disc fragment and, 387–388, *387–390*
surgery for
indications for, 274
results of, 529
Scintigraphy, in sacroiliac joint pain, 227
Sclerotherapy, 550
Sclerotomes, 602–603
Scoliosis
age-related, 494–495
Cox distraction for, *303*, 303–304, *304*
with automated distraction adjustment, 309, *309*
degenerative, spinal stenosis and, 194, *194*, *195*
sciatic, 53, *55*, *56*
in lateral vs. medial disc protrusion, 449, *450*
Lovett reverse, *56*, 62, 65
strut graft placement in, disc disease after, 494
with syrinx, 494, *494*
Screw fixation
after failed surgery, 544–550, *545–550*
literature update for, 698–699
nerve root irritation and, 543–544
Scrotal pain, 422
in disc compression of S2/S3 nerve roots, 21
Semirigid lumbosacral brace, 334, *334*
Sensory motor stimulation, 667–668, 668t
Sensory motor stimulation track, 672–676, *674*, *675*
Serum acid phosphatase, 513
Serum alkaline phosphatase, 511, 512, *512*
Serum calcium, 512, 513t
Serum parathyroid hormone, 520
Serum phosphorus, 513
Serum uric acid, 512
Shear force, resistance to
by apophyseal joints, 91–92
by discs and facets, 71–73, 77
Shear test, for sacroiliac pain, 226
Shin splints, 469
Short foot, 672, *674*
Short leg. *See* Leg length inequality
Shoulder rotation test, 446, *446*
Sicard's sign, 436
Sick role, 681, 682, 683–684
Sickle-shaped ligament compression of L5 nerve, 478
Side lying circumduction adjustment, 303, *303*
Side lying extension adjustment, 302, *302*
Side lying flexion adjustment, 301–302, *302*
Side lying lateral flexion adjustment, 302–303, *303*
Side lying position, for Cox distraction, 296, *296*
Side posture adjustment effect, on myofascial point relief, 117
Side posture manipulation, for sacroiliac joint pain, 227–229, *229*
Side support track, 671, *671*
Single leg stance test, 660–662, *662*
Single-photon emission computed tomography (SPECT)
for facet abnormalities, *113*, 113–114, *114*
in spondylolisthesis, 629–631
Sinuvertebral nerve
anatomy of, 26–28, *27*
in pain transmission, 28, *28*
Sitting posture. *See also* Posture
disc effects of, 79, *80*
physical examination in, 424–430, *429*
sacroiliac joint problems and, 223
Skeletal metastasis, 488–490, *489*, *490*
alkaline phosphatase and, 488–490, *489*, *490*
laboratory evaluation of, 520
in melanoma, 478
pain in, 377–378
in prostate cancer, 478–479, *479*
Slipped femoral capital epiphysis, 498, *498*, *499*
Small foot, 672, *674*
Smoking
chronic low back pain and, 681
disc disease and, 379
disc malnutrition and, 343
intermittent claudication and, 180, 182
low back pain and, 23–25, 379
Snapping hip, 476
Socioeconomic risk factors, 680
Sock test, 438
Somatic pain, 144
Somatiform disorder, 684
Somatization, 680
Somatosensory evoked potentials, in spinal stenosis, 177
Sorenson's static back endurance test, 660, *661*
Soto-Hall sign, 435, *435*
Spina bifida occulta, 480
Spinal analgesia/anesthesia. *See* Analgesia; Anesthesia; Pain management
Spinal canal
anatomy of, *170*
flexion effects on, 277
measurement of, 173, 174–175, *175*, 182–183, 183–184, *183–185*, 185–186
narrowing of. *See* Spinal stenosis
normal vs. abnormal, *182*, *183*
posterior border of, 183, *183*
vs. vertebral canal, 185
Spinal cord
activity in, fixation of, 159
information processing/transmission in, 159–160
Spinal cord stimulation, for failed back surgery syndrome, 539
Spinal curvature. *See also* Lordosis; Scoliosis
low back pain and, 81–82
Spinal extensors, spinal stability and, 654–655, 655
Spinal fixation, 286

- Spinal fusion, 537–540. *See also* Surgery
  - adjacent segment motion and, 91
  - approaches in, 539
  - complications of, 538
  - contraindications to, 539
  - costs of, 537
  - indications for, 537–538, 539
  - rates of, 537
  - results of, 538, 539
  - for spondylolisthesis, 631, 643
- Spinal instability
  - clinical, definition of, 449
  - muscle imbalance and, 658–659
- Spinal manipulation. *See also* Cox distraction technique; Distraction manipulation; Flexion-distraction manipulation
  - for visceral conditions, 10, 10t
- Spinal metastasis. *See* Skeletal metastasis
- Spinal nerve roots. *See* Nerve root(s)
- Spinal nerves. *See also under* Nerve(s) and names of specific nerves
  - anatomy of, 137, *138*
  - axoplasmic transport in, 132–136
  - compression of, 141–143, *142*
  - dermatomes for, 55, *57*
  - injury of, anatomy and physiology of, 137
  - innervating disc, 28
  - irritation of, sources of, 27–28
  - in pain transmission, 28, *28*
  - trophic function of, 132
- Spinal orthoses. *See* Orthotics
- Spinal reflexes, spinal fixation and, 286
- Spinal stability
  - mechanisms of, 654–655, *655*
  - models of, 654, 654t
  - paraspinal muscles and, 654–659
- Spinal stabilization training, 665–676
  - abdominal co-contraction in, 667, *667*
  - common errors in, 668t
  - fast coordinated muscular activation in, 667–668, 668t
  - pelvic tilt in, 667, *668*
  - sensory motor stimulation in, 667–668, 668t
- Spinal stenosis, 67, *67*, 169–204
  - acquired (degenerative), 169
  - age and, 177
  - anatomic factors in, 173, *173*
  - calcium pyrophosphate dihydrate crystal deposition in, 192
  - canal diameter in, 182–183
    - measurement of, 183–184, *183–185*, 185–186
  - canal size in, measurement of, 172, 174–175, *175*
  - case studies of, 199–204, *199–204*
  - cauda equina syndrome and, 195
  - causes of, 186–195
  - central
    - causes of, 187
    - ligamentectomy for, 192
  - cerebrospinal fluid pressure in, 174
  - classification of, 169–170
  - clinical relevance of, 172–176
  - congenital, 169
  - conservative treatment for, 195–196, 198–204
    - calcitonin in, 198
    - case studies of, 199–204, *199–204*
    - drug therapy in, 196
    - flexion-distraction manipulation in, 198
    - lumbar traction in, 199
    - vs. surgery, 195
    - surgery for, 196–197
    - Cox distraction for, 200, 282–284
  - degenerative scoliosis and, 194, *194*, *195*
  - developmental, 187, *191*
  - diagnosis of, 173, 174–175, *175*, 447
  - disc disease and, 179
  - disc herniation and, 186, *188*, 188–189, *190*, *198*, *199*, 199–200
    - of thoracic discs, 198
  - dorsal root ganglia in, 178
  - dual level, 180
  - dural sac size in, 175
  - epidural pressure in, 178
  - flexion-extension in, *175*, *176*
  - foraminal, 169
  - foraminal osteophytes and, 186, *189*
  - free fragments and, *188*, 189–192
  - grading of, 186–187, *187*
  - growth and, 179–180
  - iatrogenic, 169
  - imaging of, 182–184
  - infant nutrition and, 179–180
  - intermittent claudication in, 172, 178, *180*, 180–182
    - atypical, 184
  - intraosseous blood flow and, 176–178
  - intraosseous pressure in, 174, 178
  - lateral canal, 187, *190*
  - leg pain in, 177, 178
  - ligamentum flavum in, 192, 200, *201*, *203*, 203–204, *204*
  - long-term sequelae of, 179
  - low back pain in, 179
  - low compression pressure in, 177
  - lumbar radiculopathy and, 185
  - multilevel, with unilateral spondylolysis, *467*, 467–468, *468*
  - myelopathy and, 172
  - nerve root compression in, 149
  - nerve/nerve root pathology in, 156–159, 170–172
  - nondiscal causes of, 195
  - ossified posterior longitudinal ligament and, 195
  - pagetoid, 195
  - pathogenesis of, *170*, 170–172, *171*
  - posture in, 175, *175*, *176*
  - presentation of, 179
  - pseudogout and, 195
  - radiography in, 173, *173*
  - radiography of, *183*, 183–185, *184*
  - reflex sympathetic dystrophy and, 178–179
  - reversal of, 117
  - root entrapment in, 173–174
  - scalloping in, 172–173
  - somatosensory evoked potentials in, 177
  - after spinal fusion, 539
  - spondylolisthesis and, 193, *193*, *194*
  - stages of, 179
  - surgery for, 185
    - vs. conservative treatment, 195
    - multiple laminotomy in, 197
    - vs. observation, 196–197
    - outcome in, 196, 197, 699
    - side effects of, 196
  - symptoms of, 178–179
  - tandem, spondylolisthesis and, 638, *639*
  - thecal sac pressure in, 175–176
  - thecal sac size in, 172
  - thoracic, 198
  - thoracolumbar burst fractures and, 193
  - transforaminal ligaments in, *192*, 192–193
  - trefoil vertebral canals and, *183*, 187–188, 380
  - types of, 174, *174*

Spinal unloading, intra-abdominal pressure effects on, 40
Spine
  embryonic development of, 600–601
  finite element model of, 707–711
  range of motion of, assessment of, 434, *434*
Spinous process contact, 298, *298*
Spondylitis, 485, *486*
Spondyloarthropathies
  laboratory evaluation in, 520–521
  sacroiliac joint pain and, 225
Spondyloarthrosis
  Cox manipulation for, results of, 311t–313t, *314, 315*
  diagnosis of, 447
Spondylolisthesis, 611–649. *See also* Spondylolysis
  age and, 617–618
  anatomic factors in, *613,* 614, *614*
  associated conditions in, 617
  in athletes, 643–644
  case studies of, 624–626, *625–627,* 634–643, *636–640,* 646–649, *646–649*
  in children, 613
  classification of, 611
  conservative treatment of, 644–649, *644–649*
  Cox distraction for, 305, *305*
    results of, 311t–313t, *318, 319*
  degenerative, 611, 632–634
    case studies of, 634–643, *636–640*
    facets in, 634
    myelography in, 638, *640*
    nonsurgical treatment of, 641–643, *642, 643*
    sagittal facet orientation and, 643
    spinal fusion for, 643
    spinal stenosis and, 193, *193, 194*
  in diabetes mellitus, 617
  diagnosis of, 447
  disability in, 617
  disc herniation with, 631
  distraction adjustment for, 305, *305,* 641–643, *642, 643,* 645–649
  dysplastic, 611
  exercises for, 644–645, *645*
  genetic factors in, 617
  hamstring length and, 618
  imaging of, 618–619, *619–621,* 627–631, *628,* 629t, *630*
  incidence of, 613
  instability of
    symptoms and, 623–624, *624*
    treatment results and, 618–623, *620–622,* 622t, 623t
  isthmic, 611, *612*
  L4, 631
  lumbosacral support for, 644, *644*
  nerve root compression in, 616–617
  oophorectomy and, 617
  pain in, 617
    origin of, 615–616, *616*
  pars interarticularis defect in, *613,* 614, *614*
  pathogenesis of, *613,* 614, *614*
  pathologic, 641
  post-traumatic, 611
  in pregnancy, 618
  reverse, 634, *636*
  risk factors for, 617–618
  spinal stenosis and, 193, *193, 194*
  subluxation in, 634, *635*
    reduction of, 624–627
  surgery for, 631
  tandem spinal stenosis and, 638, *639*
  with transitional segment, 241, *242,* 243, *243, 244*
  traumatic, 641
  uncommon varieties of, 641
  with vacuum instability, 626
  vibrational effects in, 617
Spondylolysis, 611–613, *613. See also* Spondylolisthesis
  in athletes, 643–644
  congenital clefts and, 627, *628*
  definition of, 51
  incidence of, 613
  unilateral, with multilevel spinal stenosis, *467,* 467–468, *468*
Spondylosis
  discogenic, 33, 66
  after spinal fusion, 539
Sprain, lumbar spine, 384, *385*
  Cox distraction for, 311t–313t, *322, 323*
  diagnosis of, 447–448
Squatting, lumbar spine effects of, 40, 79
Squatting test, 664–665
Staghorn calculus, 463, *464*
Stance. *See* Posture
Steroids
  aseptic necrosis of bone and, 104
  epidural injection of, 40, 551–553, 699
  facet injection of, 603
  indications for, 336
  mechanism of action of, 336
  side effects of, 336, 339
Stoddard's osteopathic technique, 567
Straight leg raising sign, 435, *435,* 437–440
  Lindner's sign and, 436
  for sacroiliac pain, 226
Strain, lumbar spine
  Cox distraction for, 311t–313t, *322, 323*
  diagnosis of, 447–448
  vs. disc disruption, 31
Strength and endurance tests, 659–662
Stress fracture, metatarsal, 487, *488*
Stress management, for fibromyalgia, 256
Subacute bacterial endocarditis, 478
Subluxation
  bending studies of, 58–62, *59–64*
  diagnosis of, 448
  facet. *See also* Facet syndrome
    Hadley S curve and, 593, *594, 595*
  in spondylolisthesis, 634, *635*
    reduction of, 624–627
Substance P, 286
  in anular nerve fibers, 145
  in dorsal root ganglia, 140–141, 145–146, 150
Sulfate metabolism, in disc, 341
Sulphated glycosaminoglycans, for arthritis, 340–341
Superior gluteal artery, 216, *218*
Superior gluteal nerve entrapment syndrome, 117
Supine track, 670–671, *671*
Supraspinous ligaments
  innervation of, 33
  resistance of
    to flexion, 91
    to rotation, 95
Surgery. *See also* Specific sites, disorders, and procedures
  for children, 530–531
  vs. chiropractic care, 273–274
  complications of, 535–536
  costs of, 527
  epidural anesthesia in, complications of, 553

Surgery—*Continued*
indications for, 274, 407, 532, 533–534
clinical vs. imaging, 284
rate of, 528–529
geographic factors in, 528
recurrent herniation after, vs. scar tissue, 536. *See also* Failed back surgery syndrome (FBSS)
repeat, results of, 531
results of
vs. conservative care, 529
outcome scales for, 534
predictors of, 529–530
psychological factors in, 686. *See also* Psychological issues
with reoperation, 531
severity of disease and, 537
scar tissue from
pain and, 559
vs. recurrent disc herniation, 449, 536
for sciatica
vs. conservative treatment, 529
results of, 529
selection of technique in, 534–535
for spondylolisthesis, 631
Sympathetic trunk, osteophytic compression of, 141
Syndesmophytes
in ankylosing spondylitis, 466, *467*
disc protrusion and, 405
Synovial cyst, lumbar, 492, *492, 493*
Syringohydromyelia, 494, *494*

Tall stature, disc disease and, 25
Tarlov cyst, sacral, 471–473, *472*
Taylor brace, 334, *335*
Tenderness to skin pinch test, 444, *445*
Tensor fascia femoris response, 442
Testicular pain, thoracolumbar dysfunction and, 163
Testicular torsion, 497
Tetanizing current
after Cox distraction, 333, *333*
for spondylolisthesis, 644, *644*
for transitional segment, *242,* 243
Tethered cord, 473–474, *474–476*
Thecal sac pressure, in spinal stenosis, 175–176
Thecal sac size, in spinal stenosis, 172
Thermal hyperalgesia, dorsal route ganglia and, 132–143
Thermography, 452
Thigh measurement, 440, *440*
Thomas flexibility test, 659, *660*
Thoracic disc calcification, in children, 580
Thoracic disc herniation, diagnosis of, 449
Thoracic spine, osteophytes of, sympathetic trunk compression by, 141
Thoracolumbar burst fractures, spinal stenosis and, 193
Thoracolumbar fascia, innervation of, 33
Thoracolumbar spine, facet orientation in, 43, *44*
Thoracolumbosacral orthosis, 334–335, *335*
Tibial nerve compression, from Baker's cyst, 483
Tissue compliance meter, 454
Toe. *See* Great toe
Toe walk, 434, *434*
Torque, lumbar spine effects of, 84–85
Torsion. *See also* Rotation
disc injury from, vs. compression injury, 97–100, 100t
Traction manipulation. *See also* Distraction adjustment; Flexion-distraction manipulation
autotraction, 283, *283*
biomechanical effects of, 564–566, *564–566*
Burton's concepts of, 564, *564*
contraindications to, 567
intermittent, 284
purposes of, 566–567
theories of, 564–566, *564–566*
for transitional segment, 237, *242*
Transcutaneous electrical stimulation, 568
Transdural disc herniation, 561–562, *562*
Transforaminal ligaments, in spinal stenosis, *192,* 192–193
Transforming growth factor-β, for disc dehydration, 551
Transitional segment, 237–249, 239–249
acupressure for, *242*
assessment of, 448
in Bertolotti's syndrome, 239, *239, 244–248,* 245–249, 390–392, 642
case studies of, *240–248,* 241–249
classification of, 239, *240*
Cox manipulation for, results of, 311t–313t, *322, 323*
disc herniation and, 239, *244–246,* 245
dysplastic, 239, *240*
flexion distraction for, 241–243, *242*
lumbarization of, 239, *240*
mixed, 239, *240*
pseudoarticulation in, 239, *240*
pseudosacralization of, 239, 241, *241, 244,* 245, *247,* 249
sacralization, 239, *240, 240–242,* 241–243
with spondylolisthesis, 241, *242,* 243, *243, 244*
Transplantation, disc, 551
Transversus abdominis muscle, spinal stability and, 655, *656*
Treadmill test, in intermittent claudication, 182
Treatment. *See also* Spinal manipulation; Surgery
algorithm for, *580–581*
case studies of, 570–577, *570–578*
chiropractic. *See* Chiropractic care
conservative
algorithm for, *580–581*
duration of, 568
imaging changes on, 557–558
costs of, 527–528
less commonly used forms of, 550–553
literature update for, 696–697
outpatient, benefits of, 528
placebo effect in, 686
prevalence of, 527
psychological aspects of, 685–686
re-evaluation after, 448
response to, 529
return to work after, 553–554
surgical. *See* Surgery
unconventional therapy in
frequency of use of, 4–5
payment for, 4–5
Trefoil vertebral canals
back pain and, 380
spinal stenosis and, *183,* 187–188, 380
Tricyclic antidepressants, for fibromyalgia, 256
Trigger point therapy, for Cox distraction, 328, *329*
Triple joint complex, laboratory simulation of, 593
Tropism, 448
atherosclerosis and, 693
facet, *41,* 41–48, 42t, *43*
disc degeneration and, 47
disc prolapse and, 43–47
facet orientation circle for, *47,* 47–48
prevalence of, 47
radiographic assessment of, 47
Trumpet laminectomy, for spinal stenosis, 197
Trunk curl test, 664, *664*

Trunk length, low back pain and, 40
Trunk velocity, low back pain and, 90–91

Ulcerative colitis, arthritis and, 493–494
Ulcers, peptic
  nonsteroidal anti-inflammatory drugs and, 337, 338
  spinal manipulation for, 554
Unconventional therapy
  frequency of use of, 4–5
  payment for, 4–5
Unemployment, 681. *See also* Work
Upper lumbar iliocostalis muscle, pain trigger points and referral patterns in, *228*
Uric acid, 512
Urinalysis, 510–511
Urinary incontinence, low back pain and, 162–163
Urinary problems, disc disease and, 417
Urine proteins, in multiple myeloma, 519, *520*

Valsalva maneuver
  Bechterew's test and, 424–430, *429*
  Lindner's sign and, 424–430, *429*
  spinal effects of, 40
Van Akkerveeken's measurement lines, for lumbar stability, 599–601, *599–601*
Vascular changes, in disc degeneration, 108
Vena cava filter, 470, *470*
Venous thrombosis, postoperative, 536
Ventral nerve roots. *See* Nerve roots
Ventral sacroiliac ligament, 214–215, *216, 217*
Vertebrae
  embryonic development of, 600–601
  fractures of. *See* Fracture(s)
  limbus, *461*, 461–462, *462*
  lumbosacral transitional, 237–249. *See also* Transitional segment
  modeling of, 173, *173*
  scalloping of, 172–173
Vertebral bodies, in low back pain, 107
Vertebral canal. *See also* Spinal canal
  vs. spinal canal, 185
  trefoil
    back pain and, 380
    spinal stenosis and, *183*, 187–188, 380
Vertebral fracture. *See* Fracture
Vertebral osteomyelitis, laboratory evaluation in, 517–518
Vertebral osteopenia, causes of, 520, 521t
Vertebral plates, degenerative changes in, 37, *38, 39*
Vertebral subluxation. *See* Subluxation
Vertebrogenic symptom complex, disc protrusion and, 32, *32*
Vibration exposure, low back pain and, 22
Vibratory sense, assessment of, 442
Vitamin D_3, for osteosarcoma, 343
Vitamin E, for fibromyalgia, 256

Walking, biomechanics of, 223
Wandering disc, *387*, 387–388
Weakness, assessment of, 418t, *419, 420*, 421
Weight, low back pain and, 81–82
Weightbearing. *See* Load bearing
Weightlessness, low back pain and, 25
Well leg raising sign, 436–437, *437*
Wobble board, 672, *674*, 675
Work, return to, 529, 553–554, 681–682
Workers' compensation, return to work and, 553–554, 681
Work-related factors, in low back pain, 553–554, 681

Yeoman's sign, 443, *443*
Yeoman's test, for sacroiliac pain, 226

Zenith-Cox table, 2
Zygapophysial joint
  osteoarthritis of, 37, *37, 38*
  pain in, 29